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SHORT CIRCUITS AND LONG NERVES: WHEN NERVES AND TUMORS CROSS WIRES

Claudel A

ABSTRACT

BACKGROUND: Oncological conditions pose a difficult assessment challenge due to the rarity, but also consequential nature of cancers. The medical field has developed different tools to reduce the diagnostic uncertainty. Further, as some conditions are rarely seen, an accurate diagnosis may delay treatment.

DISCUSSION: Several cancers affecting the peripheral nervous system will be reviewed. Their characteristics and best diagnostic tools will be explored. The consequences of cancer treatment and their consequences on the peripheral nervous system will also be reviewed.

CONCLUSIONS: The body of knowledge and ability to assist the rest of the medical team is dependent upon the precise nerve conduction and electromyography and high resolution ultrasound. The electromyographer will impact the patient's outcome by a diligent query of the nervous system and assist the referring team on further test necessary for treating their patients.

Alain Claudel, PT, DPT, ECS



Short Circuits and Long Nerves

When Nerves and Tumors Cross Wires

ALAIN CLAUDEL, PT, DPT, ECS

Objectives

At the completion of this session, applicants will:

- Have a good understanding of neurofibromatosis, neurolymphomatosis, perineurial spread of cancers, schwannoma
- Have a good understanding of paraneoplastic neuropathies, iatrogenic radiation and antineoplastic induced neuropathies and electrodiagnostic features
- Recognize the different diagnostic options: MRN, HRUS, PET

(Note: immune mediated neuropathies are excluded)



Neurofibromatosis

- Von Recklinghausen disease
- Genetic condition
- Mutation of the NF-1 (tumor suppressor) gene on chromosome 17
- Inherited autosomal dominant pattern
- 50% spontaneous



Neurofibromatosis

- Café-au-lait spots, freckles, neurofibromas
- Paravertebral neurofibromas
- Lateral thoracic meningocele
- 1 in 3000
- Usually detected in childhood
- Benign, may become malignant (Malignant Peripheral Nerve Sheath Tumors)



Neurofibromatosis

- Diagnosis is typically clinical
- HTN, ADHD
- Treatment: no cure, symptom management, surgery, chemotherapy (cisplatin)



Neurofibromatosis

Electrophysiology:

- Detect subclinical involvement
- Localize the lesion
- Identify neuropathy:
 - plexiform neurofibroma usually condition block
 - MPNST: axon-loss

Sensory neuronopathy in a patient with neurofibromatosis type 1

A case report

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Abstract

Rationale: Neurofibromatosis type 1 (NF-1) can manifest with various neurological symptoms. However, sensory ataxia has not been reported.

Patient concerns: A 44-year-old man with NF-1 presented with several weeks of unsteady gait. He was diagnosed with gastric neuroendocrine tumor with multiple hepatic metastases 6 years ago and received palliative chemotherapy. Neurological examination revealed ataxia veering to the right side with no motor weakness.

Diagnoses: Clinical manifestations and electrodiagnostic studies suggested the dysfunction of the thoracic dorsal column (DC). Initial magnetic resonance imaging showed a lateral thoracic meningocele (LTM) located in the right paravertebral area at the T3-T4 vertebral level, but the spinal cord was unremarkable. Gait disturbance worsened after 9 months, and follow-up magnetic resonance imaging showed high signal intensity involving the right DC at the level adjacent to the LTM and spinal cord atrophy distal to the DC lesion. Tests for well-characterized paraneoplastic antibodies were negative. Ultimately, the patient was assumed to have sensory neuronopathy due to compressive damage to the dorsal root ganglia within the intervertebral foramina by LTM.

Interventions: Empirical treatment with vitamin B12 supplementation and corticosteroids failed to improve his condition. The patient underwent decompressive laminectomy and excision of the meningocele with dura repair.

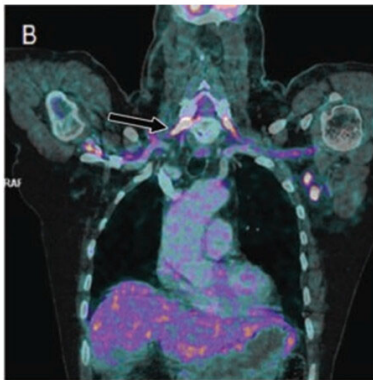
Outcomes: The patient temporarily improved to walk with assistance postoperatively. However, he developed dyspnea and hypotension 5 weeks later. Carcinoid heart disease confined the patient to the bed. The patient died of pneumonia 3 months after the operation.

Lessons: This case with NF-1 shows asymmetric sensory ataxia of subacute progression. LTM may contribute to the development of sensory neuronopathy by damaging sensory neurons of the dorsal root ganglia. The comorbidities of the patient, including gastric neuroendocrine tumor and LTM, made it challenging to investigate the pathomechanism.

Abbreviations: CIPN = chemotherapy-induced peripheral neuropathy, CSF = cerebrospinal fluid, CT = computed tomography,

Neurolymphomatosis

Images in *Radiology*

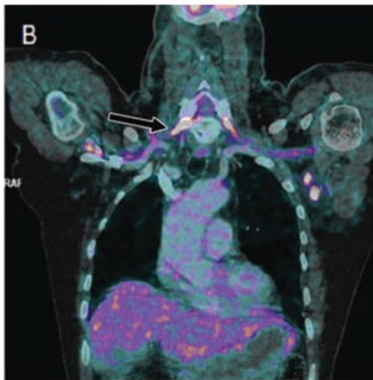


In a 62-year-old man with neurolymphomatosis, fused ^{18}F -FDG PET/CT image showed linear FDG uptake along brachial plexus nerve roots (black arrow).

- Rare manifestation of non-hodgkin's lymphoma
- Direct malignant lymphocytic invasion of peripheral nerves, plexi, spinal nerves and cranial nerves
- Must be differentiated from other complications of lymphoma:
 - Tumor compressive neuropathy
 - Paraneoplastic polyneuropathy
 - Iatrogenic toxic polyneuropathy

Neurolymphomatosis

Images in *Radiology*



In a 62-year-old man with neurolymphomatosis, fused ^{18}F -FDG PET/CT image showed linear FDG uptake along brachial plexus nerve roots (black arrow).

- Autopsy series report peripheral nerve involvement in 40% of patients who died from NHL
- Diagnosis:
 - MRI
 - F-Fludeoxyglucose PET
 - Electrodiagnostics
 - Radiculopathy and/or plexopathy
 - Mostly sensory motor axon-loss but may have demyelinating feature (as in CIDP)

Soni N and Ora M. Published Online: October 27, 2020
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Radiology



Neurolymphomatosis: a single-center experience of neuromuscular manifestations, treatments, and outcomes

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Abstract

Objectives Neurolymphomatosis (NL) is a disease characterized by the infiltration of malignant lymphocytes into the peripheral nervous system. We report clinical features, radiographic findings, modes of treatment, and outcomes of patients with NL.

Methods We retrospectively investigated patients with NL. We extracted data, including clinical features, magnetic resonance imaging (MRI), ¹⁸F-fludeoxyglucose (FDG) positron emission tomography/computed tomography (PET/CT) scans, cerebrospinal fluid cytology findings, the results of electrodiagnostic studies, as well as patient treatments and outcomes.

Results Ten NL patients were identified. All patients reported pain/paresthesia and weakness in the affected area. The MRI scans were abnormal in eight out of nine patients with an enhancement of the spinal nerve root, plexus, peripheral nerve trunk, and cranial nerve. The FDG PET/CT scans were positive in all patients. Radiculopathy or radiculoplexopathy was the most common electrodiagnostic finding. Neurological improvement was observed in only three patients. The condition of the nine patients who underwent multimodality treatments for cancer eventually deteriorated and the patients died.

Conclusions NL should be considered in the differential diagnosis of any type of neuropathy in patients with lymphoma. Because it could be confused with other neuropathies in lymphoma and various musculoskeletal diseases, a high index of suspicion and familiarity with clinical manifestation of NL are key. FDG PET/CT was the most sensitive diagnostic imaging modality to detect relevant neural invasion. The root within the spinal neural foramen was the most frequently affected neural structure. Early diagnosis of this rare neurologic manifestation of lymphoma may improve treatment outcomes.

Keywords Neurolymphomatosis · Lymphoma · Peripheral nervous system · FDG-PET/CT · MRI

Perineural cancer infiltration

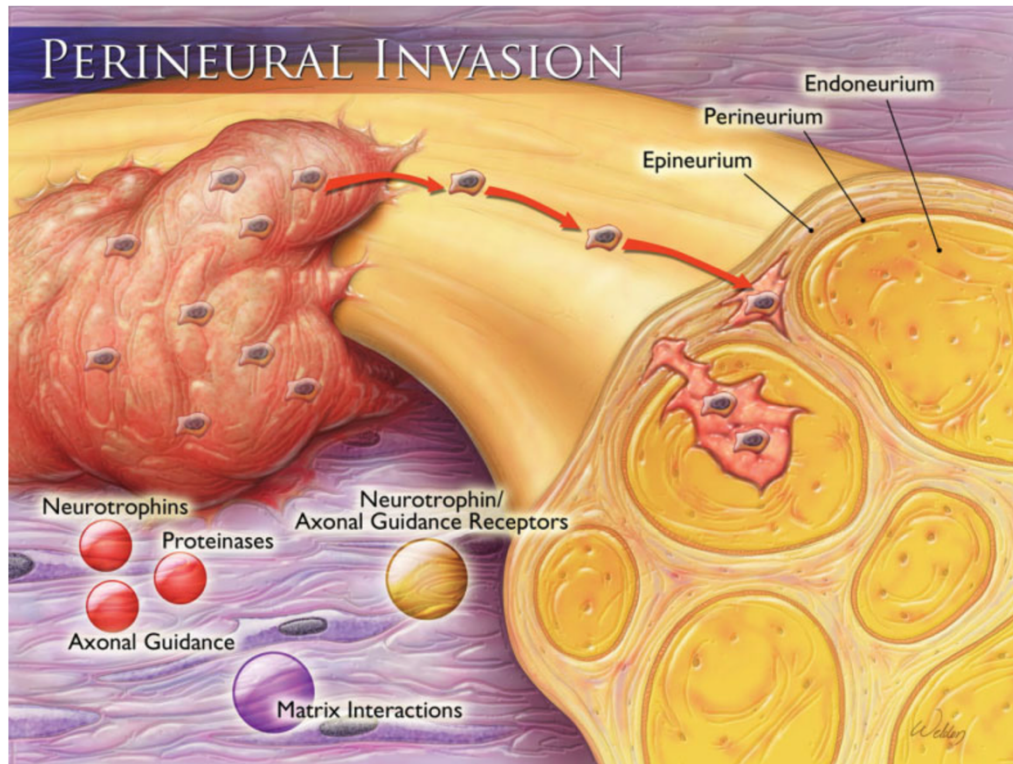


FIGURE 1. Perineural invasion: Illustrated is a tumor enveloping a peripheral nerve in cross section revealing tumor cell spread. Molecules expressed by tumors interact with surrounding stroma as well as receptors associated with peripheral nerves.

- PNI (perineural invasion) is a unrecognized route for cancer spread
- Maybe a significant as lymphovascular invasion
- Is differentiated from lymphatic metastases
- Significant in pancreas, prostate, biliary tract cancers

Perineural cancer infiltration

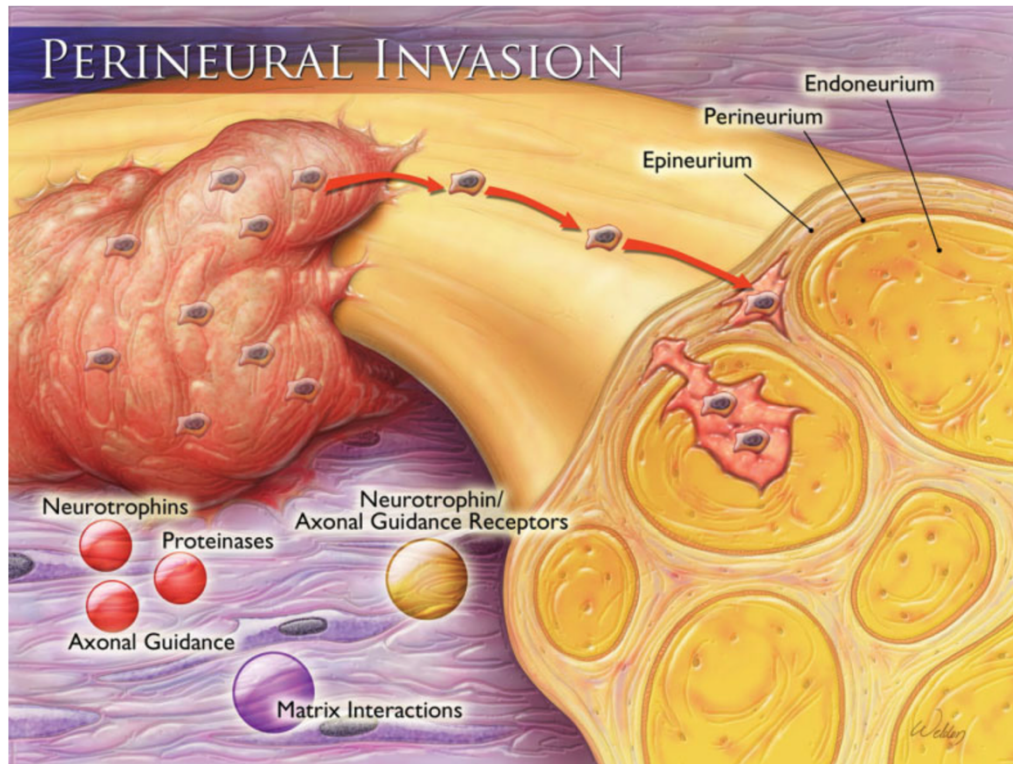


FIGURE 1. Perineural invasion: Illustrated is a tumor enveloping a peripheral nerve in cross section revealing tumor cell spread. Molecules expressed by tumors interact with surrounding stroma as well as receptors associated with peripheral nerves.

- All three levels of the supporting structures of the nerves are concerned: epineurium, perineurium, and endoneurium
- Axon growth is dependent on NGF, BDNF, neurotrophins all of which are over-expressed in certain cancers.
- Evidence of axon growth in tumors.
- Nerves serve as a conduit for intracranial extension

Perineural cancer infiltration

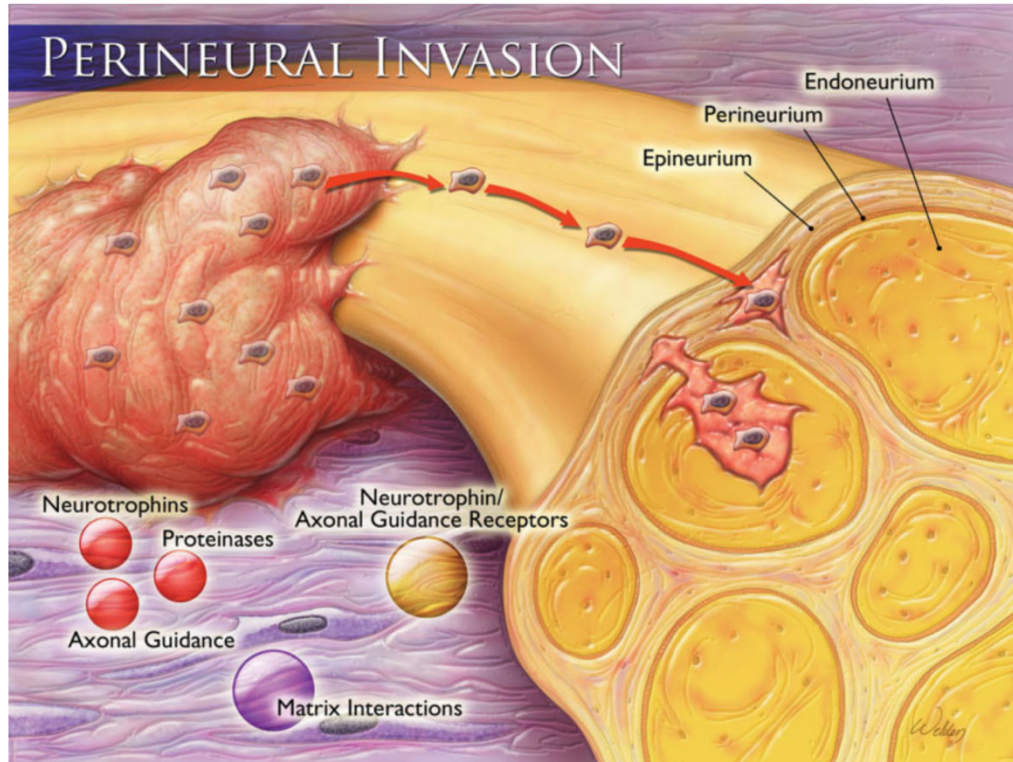


FIGURE 1. Perineural invasion: Illustrated is a tumor enveloping a peripheral nerve in cross section revealing tumor cell spread. Molecules expressed by tumors interact with surrounding stroma as well as receptors associated with peripheral nerves.

- Clinical examination: numbness and weakness.
- Panplexopathy, predominant of upper trunk and lower trunk
- Distinguishing radiation induced brachial flexography from perineural spread is difficult (myokymia?)
- Imaging:
 - MRI.
 - F-FDG PET

The spectrum of brachial plexopathy from perineural spread of breast cancer

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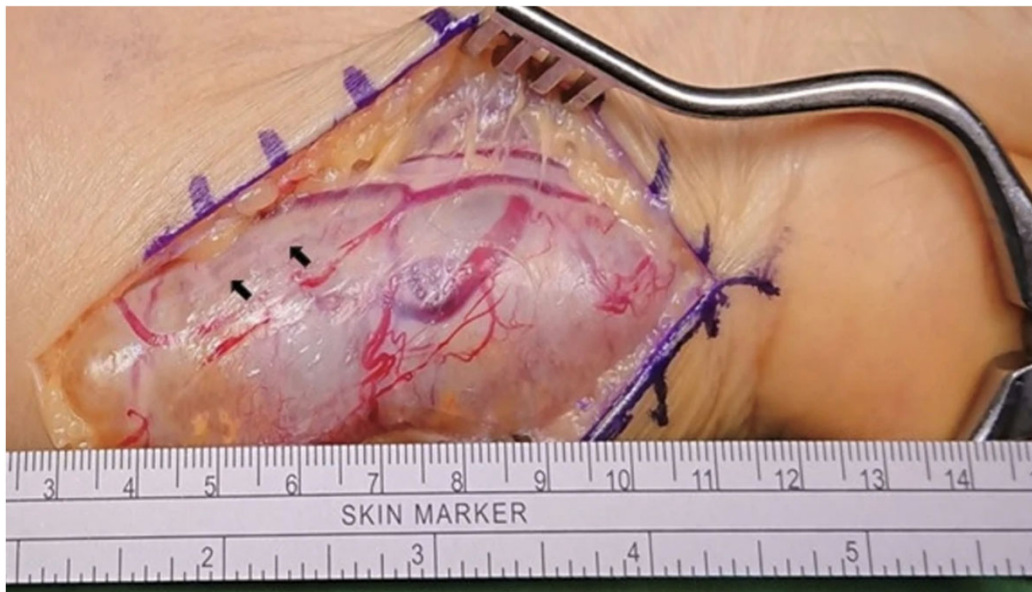
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OBJECTIVE Perineural spread of breast cancer to the brachial plexus can lead to pain, sensory alterations, and upper-extremity weakness. Although rare, perineural spread is an often-misdiagnosed long-term complication following breast cancer diagnosis. The objective of this study was to critically review the clinical, radiological, and pathological findings of biopsy-proven perineural spread of breast cancer to the brachial plexus.

METHODS This is a retrospective study from a single institution in which a total of 19 patients with brachial plexus involvement from perineural spread of breast cancer who underwent fascicular biopsy between 1999 and 2021 were identified. Clinical, radiographic, and pathological data were retrospectively collected. Descriptive statistics were calculated for the cohort.

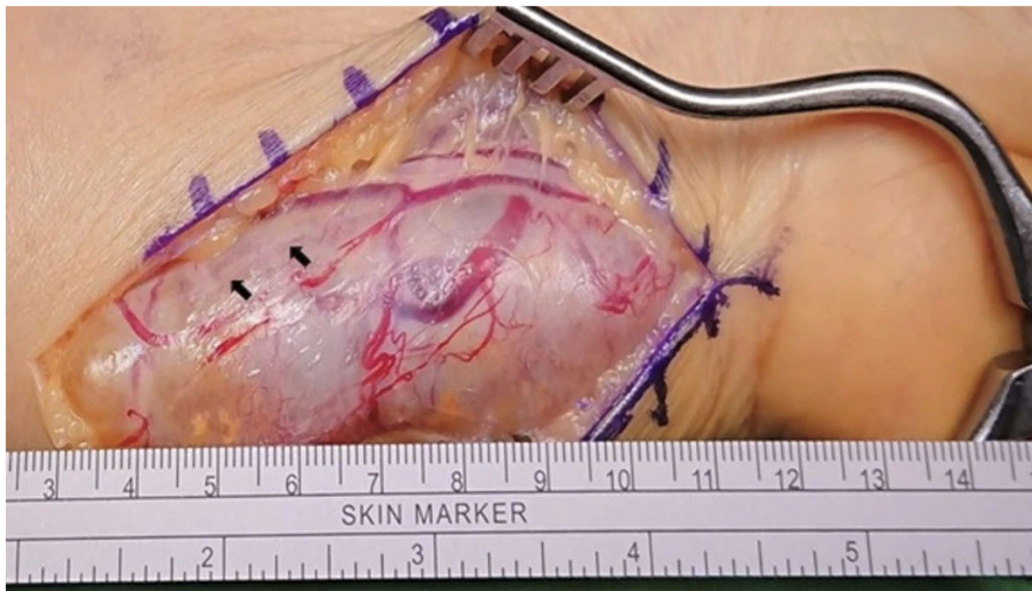
RESULTS The cohort consisted of 19 patients with biopsy-proven perineural spread of breast cancer to the brachial plexus. The median age was 62 years (range 45–78 years). The median time from breast cancer diagnosis to brachial plexopathy was 10 years (range 2–25 years). The most common clinical presentation was pain (100%), followed by sensory changes (89.5%), weakness (78.9%), and atrophy (42.1%). The most common radiological finding was a mass lesion (100%), followed by a cystic lesion (52.6%), and a solid lesion (47.4%). The most common pathological finding was perineural spread of breast cancer (100%), followed by a solid lesion (52.6%), and a cystic lesion (47.4%).

Schwannoma



- Being tumor of the Schwann cells
- Prevalence: 200,000 persons in US
- 4.4 to 5.2 per 100,000
- Incidence peaks between 50 y.o and 70 y.o.
- After 70 y.o. incidence can reach 21 per 100,000
- 60% of all schwannomas are vestibular (acoustic) schwannomas
- Can be NF-2

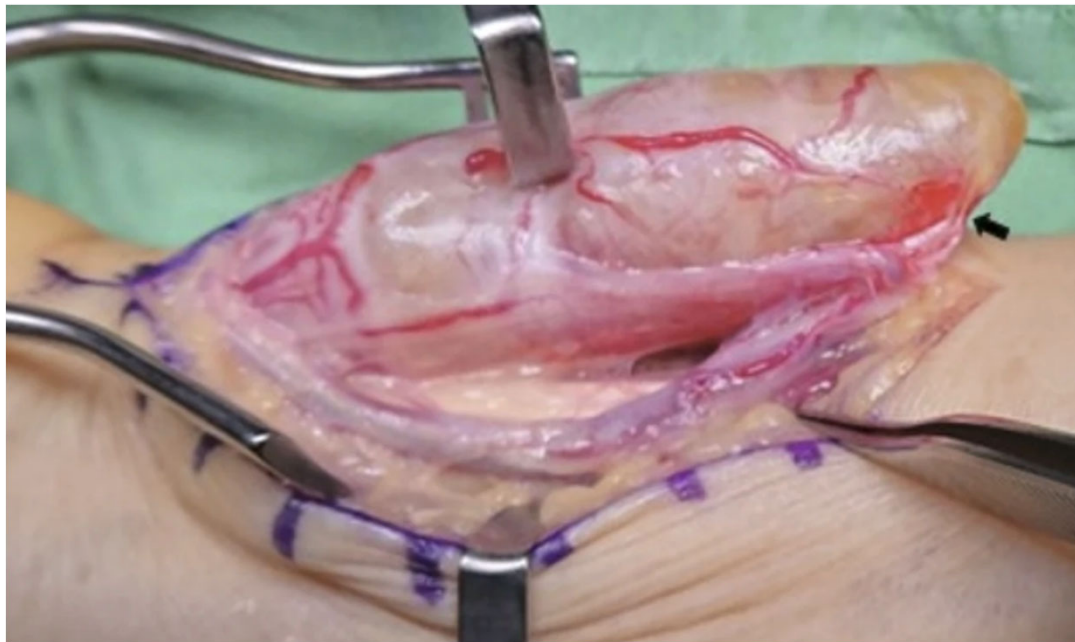
Schwannoma



Diagnostics:

- Vestibular schwannoma:
 - MRI
 - NCV/EMG: blink reflex
 - Videonystagmography
 - Audiology
- Other schwannomas:
 - HRUS
 - NCV/EMG

Schwannoma



Treatment:

- Surgery
- Radiation therapy

Case Report

Diagnostic Value of Preoperative Electrodiagnostic Analysis in a Patient with Facial Palsy and a Large Vestibular Schwannoma: Case Report

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Abstract: Although radiologic methods confirm the diagnosis of patients with large vestibular schwannomas, these methods usually indicate only the size of the tumor and its possible nerve compression. Electrodiagnostic methods can reveal the functional state of the nerves, particularly the trigeminal and facial nerves, as well as providing a basis for objectively evaluating nerve injury. Due to the lack of an established objective evaluation method, electrodiagnostic methods were utilized to assess injury to the cranial nerve in a patient with a large vestibular schwannoma. A 79-year-old woman presented with a one-month history of right facial palsy, vertigo, dizziness, right postauricular pain, and right-sided hearing disturbance. Physical examination suggested injuries to the facial and vestibulocochlear nerves. Magnetic resonance imaging identified a vestibular schwannoma and showed that the tumor mass was affecting the brainstem, including the fourth ventricle, resulting in mild obstructive hydrocephalus. Preoperative electrodiagnostic evaluation identified asymptomatic trigeminal neuropathy accompanying a vestibular schwannoma. The patient underwent surgery, consisting of a suboccipital craniotomy with additional gamma knife radiosurgery. Postoperatively, she demonstrated significant recovery from right facial palsy and partial improvement of her neurologic symptoms. Large vestibular schwannomas with facial paralysis may be accompanied by additional entrapment neuropathy. Routine preoperative electrophysiological evaluation is recommended to establish a definitive diagnosis and evaluate the function of the trigeminal nerve, facial nerve, and brainstem in patients with large and compressive vestibular schwannomas.

Keywords: vestibular schwannoma; electrodiagnostic study; blink reflex; needle electromyography



Citation: Yoo, M.C. Diagnostic Value of Preoperative Electrodiagnostic Analysis in a Patient with Facial Palsy and a Large Vestibular Schwannoma: Case Report. *Diagnostics* **2022**, *12*, 542. <https://doi.org/10.3390/diagnostics12020542>



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The role of preoperative ultrasound in the management of peripheral nerve injuries

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ARTICLE INFO

Keywords:

Peripheral nerve injuries
Preoperative
Ultrasound

ABSTRACT

Background: Peripheral nerve injury refers to any damage or trauma to the nerves located outside the central nervous system. Ultrasonography is a reliable, cheap, and minimally invasive method in clinical practice to give physicians useful information about nerve injury.

Objectives: to assess the power of ultrasound in determining the presence, localization, and extent of neural damage in patients with clinical evidence of peripheral nerve lesions before surgery.

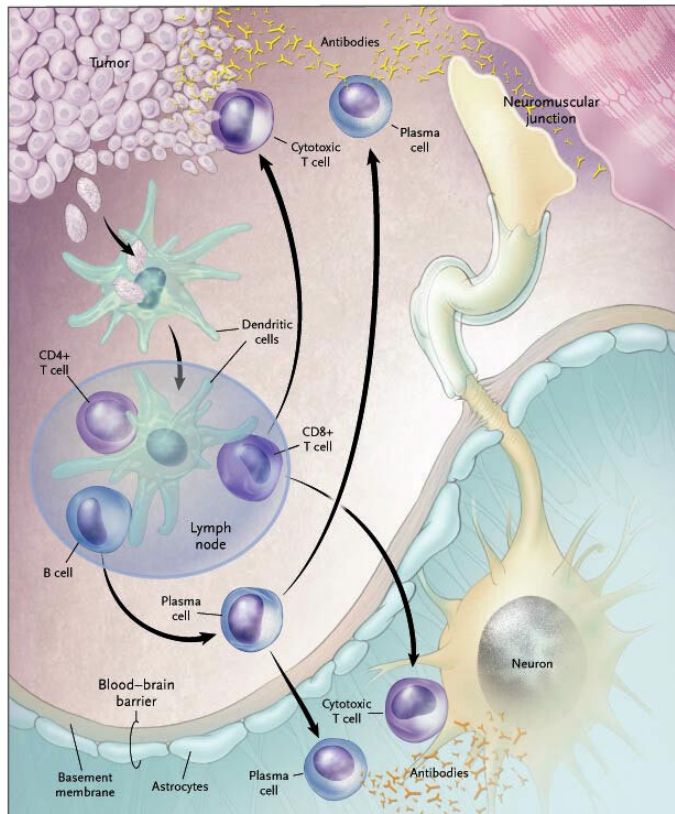
Methods: This cross-sectional study was conducted on 78 patients (56 females and 22 males, aged from 9 to 52 years) who had different pathologies including entrapment, tumoral, post-traumatic, and post-surgical nerve injuries at the Neurosurgery and Physical Medicine, Rheumatology, and Rehabilitation Departments, Tanta University Hospitals. All studied patients had preoperative evaluation; neurological examination, electrodiagnostic studies, and sonographic examinations with linear array transducers (frequencies ranging from 7.5 to 16 MHz).

Results: The most common pathological condition was entrapment neuropathy (39 patients) (50%). Ultrasound complemented the electrodiagnostic studies by determining the site of entrapment manifested by increased mean maximum cross-sectional area of the nerve proximal to the site of entrapment and nerve hypoechogenicity. In post-traumatic and iatrogenic neuropathies (35 patients) (44.9%), the ultrasound finding revealed neuroma in continuity in nine cases (11.5%), complete neurotmesis with stump neuroma in eighteen patients (23.1%), and eight cases (10.3%) showed perineural adhesion. In all cases, the nerve was hypoechoic at the site of injury. The presence of hyperechoic fibrous tissue could indicate perineural adhesion and the necessity for neurolysis. This study also included three (3.8%) cases had schwannoma, and one case (1.3%) had neurofibroma. Ultrasound was used to confirm the diagnosis by determining the tumor's size and vascular supply.

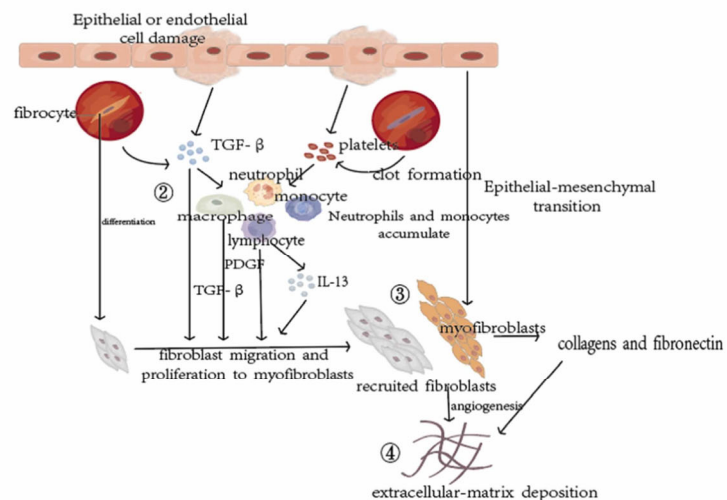
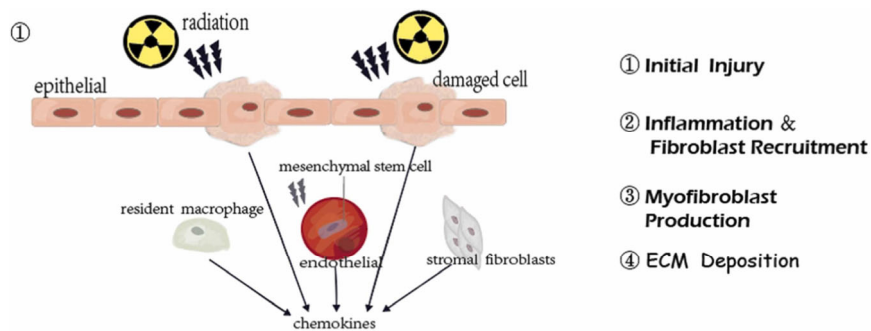
Conclusions: Ultrasonography is a diagnostic and surgical planning tool that is becoming more and more useful for the management of peripheral nerve injuries. Its high resolution and real-time capability provide safe and cost-effective scans that aid in determining the extent of injuries. For patients with peripheral nerve injuries, ultrasound is advised to be added to the routine clinical and neurophysiological evaluation. It is also advised to use ultrasound as a first-line imaging modality for tumors thought to be of nerve origin.

Paraneoplastic issues

Paraneoplastic neuropathies



- Can be quite diverse:
- Sensory neuronopathy
 - Reduced or absent SNAP
 - Normal CMAP/needle EMG
- Sensory motor neuropathy
 - Reduced SNAP and CMAP
 - Abnormal needle EMG
- Autonomic neuropathy
- LEMS



Paraneoplastic issues

Radiation induced neuropathies

Several phases:

- Inflammatory:
 - Ischemia, internal nerve edema, demyelination
- Organized fibrotic stage
 - Axon loss
- Degenerative fibrosis and destruction of normal tissue

Paraneoplastic issues

Radiation induced neuropathies

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Review

Mechanisms and protective measures for radiation-induced brachial plexus nerve injury

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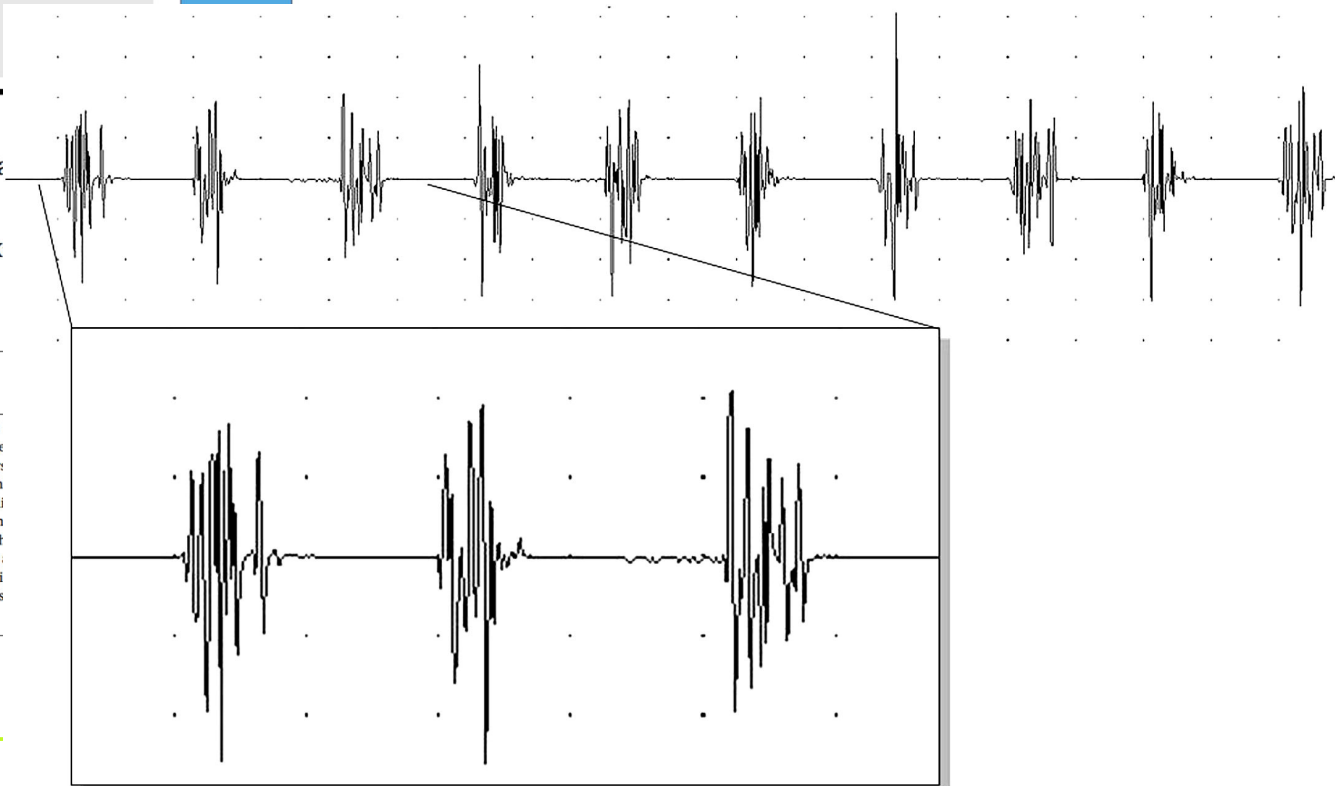
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ARTICLE INFO

Keywords:
Brachial plexus
Radiotherapy
RIBP
Nerve injury

ABSTRACT

Radiation therapy is a common treatment modality for patients with breast cancer in the chest and axilla. However, radiotherapy inevitably causes damage to the brachial plexus nerve (BP) which damage to the brachial plexus nerve (BP) is a serious adverse effect in the scapular or axillary regions, with clinical manifestations in the form of brachial plexus neuropathy (RIBP) which remains unclear. Current studies have shown that RIBP can be divided into two categories: direct injury and indirect injury, including inflammatory response, microvascular damage, cytokine production, and fibrosis. In this review, we summarize the underlying mechanisms and methods to prevent and treat RIBP.

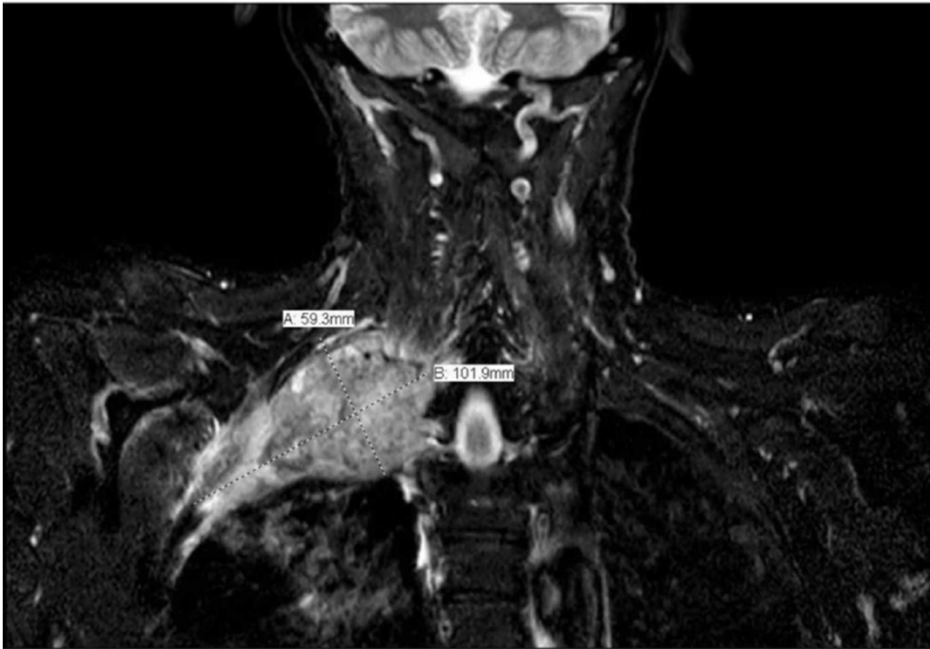


Paraneoplastic issues

Compressive neuropathies

Reminders:

- Progressive weakness and decreased sensation
- Pain may be absent
- Demographics and medical history is crucial
- Astute clinical examination is of utmost importance





When nerves and cancer cross wires

Take Home Message

ALAIN CLAUDEL, PT, DPT, ECS

A neuromuscular clinician's guide to magnetic resonance neurography

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Abstract

Magnetic resonance neurography (MRN) is increasingly used in clinical practice for the evaluation of patients with a wide spectrum of peripheral nerve disorders. This review article discusses the technical aspects of MRN highlighting the core sequences performed for clinical care. A robust, high-resolution, heavily T2-weighted fluid-sensitive sequence performed on a 3.0 Tesla magnet system remains the main workhorse MRN sequence. In specific clinical scenarios, adjunct techniques such as diffusion-weighted imaging can be added to a protocol for disease characterization. In addition, gadolinium-based contrast material can also be administered for the purposes of image optimization (suppress adjacent vascular signal) and disease characterization. Technical modifications to field of view and planes of imaging can be made based on the clinical question and discussion with the radiologist(s). On fluid-sensitive MRN sequences, a normal peripheral nerve exhibits iso- to minimally hyperintense signal relative to skeletal muscle with a predictable trajectory, preserved “fascicular” architecture, and tapered caliber from proximal to distal. Peripheral nerve abnormalities on MRN include alterations in signal, caliber, architecture, diffusion characteristics as well as enhancement and provide information regarding the underlying etiology. Although some MRN findings including nerve hyperintensity and long-segmental enlargement are nonspecific, there are certain diagnoses that can be made with high certainty based on imaging including benign peripheral nerve tumors, high-grade peripheral nerve injury, and intraneural ganglia. The purpose of this article is to familiarize a neuromuscular clinician with fundamentals of MRN acquisition and interpretation to facilitate communication with the neuromuscular radiologist and optimize patient care.

KEYWORDS

diffusion-weighted imaging (DWI), magnetic resonance neurography (MRN), peripheral nerve trauma, peripheral nerve tumors, peripheral neuropathy

Take Home Message Imaging in oncology

- MRI remains an important tool to assess cancers
- MRI of the peripheral nerves is developing
- Sequences:
 - T2
 - DWI for malignancies
 - STIR with gadolinium

Neuromuscular ultrasound: Impact on diagnosis and management

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Abstract

Introduction/Aim: High-resolution ultrasound (HRUS) is increasingly used in evaluating neuromuscular conditions. Its potential advantages include its ability to discern anatomic information and make specific etiological diagnoses. Although many studies have demonstrated HRUS effectiveness, especially in mononeuropathies, more information is needed to better determine how often and to what extent useful information is obtained; how it influences diagnosis, clinical decision-making, and patient management; and how it is used with electrodiagnostic (EDx) studies.

Methods: A retrospective cohort study was performed on patients referred for HRUS at a university laboratory during 2021. Demographic information, referral diagnoses, clinical information, HRUS findings, and follow-up patient management were analyzed. For patients who had EDx, results were compared with HRUS. Determinations were made whether HRUS did or did not aid in the diagnosis. For patients in whom HRUS resulted in a diagnosis, determination was made whether it confirmed the diagnosis made clinically or by EDx but did not change management; added additional important information; and/or made a decisive impact on subsequent management.

Results: Five hundred two patients were analyzed, most referred for mononeuropathy, brachial plexopathy, and polyneuropathy. HRUS was abnormal in 81.7% of patients. HRUS added additional useful information in 79.0% and was decisive in management in 62.7%. In patients who also had abnormal EDx, HRUS resulted in decisive management in 49.5%.

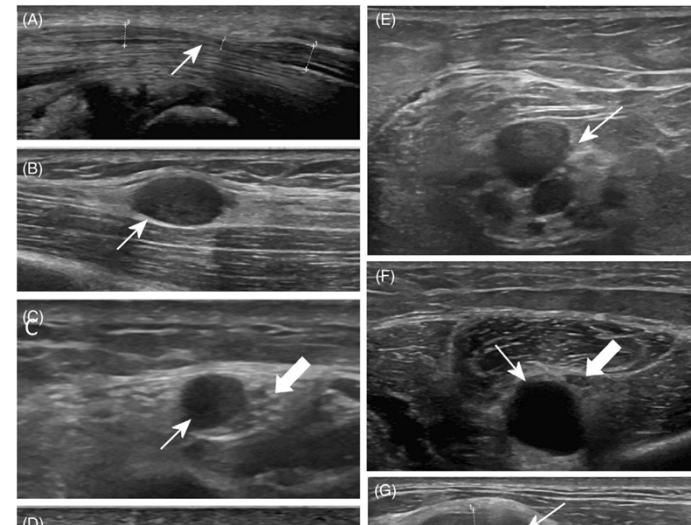
Discussion: HRUS is an effective diagnostic tool that frequently adds localizing and structural information that is otherwise not obtainable by clinical and EDx evaluation. In a substantial number of patients selected for HRUS, it is decisive in guiding further management.

Take Home Message High Resolution US

- Need I say more?

GASEMALTAYEB ET AL.

MUSCLE & NERVE WILEY 299



Take Home Message

NCV/EMG

- The PT Electromyographer may be the first to recognize pathology



A neuromuscular clinician's guide to magnetic resonance neurography

[Jennifer Pitman MD](#)
[Laura M. Fayed MD, MS](#)
[Shivani Ahlawat MD](#)
[Muscle & Nerve](#) Volume 71, Issue 3

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[Neuromuscular ultrasound: Impact on diagnosis and management](#)

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Kang YR, Hong SM, Choi JH, et al. Sensory neuropathy in a patient with neurofibromatosis type 1: A case report. *Medicine (Baltimore)*. 2022;101(45):e31718. doi:10.1097/MD.00000000000031718

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ATYPICAL PRESENTATION OF DEGENERATIVE CERVICAL MYELOPATHY: A REVIEW AND TWO CASE REPORTS

Shaffer S

ABSTRACT

BACKGROUND: The diagnosis and treatment of degenerative cervical myelopathy (DCM) is frequently delayed resulting in neurologic deterioration, significant activity limitations, and a marked reduction in quality of life.¹⁻⁴ Timely and accurate assessment is challenging secondary to the non-specific and diverse symptoms associated with DCM and often further complicated by overlapping age-related health conditions (e.g., radiculopathy, carpal tunnel syndrome, polyneuropathy).⁵⁻⁶ The purpose of this presentation is to highlight the diversity of symptoms in patients with degenerative cervical myelopathy and the contributions of electrophysiologic testing and imaging.

DISCUSSION: This presentation will provide an evidence informed review and discuss two patient reports that highlight typical and atypical (i.e., pseudoathetosis, tremor, vertigo, palpitations) clinical examination findings, electrophysiological testing, imaging, and surgical outcomes for patients with degenerative cervical myelopathy (DCM).

CONCLUSIONS: Collectively, these cases emphasize the importance of a detailed clinical examination and the benefits of electrophysiological testing and imaging (cervical and brain) for successful management of patients presenting with both typical and atypical DCM.

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Atypical Presentation of Degenerative Cervical Myelopathy: A Review & Two Case Reports



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Objectives

- 1) Describe the pathophysiology associated with degenerative cervical myelopathy.
- 2) Discuss the typical and atypical historical and physical examination findings in patients with degenerative cervical myelopathy.
- 3) Describe an evidence based clinical and electrophysiologic exam (EMG/NCS) and suspected findings for a patient with suspected cervical myelopathy.
- 4) Discuss the current outcomes for patients with atypical symptoms of degenerative cervical myelopathy undergoing cervical decompression and fusion.

Agenda

Pathophysiology and Incidence of degenerative cervical myelopathy (DCM)

Typical and atypical historical and physical examination findings associated with degenerative cervical myelopathy (DCM)

Case Study #1- A patient with lower extremity neuropathic symptoms and dizziness

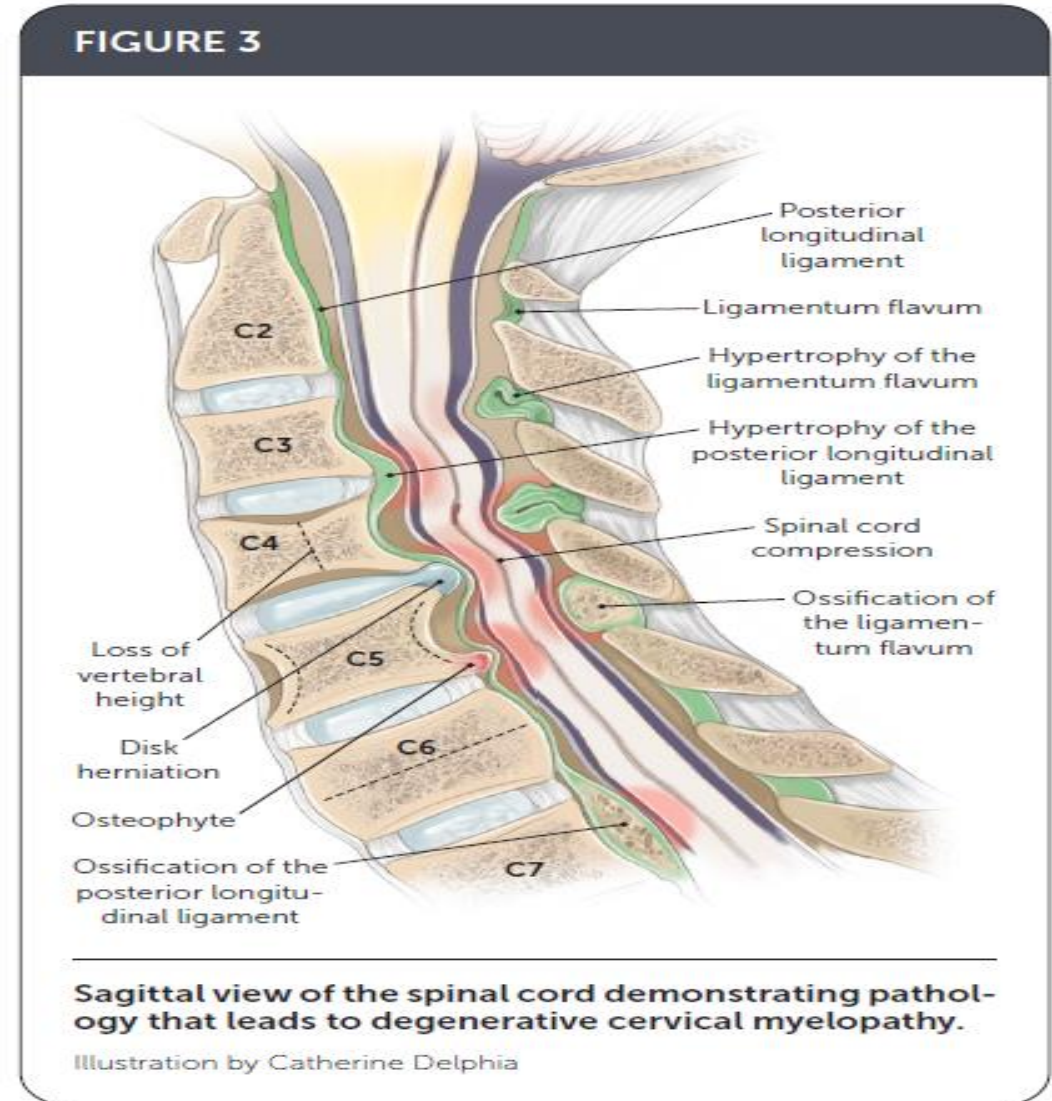
Case Study #2- A patient with athetosis and diffuse sensory loss

Surgical outcomes in patients with atypical symptoms and cervical decompression/fusion.

Conclusions and questions/answers

Degenerative (Spondylotic) Cervical Myelopathy (DCM)

- Incidence- 3.5 per 1000 cases and most common cause of spinal cord dysfunction worldwide
- Delayed Diagnosis(DX)/Treatment: DX 2.2 years, 5.2 Physician Visits to DX
- Most Common Levels- C5-7 for pathology
- Typically, Slow Progression Over Decades
- Pathology Static Factors:
 - Congenital Stenosis
 - Degenerative Disc Disease- 12-17% (age 20's); 86-89% (age 60's)
 - Hypertrophy/Ossification Ligamentum Flavum & Posterior Longitudinal Ligament
 - Vertebral body osteophytes
- Pathology Dynamic Factors
 - Degenerative spondylolisthesis
 - Physiologic narrow of the canal
 - Strain/Stretch on spinal cord with movement



Typical vs Atypical Symptoms DCM

Typical

- Neck/scapular pain
- Diffuse pain, paresthesia, and weakness (upper extremities > lower extremities)
- Decreased hand dexterity
- Gait/Balance Impairment
- Bladder dysfunction

Atypical

- Headache
- Vertigo
- Blurred Vision
- Tinnitus
- Palpitations
- Movement disorders (Tremor)

Frequency of Symptoms DCM (N=58 studies/Weighted Sensitivity (3 Studies)

Symptom	Frequency	Sensitivity
	% Range	% (95% CI)
Hand numbness	21 to 89	82 (80 to 85)
Hand paresthesia	24 to 93	79 (68 to 87)
Upper extremity numbness	4 to 96	69 (66 to 72)
Hand clumsiness	26 to 90	69 (67 to 72)
Upper extremity weakness	4 to 92	58 (55 to 60)
Upper extremity paresthesia	29 to 70	57 (54 to 60)
Neck/shoulder pain	9 to 100	51 (49 to 53)
Upper limb pain	10 to 54	43 (40 to 46)
Fine motor disturbance	22 to 71	29 (25 to 33)
Hand weakness	4 to 18	10 (3 to 24)
Gait dysfunction	10 to 100	72 (70 to 74)
Lower extremity numbness	17 to 91	61 (57 to 66)
Lower extremity weakness	3 to 81	54 (51 to 57)
Gait imbalance	4 to 25	23 (19 to 27)
Back pain	9 to 22	19 (14 to 27)
Unspecified paresthesia	85 to 92	86 (82 to 90)
Axial pain	19 to 100	41 (35 to 46)
Radicular pain	7 to 96	39 (35 to 42)

Atypical Symptoms DCM (Yuan et al. N=380 typical= 214 atypical=166)

- Vertigo (37%)
- Headache (18%)
- Blurred Vision(15%)
- Tinnitus (11%)
- Palpitations (11%)
- Movement disorders (Tremor, Apraxia- Case series)

Frequency/Specificity/Sensitivity (72 Studies) Signs/Tests for DCM

Sign	Frequency	Sensitivity	Specificity
	% Range	% (95% CI)	% (95% CI)
Tromner's	NR	94 (86 to 98)	93 (81 to 99)
Hyperreflexia (generalized)	33 to 100	72 (55 to 85)	43 (27 to 61)
Hoffman's	21 to 100	58 (53 to 63)	72 (68 to 76)
Motor impairment	9 to 100	54 (53 to 56)	64 (63 to 65)
Sensory impairment	19 to 100	48 (41 to 55)	66 (56 to 76)
Inverted supinator	NR	41 (34 to 48)	93 (89 to 96)
Babinski's	11 to 100	17 (11 to 23)	99 (97 to 100)
Clonus	3 to 73	9 (5 to 15)	99 (96 to 100)

Abbreviation: NR = not reported.

Hoffman's Vs Tromner's

It's in the stretch and direction 😊



Case 1

History-

- 60 y/o RHD male with c/c of LBP (11 years) with increased symptoms and diffuse radiating pain, tingling, and weakness in the bilateral (right>left) thighs, legs, feet, and toes by 2 years.
- He has neck “stiffness” with intermittent and diffuse numbness/tingling, “muscle cramps”, and “tremors” in the bilateral (right >left) hands and fingers by 2 years. He dropping objects and hard to button clothes with his right hand.
- Increased symptoms over the past 6 months.
- Negative recent trauma; Increased BUE/BLEs symptoms with prolonged sitting and reading.
- Decreased balance but no falls in the past year. Using a cane over the past 6 months.
- Denies n/t or weakness in the face, visual changes, bowel/bladder incontinence, or problems with swallowing/chewing/speaking
- PMHX: high blood pressure, high cholesterol, gout. PSHX: left L5-S1 minimally invasive L4-S1 decompression-2014
- No PMHX of diabetes, thyroid or renal disease, ETOH misuse or exposure to toxins
- No family history of neuromuscular disorders
- He is not taking anticoagulants

Clinical Exam

- Cranial nerves: 2-8; 11-12 intact. Negative fasciculations or atrophy in the BUE/BLEs, or tongue.
- **High frequency low amplitude tremor bilateral hands/fingers; increased with holding/fatigue.** Negative resting tremor.
- **Gait: Independent with single point case with ataxia (Improved with looking down).**
- **Strength-** 5/5 bilateral C5-T1 & L1-S2 myotomes **except right thumb abduction 4+/5 MMT, right elbow extension, right wrist flexion/radial deviation, left great toe extension and left ankle plantarflexion 4/5 MMT.**
- **MSRs:** 2+ bilateral biceps, brachioradialis, triceps, **3+ right patellar**, 2+ left patellar, **3+ right Achilles**, 2+ left Achilles reflexes.
- **Pathologic Reflexes:** **Present bilateral Hoffman, 5-6 beat right ankle clonus, Present right Babinski sign.** Absent left ankle clonus, left Babinski sign, & bilateral wrist clonus.
- **Sensory:** Bilateral C4-T1 and L1-S2 dermatomes & respective nerves intact light touch(LT)/pinprick (PP) **except decreased LT right dorsal/palmar thumb, index, middle fingers and PP decreased at the bilateral dorsal/palmar thumbs, index, middle, little fingers.**
- **Vibration absent right great toe** (Present at the medial ankle) and >10 secs left great toe.
- **Diminished 10 gram monofilament right great toe, Absent 2.83 and diminished 3.22 (2/5 trials) monofilament at the right palmar/dorsal thumb and middle fingers.**
- **Special Tests:** Negative bilateral Spurling's (**Limited neck extension/flexion/rotation with stiffness**) Negative bilateral Slump Tests. Negative bilateral Tinel's at the bilateral carpal or cubital tunnels. Negative bilateral Phalen's or elbow hyperflexion tests.

Essential Tremor



UMN/PP Loss Vibration/Touch

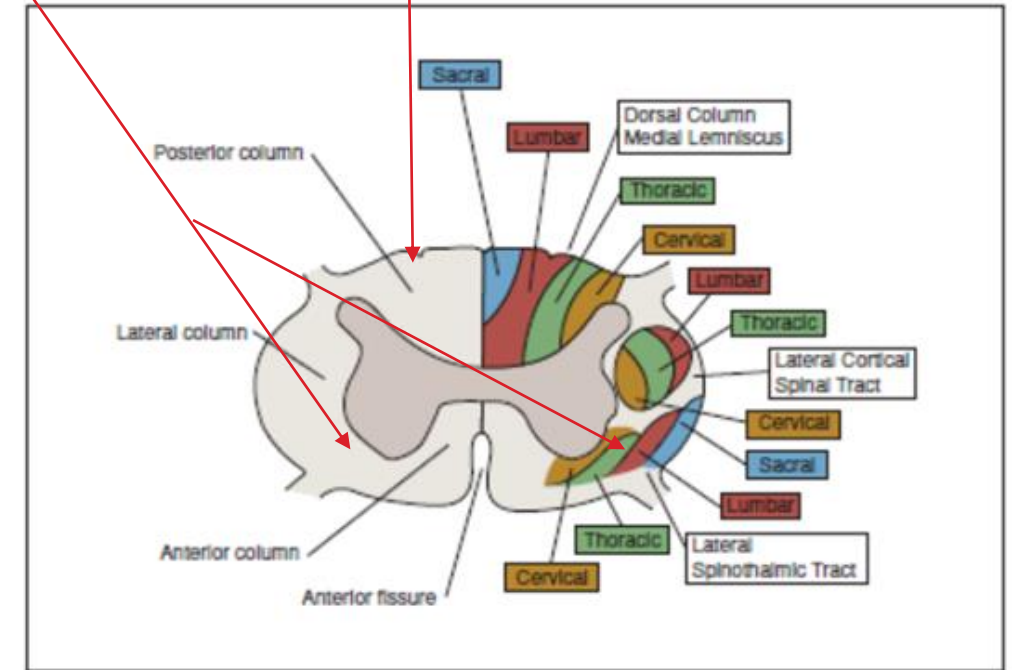


Figure 3-4 Spinal Cord Somatotopic Organization.

Sensory Nerve Conduction Studies

Stim Site	NR	Peak (ms)	Norm Peak (ms)	P-T Amp (µV)	Norm P-T Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median 2nd digit Anti Sensory (2nd Digit)											
Palm		1.9	<2.2	31.9	>15	Palm	2nd Digit	1.9	7.0	37	
Wrist		3.4	<3.6	31.4		Wrist	Palm	1.5	7.0	47	
Right Median 2nd digit Anti Sensory (2nd Digit)											
Palm		1.8	<2.2	14.8	>15	Palm	2nd Digit	1.8	7.0	39	
Wrist		4.9	<3.6	10.6		Wrist	Palm	3.1	7.0	23	
Left Radial Anti Sensory (Base 1st Digit)											
Wrist		2.0	<2.8	33.3	>10	Wrist	Base 1st Digit	2.0	10.0	50	
Right Radial Anti Sensory (Base 1st Digit)											
Wrist		1.8	<2.8	25.7	>10	Wrist	Base 1st Digit	1.8	10.0	56	
Left Ulnar 5th digit Anti Sensory (5th Digit)											
Wrist		3.1	<3.5	32.9	>10.0	Wrist	5th Digit	3.1	14.0	45	
Right Ulnar 5th digit Anti Sensory (5th Digit)											
Wrist		3.1	<3.5	26.4	>10.0	Wrist	5th Digit	3.1	14.0	45	

Motor Nerve Conduction Studies

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Motor (Abd Poll Brev)											
Wrist		3.9	<4.2	6.0	>5	Elbow	Wrist	5.0	27.0	54	>50
Elbow		8.9		6.1							
Right Median Motor (Abd Poll Brev)											
Wrist		6.3	<4.2	5.5	>5	Elbow	Wrist	5.1	27.0	53	>50
Elbow		11.4		5.4							
Left Ulnar Motor (Abd Dig Minimi)											
Wrist		3.0	<3.6	7.8	>3	B Elbow	Wrist	4.3	23.0	54	>50
B Elbow		7.3		7.4		A Elbow	B Elbow	1.7	10.0	59	>50
A Elbow		9.0		7.1							
Right Ulnar Motor (Abd Dig Minimi)											
Wrist		3.1	<3.6	9.8	>3	B Elbow	Wrist	4.2	23.0	55	>50
B Elbow		7.3		9.3		A Elbow	B Elbow	1.7	10.0	59	>50
A Elbow		9.0		8.9							

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Triceps	Radial	C6-7-8	Incr	1+	2+	Nml	Nml	2+	Reduced	Nml	
Right	Biceps	Musculocut	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	PronatorTeres	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	FlexCarRad	Median	C6-7	Incr	1+	2+	Nml	Nml	2+	Reduced	Nml	
Right	ExtDigCom	Radial (Post Int)	C7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Abd Poll Brev	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Biceps	Musculocut	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	PronatorTeres	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	FlexCarRad	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	ExtDigCom	Radial (Post Int)	C7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Abd Poll Brev	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Cervical Parasp Mid	Rami	C4-6	Nml	Nml	Nml						
Right	Cervical Parasp Low	Rami	C7-8	Nml	Nml	Nml						
Left	Cervical Parasp Mid	Rami	C4-6	Nml	Nml	Nml						
Left	Cervical Parasp Low	Rami	C7-8	Nml	Nml	Nml						

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	AntTibialis	Dp Br Fibular	L4-5	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Fibularis Longus	Sup Br Fibular	L5-S1	Nml	Nml	Nml	Nml	Nml	0	<u>Nml</u>	Nml	
Right	PostTibialis	Tibial	L5, S1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Gastroc	Tibial	S1-2	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	VastusMed	Femoral	L2-4	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	TensorFascLat	SupGluteal	L4-5, S1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	AntTibialis	Dp Br Fibular	L4-5	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Fibularis Longus	Sup Br Fibular	L5-S1	Nml	Nml	Nml	Nml	Nml	2+	Reduced	75%	
Left	PostTibialis	Tibial	L5, S1	Nml	Nml	Nml	Nml	Nml	2+	Reduced	75%	
Left	Gastroc	Tibial	S1-2	Nml	Nml	Nml	Nml	Nml	2+	Reduced	Nml	
Left	VastusMed	Femoral	L2-4	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	TensorFascLat	SupGluteal	L4-5, S1	Nml	Nml	Nml	Nml	Nml	2+	Reduced	Nml	
Right	Lumbo Parasp Up	Rami	L1-2	Nml	Nml	Nml						
Right	Lumbo Parasp Mid	Rami	L3-4	Nml	Nml	Nml						
Right	Lumbo Parasp Low	Rami	L5-S1	Nml	Nml	Nml						
Left	Lumbo Parasp Up	Rami	L1-2	Nml	Nml	Nml						
Left	Lumbo Parasp Mid	Rami	L3-4	Nml	Nml	Nml						
Left	Lumbo Parasp Low	Rami	L5-S1	Nml	Nml	Nml						
Left	VastusLat	Femoral	L2-4	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	BicepsFemL	Sciatic	L5-S2	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	VastusLat	Femoral	L2-4	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	BicepsFemL	Sciatic	L5-S2	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	

Impression

1. Chronic right C7 radiculopathy with ongoing axonal loss and reinnervation potentials in the right triceps and flexor carpi radialis. Recommend correlation with MRI.
2. Chronic left L5-S1 radiculopathy without ongoing axonal loss. Specifically, findings included reinnervation potentials in the tensor fascia lata, gastrocnemius, fibularis longus, and posterior tibialis. Recommend correlation with MRI.
3. Moderate right median sensorimotor myelinopathy and sensory fiber conduction block at or about the wrist.

The patient also exhibited clinical findings of bilateral Hoffman signs, right ankle clonus, right Babinski sign, and right lower extremity hyperreflexia potentially suggesting upper motor neuron (CNS) involvement. Recommend correlation with MRI and clinical examination.

Impression

There is no definitive electrodiagnostic evidence of right C5-6 or C8-T1 radiculopathy, left C5-T1 radiculopathy, left L2-4 radiculopathy, right L2-S2 radiculopathy, polyneuropathy, myopathy, motor neuron disease, bilateral brachial plexus axonopathy, isolated left median mononeuropathy, or bilateral ulnar, superficial radial, tibial or fibular mononeuropathies. Recommend correlation with MRI.

Patient's Imaging

Cervical Spine MRI

- C4-5: mild central HNP with stenosis that abuts the cord
- **C5-6: Mild central HNP/stenosis that abuts the cord; mild to moderate right foraminal stenosis**
- **C6-7 Moderate central HNP/stenosis that compresses cord; Moderate right foraminal stenosis**
- C3-4 and C7-T1 no central or foraminal stenosis.



MRI Imaging- Cervical Spine Cord Grading Scale

Grade	Severity	Description
Grade 0	Normal	There is absence of central canal stenosis.
Grade 1	Mild	The subarachnoid space is near completely obliterated, including obliteration of the arbitrary subarachnoid space exceeding 50%, without signs of cord deformity.
Grade 2	Moderate	The spinal cord is compressed and deformed but without cord signal change.
Grade 3	Severe	There is signal change of the spinal cord near the compressed level on T2-weighted images.

Follow-up

- **Brain MRI:** WNL
- **Lumbar MRI:** moderate bilateral L4-S1 foraminal stenosis
- **C5-7 ACDF 6 weeks after NCS/EMG.**
- **Day of the procedure:** Somatosensory Evoked Potentials(SSEP) conducted and unable to obtain potentials in the bilateral upper extremities and right lower extremity.
- **6 months post-operative:** Self-report improvement in **bilateral lower extremity pain, weakness, tingling**; symptoms (**muscles cramps, sensation, tremor, dropping objects**) in the bilateral hands/fingers; **no longer using cane on level surfaces.**
- Follow-up 2 months to reassess right hand symptoms

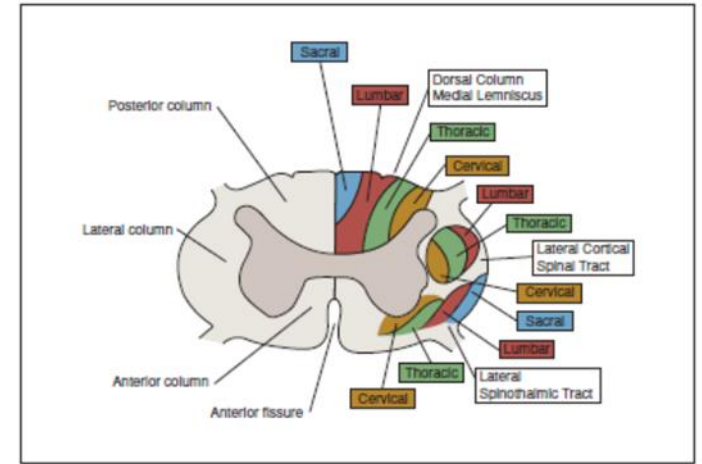


Figure 3-4 Spinal Cord Somatotopic Organization.

Discussion/Lessons Learned

- Atypical Symptoms- Resting tremor, lower extremity symptoms
> upper extremity symptoms
- Use of Monofilaments not limited to CTS and polyneuropathy?
- Value of Somatosensory Evoked Potentials

Case 2

History-

- 64 y/o RHD male reports with mild neck pain (3/10) and significant (7/10) and diffuse burning pain, numbness and weakness in bilateral forearms, hands, fingers, and thumbs by 6 months. Symptoms started after a fall onto his hands and knees.
- He also has diffuse radiating pain and numbness/tingling in his **trunk** (anterior/posterior) and bilateral lower extremities to his toes. He denies weakness in the BLEs.
- He denies n/t or weakness in the face but has periodic **occipital headaches and dizziness/light headedness** since initial fall
- Decreased balance with 4 falls in the past 6 months
- No problems with vertigo, vision, chewing/swallowing/speaking, saddle n/t, or bowel/bladder incontinence
- PMHX: High cholesterol, Diabetes (2 years; Last A1C=6.3%). No past surgeries
- Patient denies past medical history of thyroid or renal disease, ETOH misuse, or exposure to toxins
- Negative past family history of progressive neuromuscular disorders
- The patient is not taking blood thinners

Clinical Exam

- Cranial nerves: 2-8; 11-12 intact.
- **Observation: Continuous slow, involuntary, rhythmic and asynchronous movements of the fingers and thumb- Decreased with visual cueing.** Negative atrophy, resting tremor, intention tremor, or fasciculations in the BUEs, BLEs, or tongue
- **Gait:** Independent with cane with mild ataxia. Using scooter/wheelchair for community ambulation/safety.
- **Strength-** 5/5 MMT bilateral C4-T1 and L2-S2 myotomes except **bilateral finger adduction/abduction/flexion/extension and thumb flexion/extension 4/5 MMT.** Difficulty with fine motor control thumb to fingers.
- **MSR:** 2+ bilateral biceps, 2+ brachioradialis, **3+ triceps, 3+ patellar,** and 2+ Achilles reflexes.
- **Pathologic Reflex:** Absent bilateral Hoffman signs, Babinski signs, invertor supinator signs, and ankle/wrist clonus.
- **Sensory:** Bilateral C4-T1 dermatomes & respective nerve fields intact to light touch(LT)/pinprick (PP) **except decreased LT and PP distal to the bilateral elbow. Vibration absent distal to the mid forearms. 2/5 trials incorrect thumbs up/down.** Vibration bilateral great toes and 10 gram monofilament 5/5 correct.
- **Special Tests:** **Mild (2/10) neck pain and decreased bilateral cervical rotation (20-25 degrees).** Negative Tinel's at the bilateral carpal or cubital tunnels. **Positive right Phalen's and right elbow hyperflexion test.** Negative left Phalen's or elbow hyperflexion test.

Sensory Nerve Conduction Studies

Stim Site	NR	Peak (ms)	Norm Peak (ms)	P-T Amp (µV)	Norm P-T Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median 2nd digit Anti Sensory (2nd Digit)											
Palm		1.7	<2.2	17.7	>15	Palm	2nd Digit	1.7	7.0	41	
Wrist		3.3	<3.6	23.7		Wrist	Palm	1.6	7.0	44	
Right Median 2nd digit Anti Sensory (2nd Digit)											
Palm		1.9	<2.2	16.8	>15	Palm	2nd Digit	1.9	7.0	37	
Wrist		3.7	<3.6	19.7		Wrist	Palm	1.8	7.0	39	
Left Radial Anti Sensory (Base 1st Digit)											
Wrist		1.9	<2.8	56.6	>10	Wrist	Base 1st Digit	1.9	10.0	53	
Right Radial Anti Sensory (Base 1st Digit)											
Wrist		1.8	<2.8	30.2	>10	Wrist	Base 1st Digit	1.8	10.0	56	
Left Ulnar 5th digit Anti Sensory (5th Digit)											
Wrist		3.1	<3.5	47.2	>10.0	Wrist	5th Digit	3.1	14.0	45	
Right Ulnar 5th digit Anti Sensory (5th Digit)											
Wrist		3.1	<3.5	39.4	>10.0	Wrist	5th Digit	3.1	14.0	45	

Motor Nerve Conduction Studies

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Motor (Abd Poll Brev)											
Wrist		3.8	<4.2	7.0	>5	Elbow	Wrist	4.7	24.0	51	>50
Elbow		8.5		6.9							
Right Median Motor (Abd Poll Brev)											
Wrist		3.9	<4.2	7.1	>5	Elbow	Wrist	4.6	24.0	52	>50
Elbow		8.5		7.0							
Left Ulnar Motor (Abd Dig Minimi)											
Wrist		3.1	<3.6	8.3	>3	B Elbow	Wrist	3.9	22.0	56	>50
B Elbow		7.0		7.6		A Elbow	B Elbow	1.5	10.0	67	>50
A Elbow		8.5		7.7							
Right Ulnar Motor (Abd Dig Minimi)											
Wrist		3.0	<3.6	8.7	>3	B Elbow	Wrist	3.6	22.0	61	>50
B Elbow		6.6		8.4		A Elbow	B Elbow	2.5	10.0	40	>50
A Elbow		9.1		6.8		Axilla	A Elbow	1.4	10.0	71	
Axilla		10.5		6.8							

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Biceps	Musculocut	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	PronatorTeres	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	FlexCarRad	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	ExtDigCom	Radial (Post Int)	C7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Abd Poll Brev	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Biceps	Musculocut	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	PronatorTeres	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	FlexCarRad	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	ExtDigCom	Radial (Post Int)	C7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Abd Poll Brev	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Cervical Parasp Mid	Rami	C4-5	Incr	2+	2+						
Right	Cervical Parasp Low	Rami	C6-8	Nml	Nml	Nml						
Left	Cervical Parasp Mid	Rami	C4-6	Nml	Nml	Nml						
Left	Cervical Parasp Low	Rami	C7-8	Nml	Nml	Nml						
Right	FlexDigProf	Ulnar	C8,T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Trapezius	SpinalAcc	CN XI, C3-4	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Trapezius	SpinalAcc	CN XI, C3-4	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	T1 Parasp	Rami	T1	Nml	Nml	Nml						
Right	T1 Parasp	Rami	T1	Nml	Nml	Nml						
Left	Cervical Parasp Up	Rami	C1-3	Nml	Nml	Nml						
Right	Cervical Parasp Up	Rami	C1-3	Nml	Nml	Nml						

Impression

There is electrodiagnostic evidence most consistent with:

1. **Cervical radiculopathy on the right (posterior primary rami axonopathy).** This was evidenced by abnormal electromyographic activity recorded from right mid cervical (C4-5) paraspinal musculature. It is not possible to clearly determine the nerve root level(s) involved due to normal findings in limb musculature and myotomal overlap in the paraspinals. Recommend correlation with MRI.
2. **Mild right median sensory mononeuropathy (myelinopathy) at or about the wrist.**
3. **Mild right ulnar mononeuropathy (motor fiber myelinopathy/conduction block) at or about the elbow.**

The patient also exhibited clinical findings of hyperreflexia in the bilateral upper/lower extremities as well as athetoid movements in the bilateral fingers suggesting potential upper motor neuron (CNS) involvement. Recommend correlation with MRI (cervical spine, Brain) and clinic exam.

Impression

- There is no definitive electrodiagnostic evidence of left C5-T1 radiculopathy in the LUE or C2-T1 paraspinals, right C5-T1 radiculopathy in the RUE or C6-T1 paraspinals, bilateral spinal accessory mononeuropathies, bilateral brachial plexus axonopathy, or isolated left median, left ulnar, or bilateral superficial radial mononeuropathies. Recommend correlation with MRI.

Patient's Imaging

Cervical Spine MRI

- C2-3: Autofusion
- **C3-4: HNP with severe central canal stenosis with myelomalacia and moderate to severe right foraminal stenosis**
- **C4-5: HNP with moderate central stenosis and moderate right > left foraminal stenosis**
- C5-6: HNP with moderate central stenosis and moderate left foraminal stenosis
- C6-7 HNP with moderate left > right foraminal stenosis
- C7-T1 mild HNP without central or foraminal stenosis.



MRI Imaging- Cervical Spine Cord Grading Scale

Grade	Severity	Description
Grade 0	Normal	There is absence of central canal stenosis.
Grade 1	Mild	The subarachnoid space is near completely obliterated, including obliteration of the arbitrary subarachnoid space exceeding 50%, without signs of cord deformity.
Grade 2	Moderate	The spinal cord is compressed and deformed but without cord signal change.
Grade 3	Severe	There is signal change of the spinal cord near the compressed level on T2-weighted images.

After EMG & Follow-up

- Brian MRI: WNL (mild microvascular change-white matter)
- **Posterior** decompression/fusion C4-5 and **anterior** decompression/fusion C3-6 three weeks after NCS/EMG
- **8 week follow-up**: UE pain ↓(7 to 3 NPRS); ↑strength/sensation in the forearm/hands/fingers; Improving fine motor (tying shoes, buttons); Walking without cane short distance; Complete flight of stairs with railing; ↓ intensity/frequency involuntary rhythmic movements, ↓dizziness; no changes headache intensity
- Ongoing PT.
- Severity Cervical Myelopathy-Modified Japanese Orthopaedic Scoring System(**Patient=8 Initial visit**)
 - Normal=18
 - Mild =13- 17 **(Patient F/U= 11)**
 - Moderate to severe< 13

Table 3. Modified Japanese Orthopaedic Association scoring system

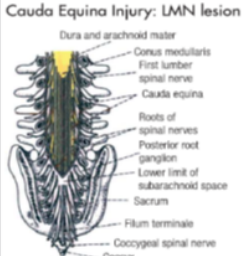
Criterion	Content	Point
Motor	Upper extremities	
	Unable to move hands	0
	Unable to eat with a spoon but able to move hands	1
	Unable to button shirt but able to eat with a spoon	2
	Able to button shirt with great difficulty	3
	Able to button shirt with slight difficulty	4
	No dysfunction	5
	Lower extremities	
	Complete loss of motor & sensory function	0
	Sensory preservation without ability to move legs	1
	Able to move legs but unable to walk	2
	Able to walk on flat floor with a walking aid (cane or crutch)	3
	Able to walk up- &/or downstairs w/aid of a handrail	4
	Moderate-to-significant lack of stability but able to walk up- &/or downstairs without handrail	5
Sensory	Upper extremities	
	Complete loss of hand sensation	0
	Severe sensory loss or pain	1
	Mild sensory loss	2
	No sensory loss	3
	Sphincter dysfunction	
Sphincter dysfunction	Unable to micturate voluntarily	0
	Marked difficulty in micturition	1
	Mild-to-moderate difficulty in micturition	2
	Normal micturition	3
Total		18

Discussion/Lessons Learned

- Atypical Symptoms Patient #2-
 - Pseudoathetosis- Rhythmic movement that mimics athetoid- secondary to large sensory loss (Ranjan A, 2024)
 - Sensory screening, motor control tasks, NCS/EMG/SSEP and Imaging to discern earlier Rx??
 - Dizziness
 - Headaches

Table 3-16

Spinal Cord Injury Syndromes

LESION	CHARACTERISTICS
Complete Cord Lesion: UMN lesion 	Complete bilateral loss of all sensory modalities Bilateral loss motor function with spastic paralysis below level of lesion Loss of bladder and bowel functions with spastic bladder and bowel
Central Cord Lesion: UMN lesion 	Cavitation of central cord in cervical section Loss of spinothalamic tracts with bilateral loss of pain and temperature Loss of ventral horn with bilateral loss of motor function: primarily upper extremities Preservation of proprioception and discriminatory sensation
Brown-Sequard Syndrome: UMN lesion 	Hemisection of spinal cord Ipsilateral loss of dorsal columns with loss of tactile discrimination, pressure, vibration, and proprioception Ipsilateral loss of corticospinal tracts with loss of motor function and spastic paralysis below level of lesion Contralateral loss of spinothalamic tract with loss of pain and temperature below level of lesion; at lesion level, bilateral loss of pain and temperature
Anterior Cord Syndrome: UMN lesion 	Loss of anterior cord Loss of lateral corticospinal tracts with bilateral loss of motor function, spastic paralysis below level of lesion Loss of spinothalamic tracts with bilateral loss of pain and temperature Preservation of dorsal columns: proprioception, kinesthesia, and vibratory sense
Posterior Cord Syndrome: UMN lesion 	Loss of dorsal columns bilaterally Bilateral loss of proprioception, vibration, pressure, and epicritic sensations (stereognosis, two-point discrimination) Preservation of motor function, pain, and light touch
Cauda Equina Injury: LMN lesion 	Loss of long nerve roots at or below L1 Variable nerve root damage (motor and sensory signs); incomplete lesions common Flaccid paralysis with no spinal reflex activity Flaccid paralysis of bladder and bowel Potential for nerve regeneration; regeneration often incomplete, slows and stops after about 1 year

LMN = lower motor neuron; UMN = upper motor neuron

Atypical Symptoms DCM, Age and Surgical Impact (Yuan et al. N=380 typical= 214 atypical=166)

- Older patients more likely to have blurred vision and tinnitus
- Incidence of C3-5 cord compression more likely with vertigo group
- C4-5 cord compression more likely in the headache group
- C6-7 cord compression more likely in the palpitation group
- Scores for vertigo (Dizziness Handicap Inventory), headache (NPRS) and palpitations decreased after cervical surgical decompression
- Only Scores for vertigo and headache decreased after non-surgical treatment
- Tinnitus (Tinnitus handicap inventory) and Blurred Vision (visual function 14 inventory) did not significant change with surgical or non-surgical treatment

Future Research

- Clinical examination items, outcome measures, and treatment techniques that address the spectrum of signs/symptoms associated with degenerative cervical myelopathy
- Clear need for functional outcome measures (both subjective and objective) for patients with cervical myelopathy
- Is electrophysiology testing a valuable biomarker for the screening, diagnosis, and/or to determine the prognosis and treatment of patients with degenerative cervical myelopathy?

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SEVERE BRACHIAL PLEXOPATHY FOLLOWING SHOULDER HEMIARTHROPLASTY: A STUDY THAT INCLUDES THE EFFECT OF CONTEXTUAL FACTORS ON OUTCOMES

Sellin R

ABSTRACT

BACKGROUND: Brachial plexus injuries following shoulder arthroplasties are uncommon, but not rare, and vary in degree of pathology. In this case the pathology was the most severe this clinician had observed in over 40 years of practice. Immediately following surgery the patient had no function of muscles innervated by the suprascapular, axillary, median or radial nerves. She had sparing of the ulnar sensory and motor nerves and the medial cutaneous nerve of the forearm.

DISCUSSION: By the projected time of this presentation the patient will have had four electrophysiologic evaluations showing an interesting pattern in her neural regeneration and functional abilities. The electrophysiologic changes over time, along with the prognostic predictions based on each study's progressive findings are presented. In addition, there will be specific interview data on the patient's perception of the contextual effects observed during her post-operative care and of the patient's response to this life altering event.

CONCLUSIONS: While the patient showed only a few regenerative motor units in her deltoid at one month post-surgery she had a moderate return of motor units in the deltoid at three months, with a few motor units in the triceps. She still had no evidence of neurologic return in any other involved muscle. At six months dramatic changes were noted with hope for more when she will be tested prior to presentation in March. Her perpetually positive attitude and her real and perceived value from the ongoing support from the surgical team and her regular physical therapy will be discussed.

Robert Sellin, PT, DSc, ECS

Severe Brachial Plexopathy Following Shoulder Hemiarthroplasty: A Study Including The Effect Of Contextual Factors on Outcomes

Robert A. Sellin, PT, DSc, ECS

37th Annual

ENMG Symposium 2026



**Rocky Mountain
University**
of Health Professions

Learning Objectives

The participant will be able to discuss the varied possible causes of peripheral nerve injuries following shoulder arthroplasties.

The participant will be able to analyze clinical electrophysiologic findings, post nerve injury.

The participant will be able to discuss the impact of contextual factors on a patient's recovery in physical therapy.



Learning Objectives

The participant will understand the psychoneurobiological determinants of positive (placebo) or negative (nocebo) biological effects on recovery.

Abbreviations:

PNI Peripheral nerve injury

RSA Reverse shoulder arthroplasty

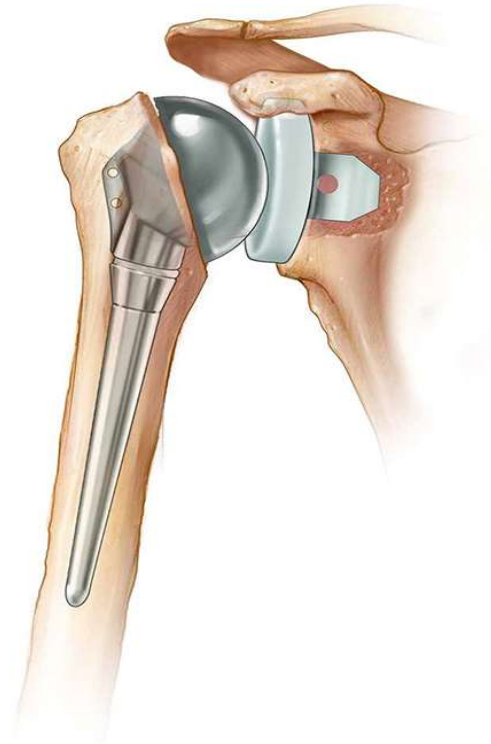
SA Shoulder arthroplasty



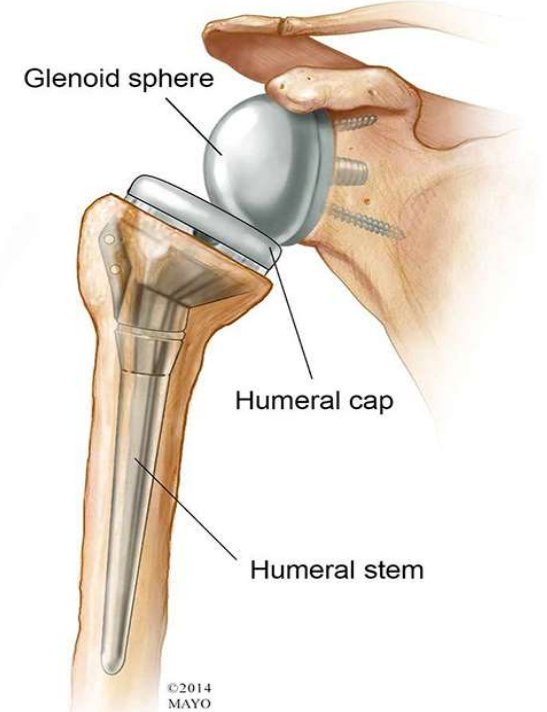


Hemiarthroplasty

Total shoulder arthroplasty



Reverse shoulder arthroplasty



Peripheral Nerve Injuries Following SA's

There is conflicting data on the incidence of peripheral nerve injuries following reverse total shoulder arthroplasties (RSA) and shoulder arthroplasties (SA).

A systematic review by North et al in 2023 (9), reported nerve injuries occur at a rate of 1.3% after primary RSA compared to 2.4 % after revision RSA.



Peripheral Nerve Injuries Following SA's

“The true incidence of neurologic injury following RSA is likely much higher than previously reported as many of these nerve injuries are transient and likely not clinically recognized or documented before resolution.” Burnett et al (2)



Peripheral Nerve Injuries Following SA's

Ball (1) attempted to identify the true incidence of nerve injury following shoulder joint replacement (SJRs) using comprehensive postoperative evaluation.

211 SJRs in 202 patients over five years

89 male, 122 female, average age 70

20.9 % had nerve complications

36 female (81.8%); 8 male (18.2%)

More complications with RSA than SA



Peripheral Nerve Injuries Following SA's

Median nerve (25 patients) and musculocutaneous (8 patients) were most commonly affected. Most of all injuries resolved by six months with “rare” permanent sequelae requiring surgical intervention.

Most injuries were transient neuropraxias involving the lateral cord of the plexus



Peripheral Nerve Injuries Following SA's

Kim et al (6)- A study of the prevalence and outcomes of neurologic deficit after RSA and correlate with distalization

Distalization refers to the downward shift of the center of rotation of the glenohumeral joint, achieved by implanting the humeral component lower than the original anatomy. (Google)

Purpose: Increase deltoid muscle function, improve stability to optimize outcomes



Peripheral Nerve Injuries Following SA's

The optimum distalization shoulder angle (DSA) is still a subject of ongoing research.

In the Kim et al study they reported 19% of RSA patients had a neurologic deficit. Those injuries were significantly correlated with humeral distalization post surgery.

In this study the axillary (41.2 %) and radial (15%) nerves were the most common injuries.



Peripheral Nerve Injuries Following SA's

From Kim et al

30 shoulders (88%) had neuropraxia

4 shoulders (12%) axonotmesis

All achieved complete recovery without additional surgery

Mean recovery period 7.4 months (2-38 months)



Potential Causes of PNI's in SA's

A number of potential causes for nerve injuries in RSA's were discussed in the systematic review by North et al (9)

- Brachial plexus stretch
- Risk of iatrogenic nerve injury with extensive glenoid exposure
- Axillary nerve damage due to its location
- Retractor damage
- Interscalene block



Potential Causes of PNI's in SA's

In a review article by Kim (7) they reported one case in 438 RSA cases of what they termed a “brachial plexus palsy.” The patient had only partial return after three years. She had immediate weakness in shoulder elevation, wrist drop and grip strength (no initial EMG data reported).

Two years later “a nerve conduction study showed a brachial plexus lesion.”



Potential Causes of PNI's in SA's

Kim et al state, "One possible cause of injury was excessive positioning such as hyperextension, external rotation, and anterior translation during the humeral procedure."

They further reported special care to avoid these situations and then performed 236 RSA's without brachial plexus injury



Potential Causes of PNI's in SA's

Kam et al, (5) in a cadaveric study:
6 fresh frozen cadaveric upper limb
specimens

Digital load cells attached to the
lateral, medial and posterior cords

Each specimen underwent shoulder
hemiarthroplasty

Then a revision RSA to all specimens.
All surgeries were performed by
the same surgeon



Potential Causes of PNI's in SA's

Strains tolerated by human nerves have not been studied in vivo but have been studied in animal models. The consensus from the animal study findings are (5):

1. Strains of 8% cause reversible nerve dysfunction
2. Strains of 10% may cause nerve injury
3. Strains of 12% cause nerve injury



Potential Causes of PNI's in SA's

Kam et al, (5) found “strain in the cords of the brachial plexus sufficient to cause neurologic injury (10%) occurs during:

1. shoulder abduction greater than 70 degrees,
2. the combination of 70 deg. shoulder abduction with external rotation
3. the combination of shoulder external rotation greater than 60 deg. with extension greater than 50 deg.”



Potential Causes of PNI's in SA's

In the Kam study two of the specimens were supported under the elbow, by an assistant, for both surgeries. Four of the specimens were held only by the forearms.

In hemiarthroplasty the specimens with support under the elbow did not experience increased cord tension

Without support there was increased tension on the medial cord



Potential Causes of PNI's in SA's

Kam et al, states “The study strongly supports the hypothesis that brachial plexus injury during shoulder surgery is due to intraoperative stretching forces.”

Supporting the upper limb from under the elbow reduced medial cord tensions.



Case: Brachial Plexopathy

63 year old healthy female had a right shoulder hemiarthroplasty on March 06, 2025. She gave verbal permission to use her case for presentation or publication.

- First test date April 01, 2025
- Initially following surgery she reported complete arm numbness with no active motion.
- She arrives in a sling and swath for the right upper extremity



Case: Brachial Plexopathy

- The uninvolved left upper extremity had full ROM, normal strength and sensation with 2+ MSR's
- Full pain free cervical ROM
- No change in symptoms in the 26 days since surgery
- MMT – 1/5 right shoulder abduction and 4/5 shoulder external and internal rotation



Case: Brachial Plexopathy

- 0/5 right shoulder flexion, elbow flexion and extension, wrist flexion and extension, finger and thumb flexion and extension
- Except trace active flexion of the long, ring and small fingers with concerted effort
- Sensation was severely diminished throughout the entire right arm except in the distribution of the ulnar nerve



Case: Brachial Plexopathy Sensory NCV Test 1

Nerve/Sites	Rec. Site	Peak Lat ms	NP Amp uV	Segments	Distance cm	Vel. Pk m/s
R Radial – Anatomical snuff box (Forearm)						
Forearm	Wrist	0.0	0.0	Forearm- wrist	10	
R Median – Orthodromic (Dig II, Mid Palm)						
Mid Palm	Wrist	0.0	0.0	Mid palm – wrist	8	
Dig II/III	Wrist	0.0	0.0	Dig II/III - wrist	14	
R Ulnar – Orthodromic (Dig V, Mid palm)						
Mid Palm	Wrist	1.4	16.2	Mid palm- wrist	8	56.9
Dig V	Wrist	2.3	14.0	Dig V - wrist	12	52.4



Case: Brachial Plexopathy Motor NCV Test 1

Nerve/Sites	Muscle	Latency ms	Amp. mV	Segments	Distance cm	Velocity M/S
R Median – APB						
Wrist	APB	0.0	0.0	Wrist - APB	8	
Elbow	APB	0.0	0.0	Elbow-wrist	18	
R Ulnar - ADM						
Wrist	ADM	2.0	9.9	Wrist – ADM	8	
B. Elbow	ADM	4.8	10.1	B. Elbow - wrist	17	69
A. Elbow	ADM	6.2	10.3	A. Elbow – B Elbow	10	60

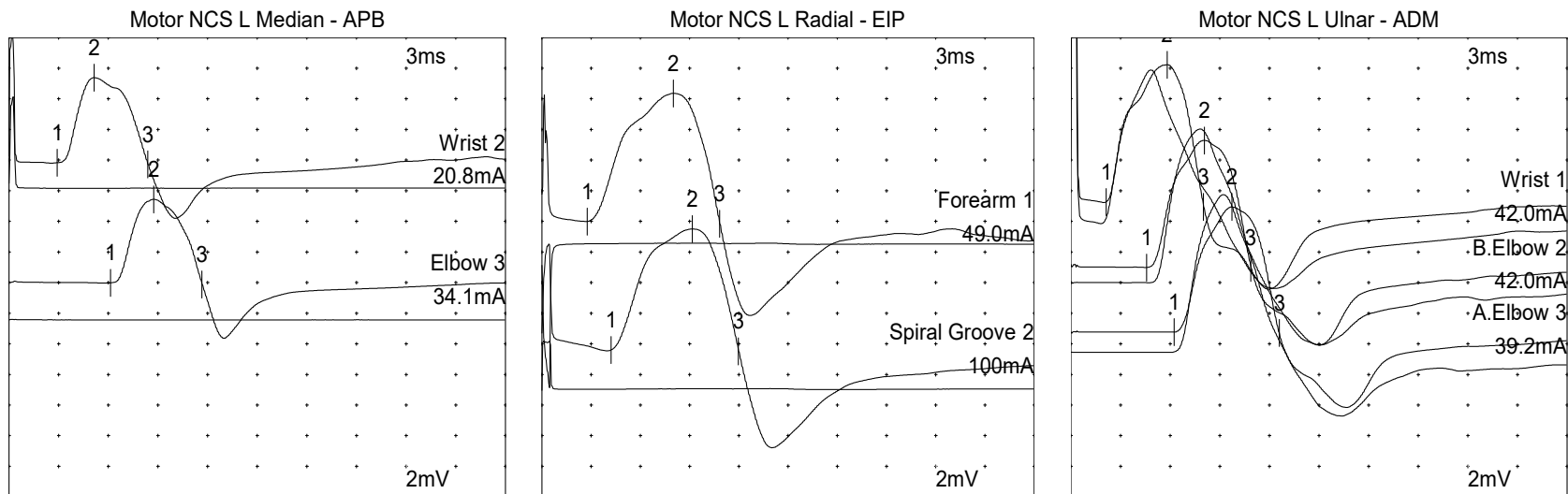


Case: Brachial Plexopathy Motor NCV Test 1

Nerve/Sites	Muscle	Latency ms	Amp. mV	Segments	Distance cm	Velocity M/S
R Radial – EIP						
Forearm	EIP	0.0	0.0	Forearm - EIP	8	
Spiral Groove	EIP	0.0	0.0	Sp. Groove - Forearm	11	



Case: Brachial Plexopathy Motor NCV Test 1



Case: Brachial Plexopathy EMG Test 1

EMG Summary Table Right Side									
Spontaneous						MUAP			Recruitment
Muscle	IA	Fib	PSW	FASC	H.F.	AMP	DUR	PPP	Pattern
Deltoid	Inc	3+	3+	None	None				No Activity
Biceps brachii	Inc	3+	3+	None	None				No Activity
Triceps brachii	Inc	3+	3+	None	None				No Activity
Brachioradialis	Inc	3+	3+	None	None				No Activity
Pronator teres	Inc	3+	3+	None	None				No Activity
Extensor Dig.	Inc	3+	3+	None	None				No Activity

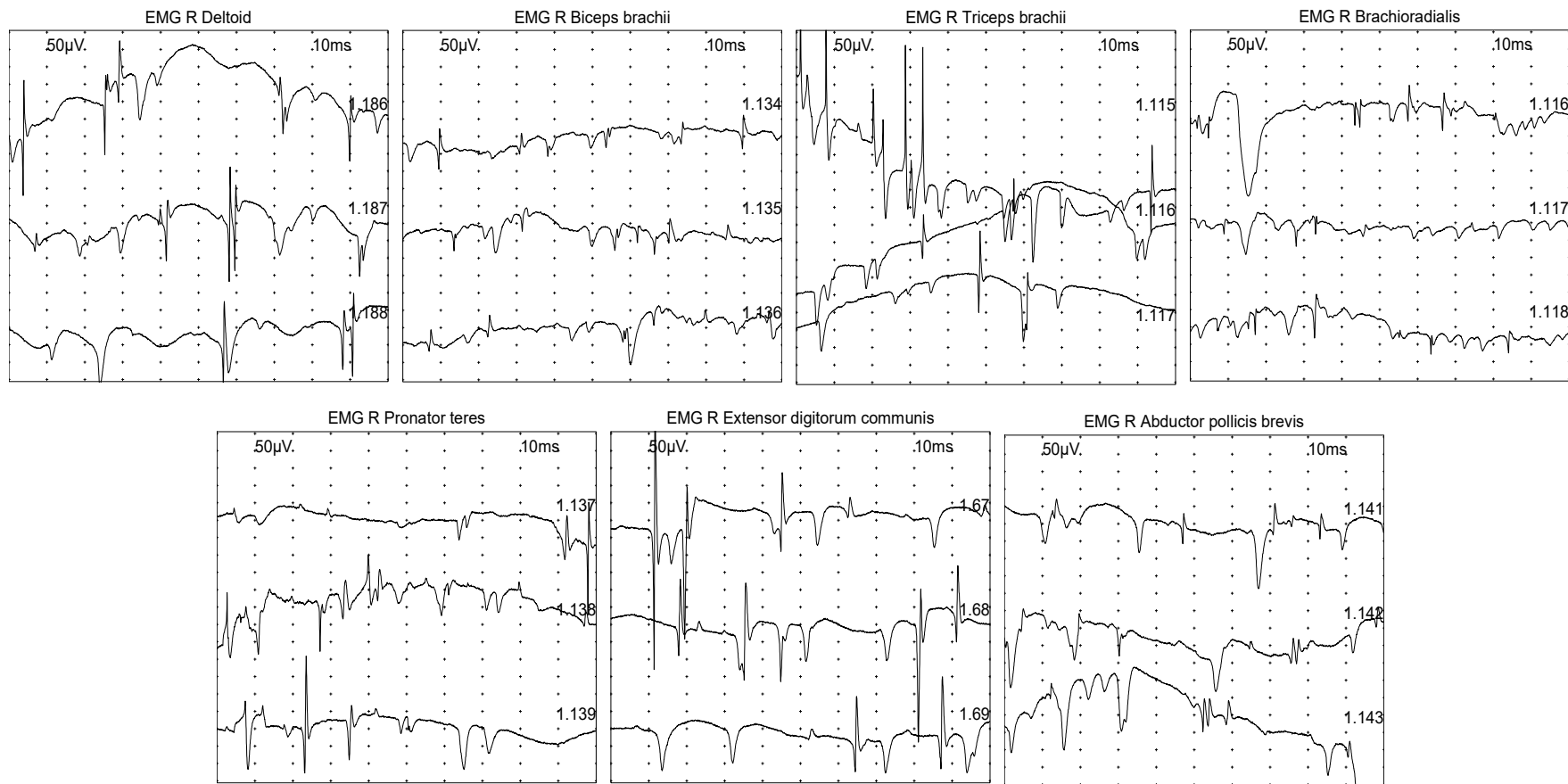


Case: Brachial Plexopathy EMG Test 1

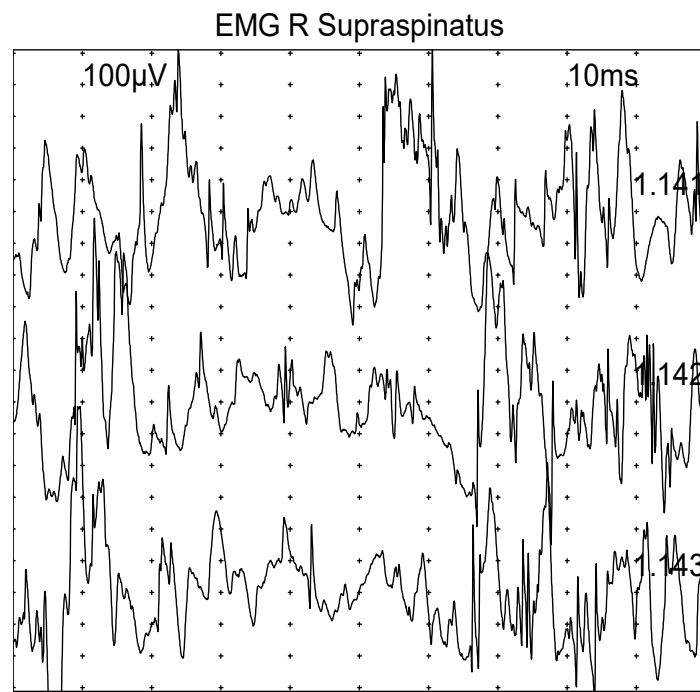
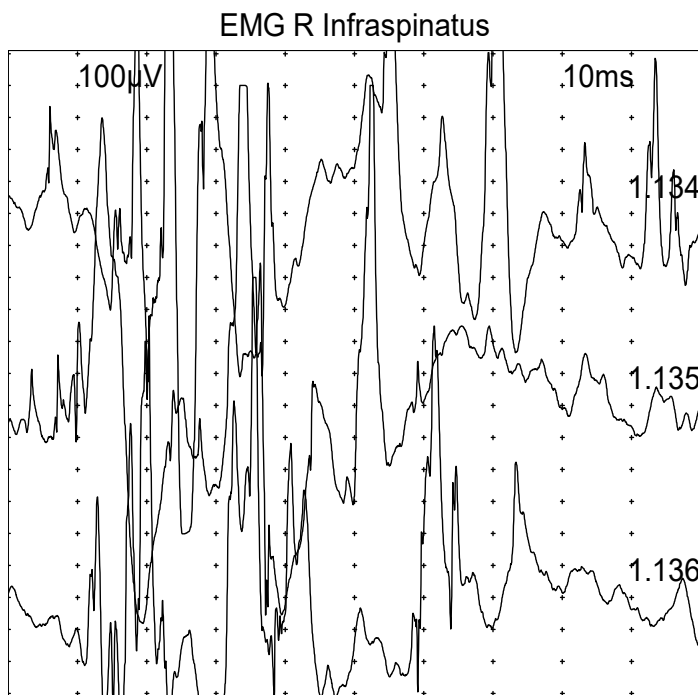
EMG Summary Table Right Side									
Spontaneous			MUAP			Recruitment			
Muscle	IA	Fib	PSW	FASC	H.F.	AMP	DUR	PPP	Pattern
Flexor carpi ulnaris	N	No	No	None	None	N	N	N	N
Flex Dig Pro (Ulnar)	N	No	No	None	None	N	N	N	N
Flex Dig Pro (Median)	Inc	2+	2+	None	None				No activity
FDI and ADM	N	No	No	None	None	N	N	N	N
APB	Inc	3+	3+	None	None				No activity
Infraspinatus	N	No	No	None	None	N	N	1+	Reduced
Supraspinatus	N	No	No	None	None	N	3+	3+	Reduced



Case: Brachial Plexopathy EMG Test 1



Case: Brachial Plexopathy EMG Test 1



Case: Brachial Plexopathy Test 1

IMPRESSION:

Acute axonal pathology involving the right brachial plexus that is electrophysiologically severe, with some relative sparing of the upper trunk and medial cord of the plexus.



Case: Brachial Plexopathy Test 2

- Test date June 17, 2025: 3 months post-op
- She reports some increase in strength in her shoulder with tingling down the arm
- 2/5 Deltoid and 1/5 triceps
- Makes a partial fist with ulnar side of hand
- Sensation returned to proximal arm



Case: Brachial Plexopathy Test 2

- Sensation decreased in the lateral arm and severely decreased in a median nerve distribution in the hand
- MSR's 1+ at triceps and absent at biceps brachii and brachioradialis
- Ulnar nerve innervated muscles normal strength, ulnar sensation normal
- All other upper extremity muscles 0/5



Case: Brachial Plexopathy Sensory NCV Test 2

Nerve/Sites	Rec. Site	Peak Lat ms	NP Amp uV	Segments	Distance cm	Vel. Pk m/s
R Radial – Anatomical snuff box (Forearm)						
Forearm	Wrist	0.0	0.0	Forearm-wrist	10	
R Median – Orthodromic (Dig II, Mid Palm)						
Mid Palm	Wrist	0.0	0.0	Mid palm – wrist	8	
Dig II/III	Wrist	0.0	0.0	Dig II/III - wrist	14	
R Ulnar – Orthodromic (Dig V, Mid palm)						
Mid Palm	Wrist	1.4	16.2	Mid palm-wrist	8	56.9
Dig V	Wrist	2.3	14.0	Dig V - wrist	12	52.4



Case: Brachial Plexopathy Sensory NCV Test 2

Nerve/Sites	Rec. Site	Peak Lat ms	NP Amp uV	Segments	Distance cm	Vel. Pk m/s
R Lateral cutaneous nerve of the forearm (elbow)						
Elbow	Forearm	0.0	0.0	Elbow - Forearm	10	
R Medial cutaneous nerve of the forearm (elbow)						
Elbow	Forearm	2.0	16.3	Elbow - Forearm	10	49.2



Case: Brachial Plexopathy Motor NCV Test 2

Nerve/Sites	Muscle	Latency ms	Amp. mV	Segments	Distance cm	Velocity M/S
R Median – APB						
Wrist	APB	0.0	0.0	Wrist - APB	8	
Elbow	APB	0.0	0.0	Elbow-wrist	18	
R Ulnar - ADM						
Wrist	ADM	2.2	9.9	Wrist – ADM	8	
B. Elbow	ADM	5.0	9.8	B. Elbow - wrist	19	68
A. Elbow	ADM	6.4	9.8	A. Elbow – B Elbow	10	71



Case: Brachial Plexopathy Motor NCV Test 2

Nerve/Sites	Muscle	Latency ms	Amp. mV	Segments	Distance cm	Velocity M/S
R Radial – EIP						
Forearm	EIP	0.0	0.0	Forearm - EIP	8	
Spiral Groove	EIP	0.0	0.0	Sp. Groove - Forearm	11	
R Axillary – Deltoid						
Erb's Pt.	Deltoid	0.0	0.0			



Case: Brachial Plexopathy EMG Test 2

EMG Summary Table Right Side

Muscle	Spontaneous					MUAP			Recruitment
	IA	Fib	PSW	FASC	H.F.	AMP	DUR	PPP	Pattern
Deltoid	Inc	1+	1+	None	None	N	3+	3+	Reduced
Biceps brachii	Inc	3+	3+	None	None				No Activity
Triceps brachii	Inc	3+	3+	None	None	N	N	N	Discrete
Brachioradial.	Inc	3+	3+	None	None				No Activity
Pronator teres	Inc	3+	3+	None	None				No Activity
Extensor Dig.	Inc	3+	3+	None	None				No Activity



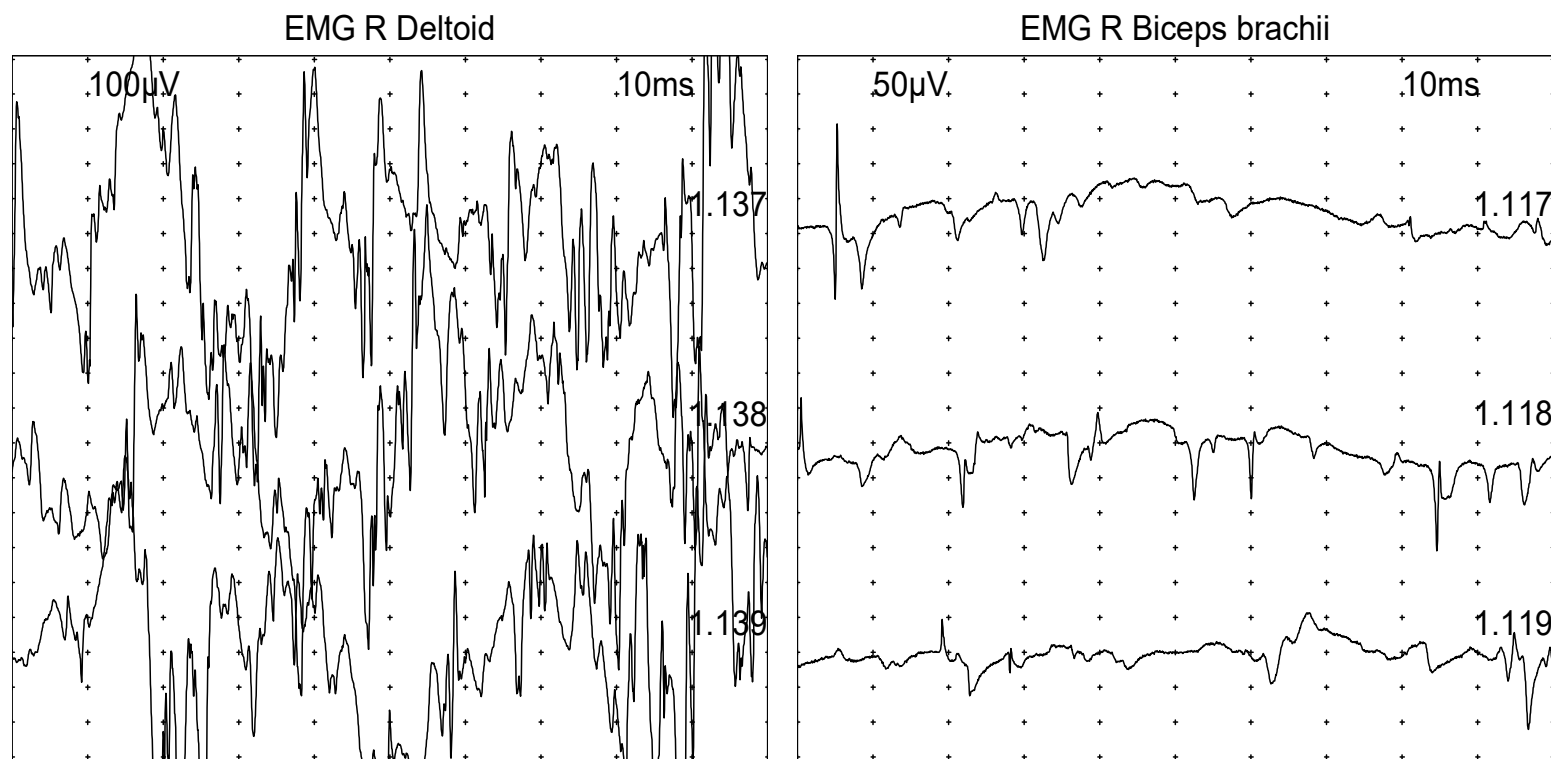
Case: Brachial Plexopathy EMG Test 2

EMG Summary Table Right Side

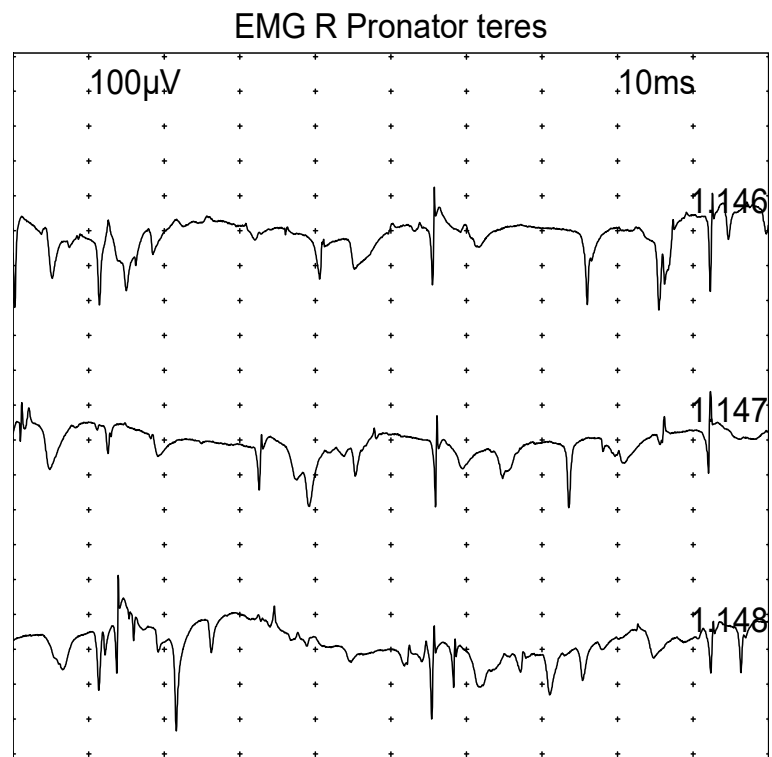
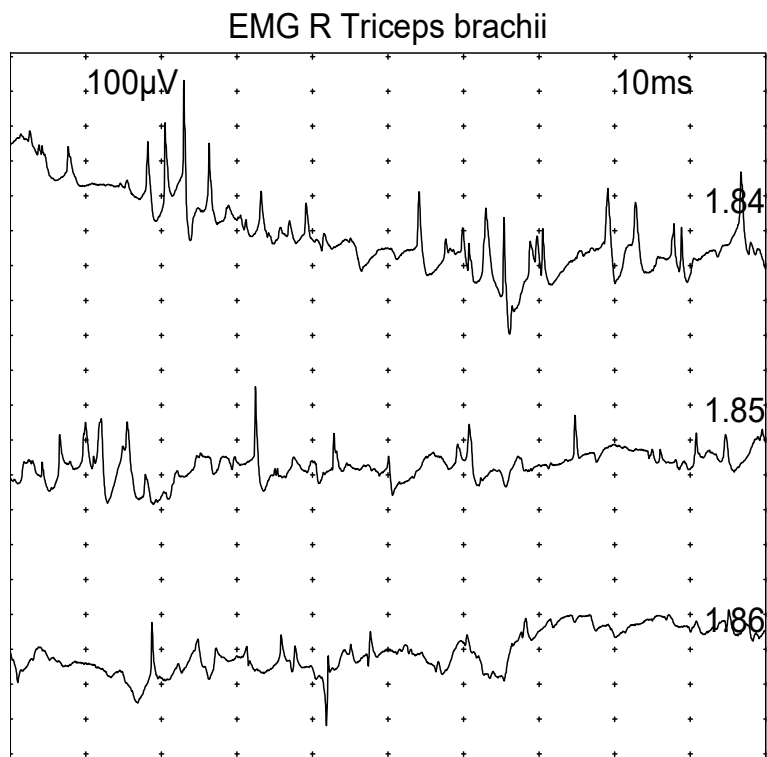
Spontaneous			MUAP				Recruitment		
Muscle	IA	Fib	PSW	FASC	H.F.	AMP	DUR	PPP	Pattern
FDI	N	No	No	None	None	N	2+	2+	Reduced
ADM	N	No	No	None	None	N	N	1+	Reduced
APB	Inc	3+	3+	None	None				No activity
Infraspinatus	N	No	No	None	None	N	2+	2+	Reduced



Case: Brachial Plexopathy EMG Test 2

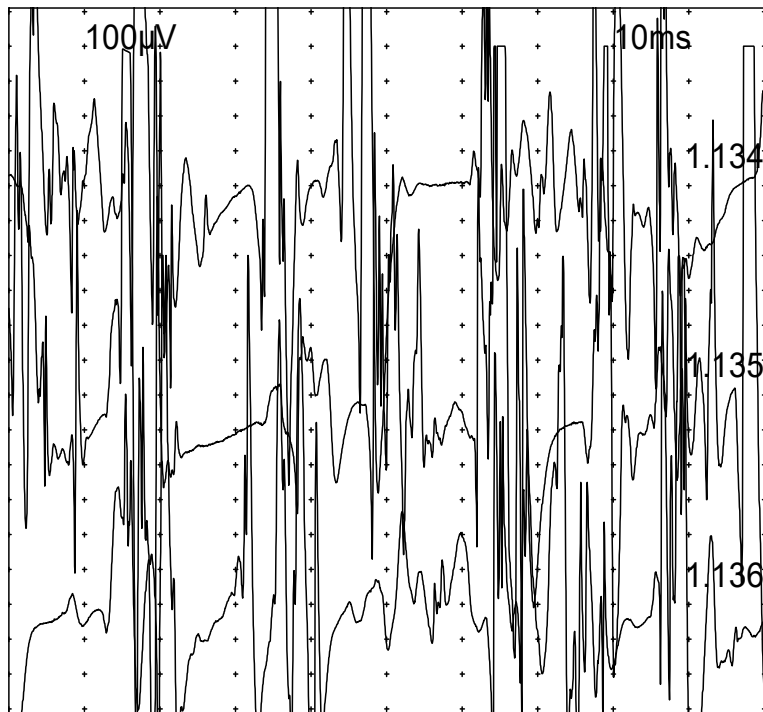


Case: Brachial Plexopathy EMG Test 2

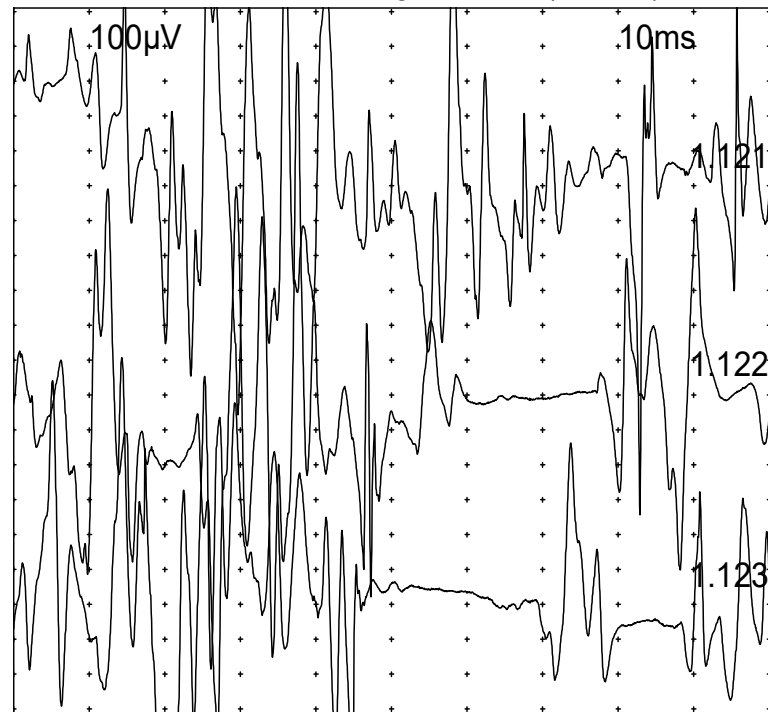


Case: Brachial Plexopathy EMG Test 2

EMG R First dorsal interosseus



EMG R Abductor digiti minimi (manus)



Case: Brachial Plexopathy Test 2

IMPRESSION:

Acute axonal pathology involving the right brachial plexus that is electrophysiologically severe, with some relative sparing of the upper trunk and medial cord of the plexus.

There is evidence of good neural regeneration to the deltoid muscle and a few discrete active motor units in the triceps. These data are indicative of proximal neural regeneration. Follow up study in 3 – 4 months.



Case: Brachial Plexopathy Test 3

- Test date September 17, 2025: 6 months post-op
- She reports improved movement in the proximal arm but not distally
- Tingling down the lateral arm with dense numbness in a median nerve distribution
- Active shoulder flexion/abduction to 90-95 degrees with full passive ROM



Case: Brachial Plexopathy Test 3

- Active flexion and extension of the elbow but no active motion at the wrist
- 4-/5 shoulder flexion/abduction
- 5/5 shoulder external and internal rotation
- 3+/5 elbow flexion
- 3/5 elbow extension
- Trace pronation and wrist flexion



Case: Brachial Plexopathy Test 3

- 0/5 wrist and digit extension
- MSR's 1+ triceps and absent biceps brachii and brachioradialis



Case: Brachial Plexopathy Sensory NCV Test 3

Nerve/Sites	Rec. Site	Peak Lat ms	NP Amp uV	Segments	Distance cm	Vel. Pk m/s
R Radial – Anatomical snuff box (Forearm)						
Forearm	Wrist	0.0	0.0	Forearm- wrist	10	
R Median – Orthodromic (Dig II, Mid Palm)						
Mid Palm	Wrist	0.0	0.0	Mid palm – wrist	8	
Dig II/III	Wrist	0.0	0.0	Dig II/III - wrist	14	
R Ulnar – Orthodromic (Dig V, Mid palm)						
Mid Palm	Wrist	1.5	13.7	Mid palm- wrist	8	54.9
Dig V	Wrist	2.2	20.6	Dig V - wrist	12	53.6



Case: Brachial Plexopathy Sensory NCV Test 3

Nerve/Sites	Rec. Site	Peak Lat ms	NP Amp uV	Segments	Distance cm	Vel. Pk m/s
R Lateral cutaneous nerve of the forearm (elbow)						
Elbow	Forearm	0.0	0.0	Elbow - Forearm	10	
R Medial cutaneous nerve of the forearm (elbow)						
Elbow	Forearm	1.9	10.5	Elbow - Forearm	10	53.3



Case: Brachial Plexopathy Motor NCV Test 3

Nerve/Sites	Muscle	Latency ms	Amp. mV	Segments	Distance cm	Velocity M/S
R Median – APB						
Wrist	APB	0.0	0.0	Wrist - APB	8	
Elbow	APB	0.0	0.0	Elbow-wrist	18	
R Ulnar - ADM						
Wrist	ADM	2.1	12.0	Wrist – ADM	8	
B. Elbow	ADM	4.9	11.9	B. Elbow - wrist	18	65
A. Elbow	ADM	6.6	11.1	A. Elbow – B Elbow	10	60



Case: Brachial Plexopathy Motor NCV Test 3

Nerve/Sites	Muscle	Latency ms	Amp. mV	Segments	Distance cm	Velocity M/S
R Radial – EIP						
Forearm	EIP	0.0	0.0	Forearm - EIP	8	
Spiral Groove	EIP	0.0	0.0	Sp. Groove - Forearm	13	
R Axillary – Deltoid						
Erb's Pt.	Deltoid	6.7	5.8			

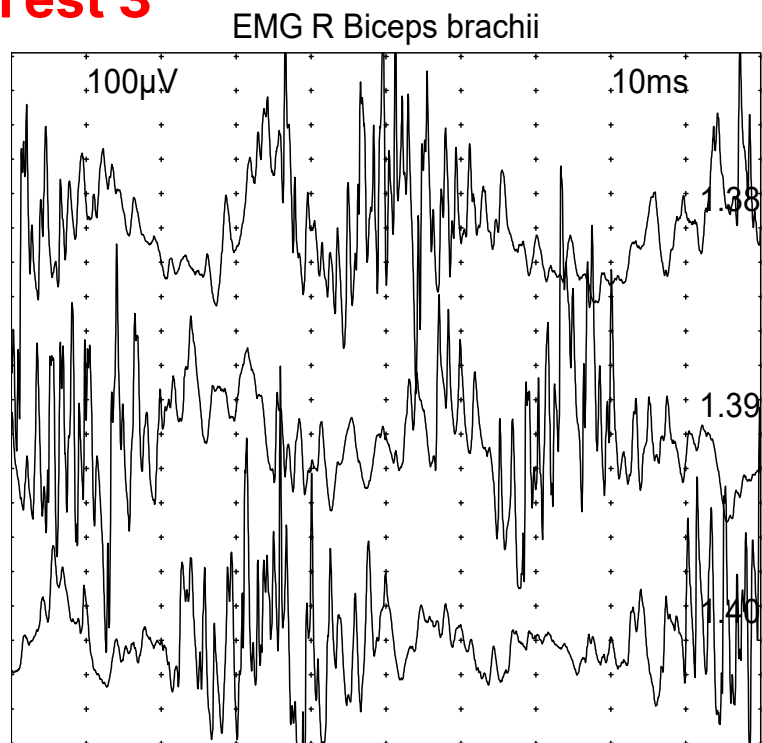


Case: Brachial Plexopathy EMG Test 3

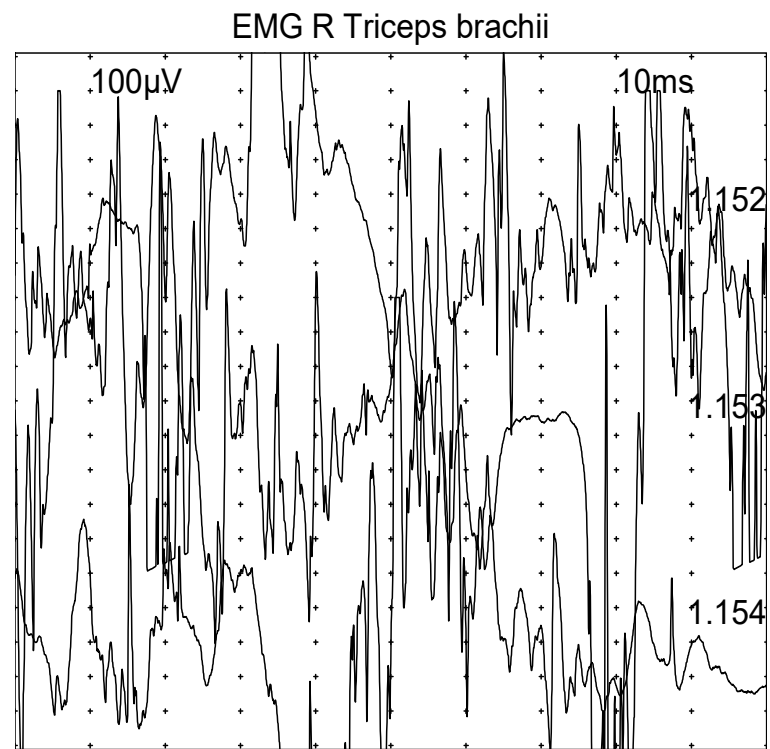
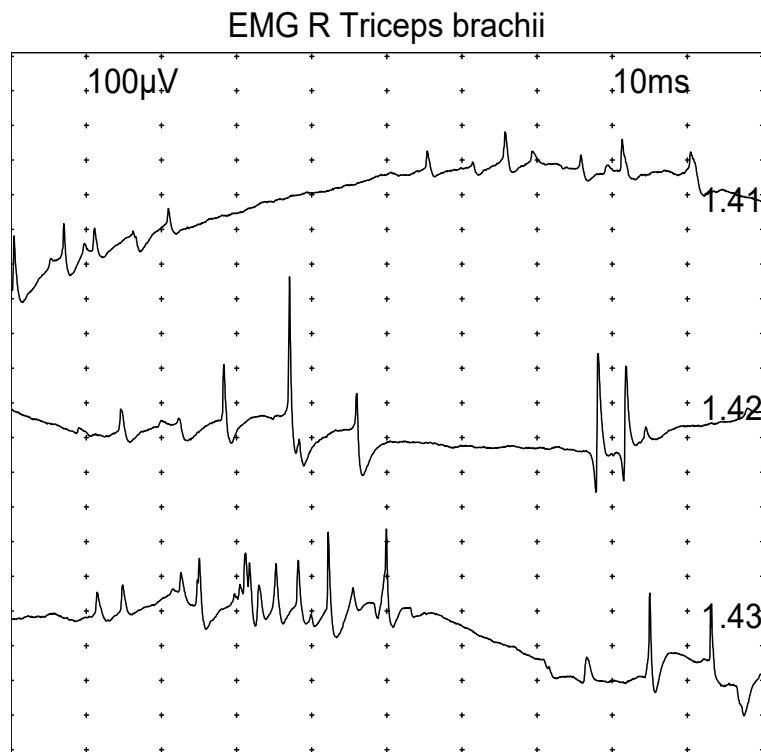
EMG Summary Table Right Side									
Spontaneous						MUAP		Recruitment	
Muscle	IA	Fib	PSW	FASC	H.F.	AMP	DUR	PPP	Pattern
Deltoid	N	No	No	None	None	N	N	1+	Reduced
Biceps brachii	Inc	1+	1+	None	None	N	3+	3+	Reduced
Triceps brachii	Inc	1+	1+	None	None	N	3+	3+	Reduced
Brachioradial.	Inc	2+	2+	None	None	N	2+	2+	Discrete
Pronator teres	Inc	1+	1+	None	None	N	2+	2+	Reduced
Extensor Carpi Radialis	Inc	4+	4+	None	None				No Activity
Extensor Dig.	Inc	4+	4+	None	None				No Activity



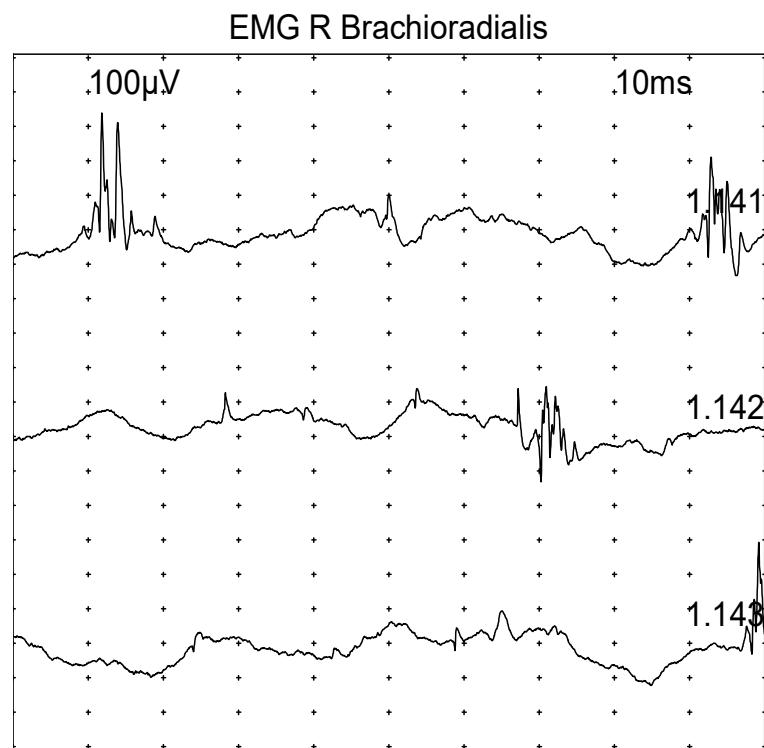
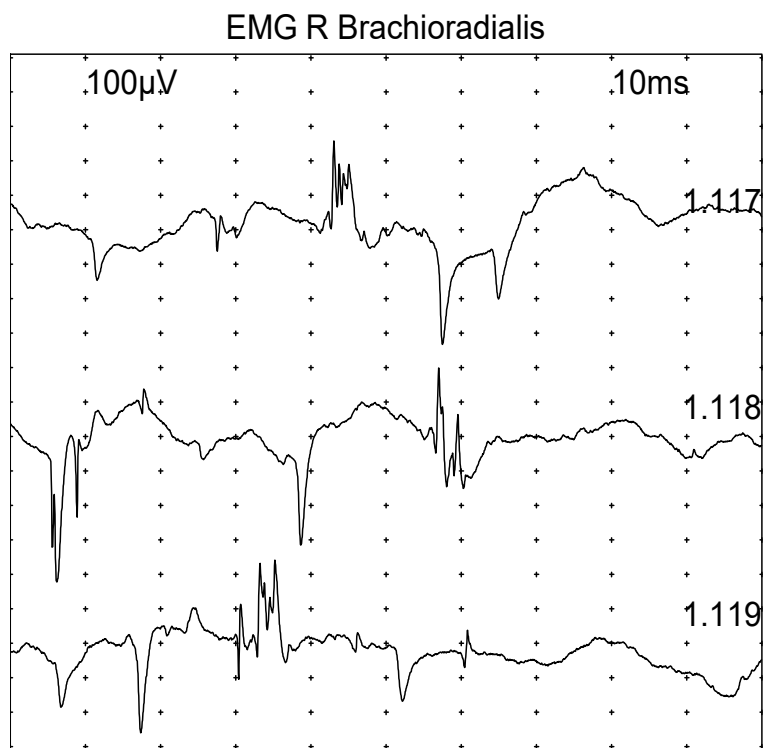
Case: Brachial Plexopathy EMG Test 3



Case: Brachial Plexopathy EMG Test 3



Case: Brachial Plexopathy EMG Test 3



Case: Brachial Plexopathy EMG NCV Test 3

EMG Summary Table Right Side

Spontaneous			MUAP			Recruitment			
Muscle	IA	Fib	PSW	FASC	H.F.	AMP	DUR	PPP	Pattern
FDI	N	No	No	None	None	N	N	N	N
APB	Inc	3+	3+	None	None				No activity
Infraspinatus	N	No	No	None	None	N	N	N	N
Trapezius (upper)	N	No	No	None	None	N	N	N	N
Supraspinatus	N	No	No	None	None	N	N	N	N



Case: Brachial Plexopathy Test 3

IMPRESSION:

Electrophysiologically severe axonopathic pathology of the right brachial plexus with evidence of marked neurological improvement since our previous study,

There is almost complete neural regeneration of the right axillary nerve to the deltoid muscle with resolution of the acute denervation, a near normal interference pattern, and now a measurable evoked response from the axillary nerve.



Case: Brachial Plexopathy Test 3

IMPRESSION:

There has been strong neural regeneration to the biceps and triceps muscles with early regeneration to the brachioradialis and pronator teres muscles.

There were no active motor units on previous testing of these muscles.



Case: Brachial Plexopathy Test 3

IMPRESSION:

These data show an active ongoing neural regeneration process with good prognosis for further healing. She is doing very well in physical therapy, maximizing her strength gains, and maintaining full passive ROM of the right upper extremity. Recommend a follow up study in 4-5 months.



Case: Brachial Plexopathy Test 4

- Test date January 14, 2026: 10 months post-op
- She reports improved movement in the proximal and distal arm
- Tingling down the lateral arm with tingling and decreased sensation in a median nerve distribution
- Active shoulder flexion/abduction to 140 degrees with full passive ROM



Case: Brachial Plexopathy Test 4

- Active flexion and extension of the elbow with active flexion and extension at the wrist
- 5/5 shoulder flexion/abduction
- 5/5 shoulder external and internal rotation
- 4/5 elbow flexion
- 4/5 elbow extension
- 4-/5 supination
- 3/5 pronation



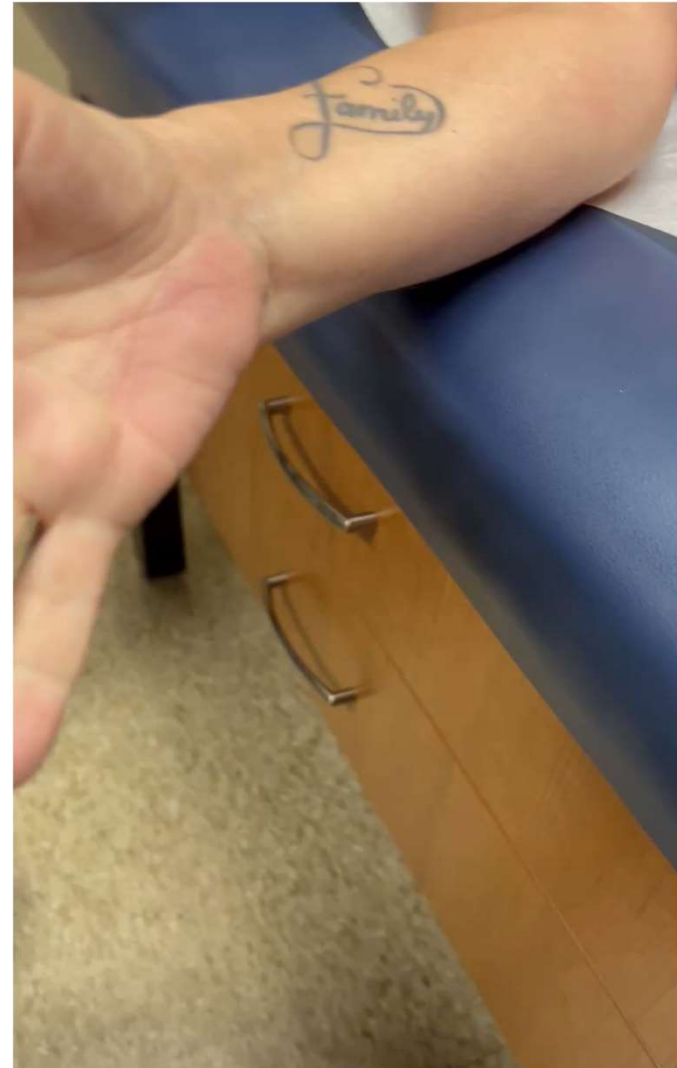
Case: Brachial Plexopathy Test 4

- 1+/5 wrist and digit extension
- 4/5 finger flexion and thumb DIP flexion
- MSR's 1+ triceps and absent biceps brachii and brachioradialis



Case: Brachial Plexopathy Test 4

- 1+/5 wrist and digit extension
- 4/5 finger flexion and thumb DIP flexion
- MSR's 1+ triceps and absent biceps brachii and brachioradialis



Case: Brachial Plexopathy Sensory NCV Test 4

Nerve/Sites	Rec. Site	Peak Lat ms	NP Amp uV	Segments	Distance cm	Vel. Pk m/s
R Radial – Anatomical snuff box (Forearm)						
Forearm	Wrist	2.3	3.1	Forearm-wrist	10	
R Median – Orthodromic (Dig II, Mid Palm)						
Mid Palm	Wrist	1.4	6.6	Mid palm – wrist	8	
Dig II/III	Wrist	0.0	0.0	Dig II/III - wrist	14	
R Ulnar – Orthodromic (Dig V, Mid palm)						
Mid Palm	Wrist	1.4	21.4	Mid palm-wrist	8	54.9
Dig V	Wrist	2.3	15.1	Dig V - wrist	12	53.6



Case: Brachial Plexopathy Sensory NCV Test 4

Nerve/Sites	Rec. Site	Peak Lat ms	NP Amp uV	Segments	Distance cm	Vel. Pk m/s
R Lateral cutaneous nerve of the forearm (elbow)						
Elbow	Forearm	0.0	0.0	Elbow - Forearm	10	
R Medial cutaneous nerve of the forearm (elbow)						
Elbow	Forearm	2.1	7.5	Elbow - Forearm	10	53.3



Case: Brachial Plexopathy Motor NCV Test 4

Nerve/Sites	Muscle	Latency ms	Amp. mV	Segments	Distance cm	Velocity M/S
R Median – APB						
Wrist	APB	0.0	0.0	Wrist - APB	8	
Elbow	APB	0.0	0.0	Elbow-wrist	18	
R Ulnar - ADM						
Wrist	ADM	2.1	10.1	Wrist – ADM	8	
B. Elbow	ADM	5.2	9.7	B. Elbow - wrist	19	61
A. Elbow	ADM	6.7	9.6	A. Elbow – B Elbow	10	69



Case: Brachial Plexopathy Motor NCV Test 4

Nerve/Sites	Muscle	Latency ms	Amp. mV	Segments	Distance cm	Velocity M/S
R Radial – EIP						
Forearm	EIP	0.0	0.0	Forearm - EIP	8	
Spiral Groove	EIP	0.0	0.0	Sp. Groove - Forearm	13	

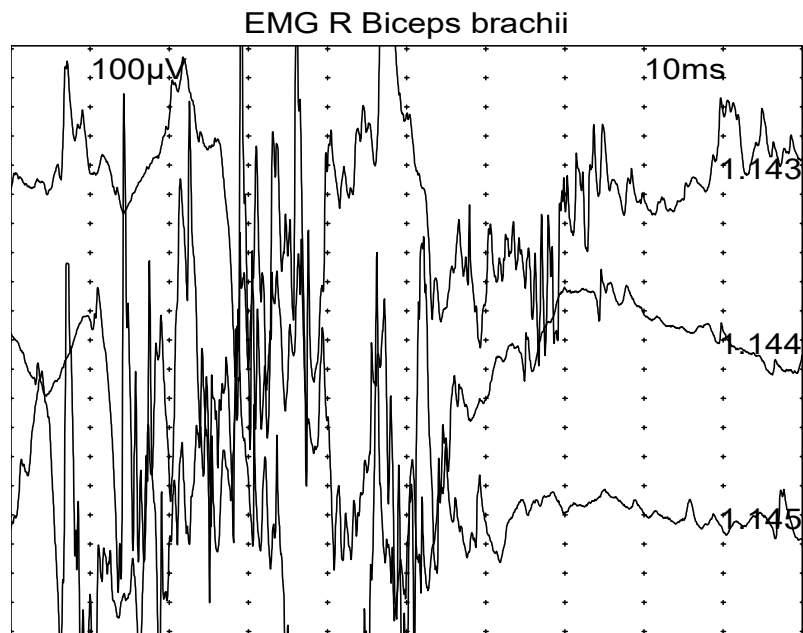


Case: Brachial Plexopathy EMG Test 4

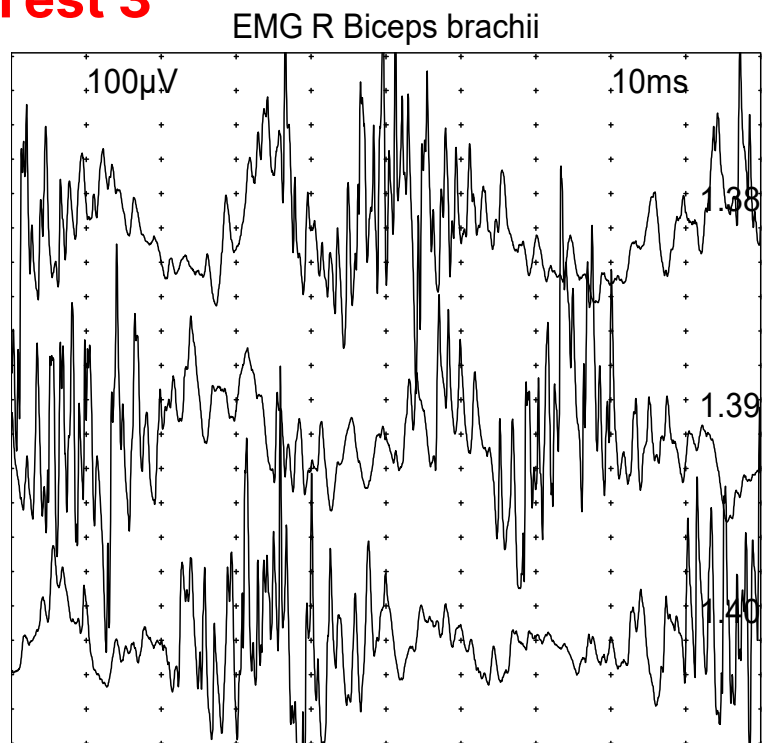
EMG Summary Table Right Side									
Spontaneous			MUAP				Recruitment		
Muscle	IA	Fib	PSW	FASC	H.F.	AMP	DUR	PPP	Pattern
Deltoid	N	No	None	None	None	N	N	N	N
Biceps brachii	Inc	1+	1+	None	None	N	3+	3+	Reduced
Triceps brachii	N	No	None	None	None	2+	N	1+	Reduced
Brachioradial.	Inc	1+	1+	None	None	1+	1+	1+	Reduced
Pronator teres	Inc	No	None	None	None	N	2+	2+	Reduced
Extensor Carpi Radialis	Inc	1+	1+	None	None	Dec.	3+	3+	Markedly Reduced
Extensor Dig.	Inc	2+	2+	None	None	Dec.	3+	3+	Discrete



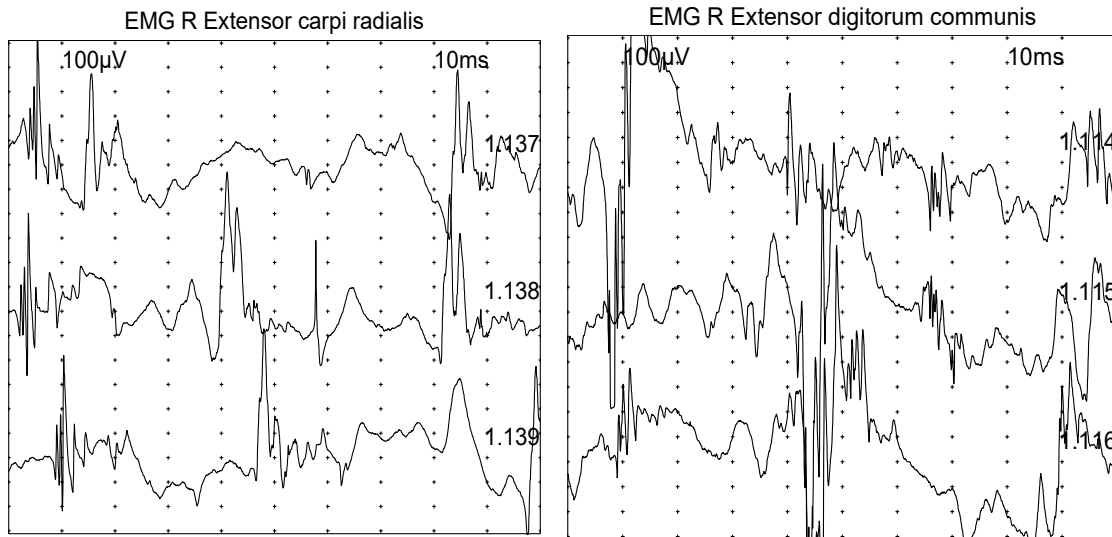
Case: Brachial Plexopathy EMG Test 4



Case: Brachial Plexopathy EMG Test 3



Case: Brachial Plexopathy EMG Test 4



Case: Brachial Plexopathy EMG Test 3

EMG Summary Table Right Side									
Spontaneous						MUAP		Recruitment	
Muscle	IA	Fib	PSW	FASC	H.F.	AMP	DUR	PPP	Pattern
Deltoid	N	No	No	None	None	N	N	1+	Reduced
Biceps brachii	Inc	1+	1+	None	None	N	3+	3+	Reduced
Triceps brachii	Inc	1+	1+	None	None	N	3+	3+	Reduced
Brachioradial.	Inc	2+	2+	None	None	N	2+	2+	Discrete
Pronator teres	Inc	1+	1+	None	None	N	2+	2+	Reduced
Extensor Carpi Radialis	Inc	4+	4+	None	None				No Activity
Extensor Dig.	Inc	4+	4+	None	None				No Activity



Case: Brachial Plexopathy EMG NCV Test 4

EMG Summary Table Right Side

Spontaneous			MUAP				Recruitment		
Muscle	IA	Fib	PSW	FASC	H.F.	AMP	DUR	PPP	Pattern
FDI	N	No	No	None	None	N	N	N	N
APB	Inc	3+	3+	None	None				No activity



Case: Brachial Plexopathy Test 4

IMPRESSION:

Electrophysiologically severe axonopathic pathology of the right brachial plexus with evidence of marked neurological improvement since our previous study,

There is complete neural regeneration of the right axillary nerve to the deltoid muscle. There is continued strong neural regeneration to the biceps and triceps muscles with markedly improved regeneration to the brachioradialis and pronator teres muscles



Case: Brachial Plexopathy Test 4

IMPRESSION:

There are now active regenerative motor units in the extensor carpi radialis and extensor digitorum muscles.

These data show an active ongoing neural regeneration process with good prognosis for further neural healing. She has maxed out of her possible physical therapy but has an excellent home exercise program and works on ROM and strengthening daily. I did notice a mild flexion contracture of the right elbow and taught her stretching techniques for the elbow. Recommend a follow up study in six months.



Placebo and Nocebo Effects on Rehabilitation

Placebo – From the Latin
“I shall please”

Nocebo – From the Latin
“I shall harm”



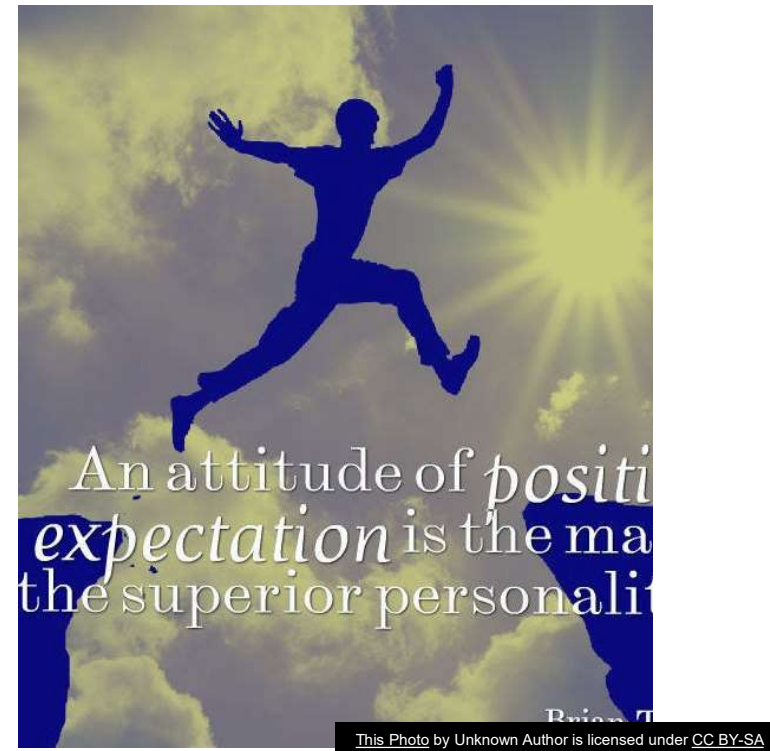
Placebo and Nocebo Effects on Rehabilitation

Rossettini et al (10) state, “Placebo and nocebo effects embody complex and distinct psychoneurobiological phenomena where behavioral, neurophysiological, perceptive and cognitive changes occur during therapeutic encounter, between the (physical) therapist and the patient in the health care context.”



Placebo and Nocebo Effects on Rehabilitation –Contextual Factors

- The therapist's professionalism, mindset and appearance
- The patient's beliefs, experiences and expectations about the disorder and the therapy
- The words, gestures, and behavior of the therapist in the PT-patient relationship during treatments



Placebo and Nocebo Effects on Rehabilitation –Contextual Factors

- The rituality, the invasiveness and the overt application of the intervention



Placebo and Nocebo Effects on Rehabilitation –Contextual Factors

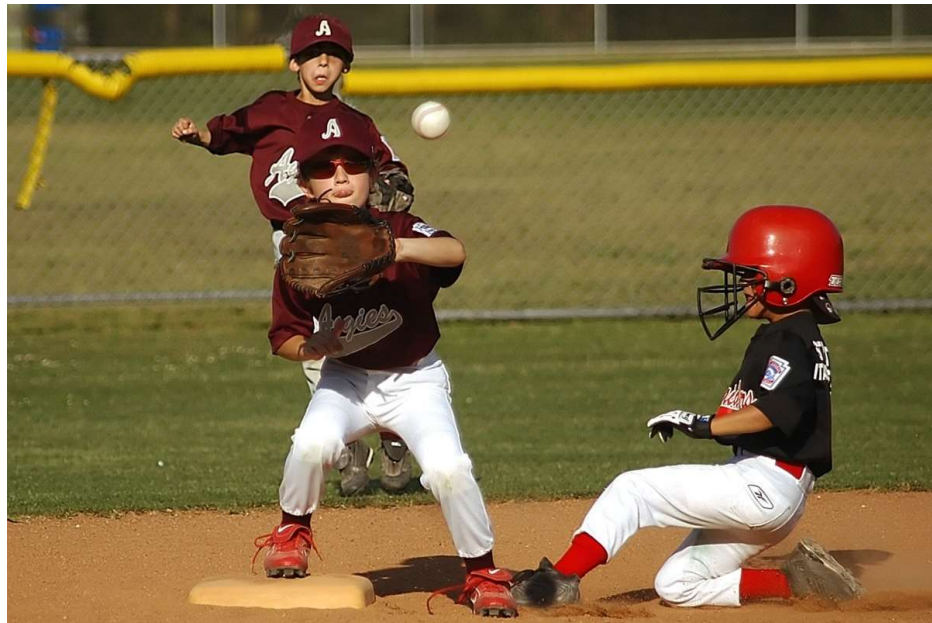
- The furniture, the architectural design and the overall impression of the clinic



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Psychological Determinants

- Psychological mechanisms in placebo, nocebo and context related effects:
 - Expectation
 - Learning process (classical conditioning and observational learning)
 - Reinforced expectations
 - Mindset and personality traits



Neurophysiological Mechanisms

- Endogenous opioid, endocannabinoid, dopaminergic systems
- Neuroimaging identified different brain region, such as;
 - Dorsolateral prefrontal cortex
 - Rostral anterior cingulate cortex
 - Periaqueductal gray
 - Dorsal horn of the spine



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Neurophysiological Mechanisms

- Despite growing interest in the physiotherapy field, the knowledge and the awareness of using contextual factors to enhance placebo and avoid nocebo effects is still limited among physiotherapists (10).
- The same unawareness has also been shown among other healthcare providers such as nurses and physicians (10).



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Contextual Factors Related to This Case

- Immediate and compassionate response by the surgeon and the orthopedic staff
- The patient was severely disabled but was treated with very positive, but very honest care, beginning immediately following surgery
- She began early physical therapy intervention that she stated was critical to her maintaining a positive attitude throughout the ordeal



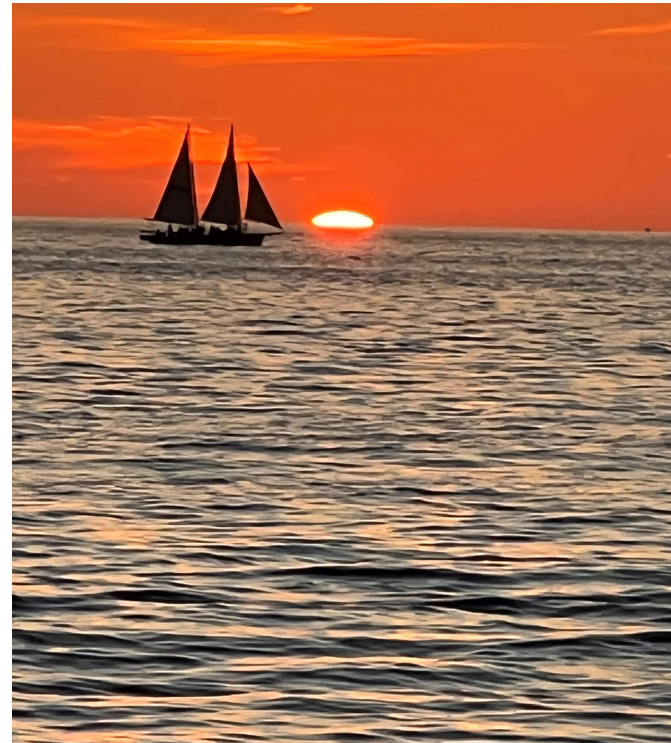
Contextual Factors Related to This Case

- Therapy and positive reinforcement from her surgeon and the orthopedic staff helped her maintain a constantly positive attitude, especially in the first four months when little progress was clinically or electromyographically measurable.
- She maintained full passive ROM of the upper extremity
- When significant return began after five months she worked diligently with PT to maximize function



Contextual Factors Related to This Case

- Open discussion of our collective experiences with nerve injuries that are not adequately addressed from the outset.



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DOUBLE-CRUSH SYNDROME: MYTH OR REALITY? ANATOMICAL EVIDENCE AND REVIEW OF THE LITERATURE

Nitz AJ

ABSTRACT

BACKGROUND: Identifying the underlying neural cause for patient's complaints is a key element of clinical examination and is supported by data derived from electroneuromyographic testing. Theoretical constructs and hypotheses, such as the *double-crush* hypothesis, have been devised to guide the clinician with helpful context for interpreting such tests. However, it is useful to periodically analyze such hypotheses *critically* when conflicting or contradictory evidence challenges the accepted construct.

DISCUSSION AND LITERATURE REVIEW: The *double-crush* hypothesis was first put forward in 1973 by Upton and McComas to account for their observation that an unusually high percentage of patients with *distal* compressive nerve pathology (e.g. carpal tunnel syndrome) were also found to have electrophysiologic evidence of *proximal* nerve pathology (e.g. cervical nerve root compression). The *double-crush* concept gained rapid acceptance as it appears to comport with the clinical observations of many clinicians who examine patients with neural complaints. The concept was also supported by initial experimental studies, notably Nemoto *et al* in 1987, who developed a dog model to test the effects of multiple sites of nerve compression. In spite of generally wide acceptance of the *double-crush* hypothesis, challenges to the logic of its underlying anatomic and physiologic rationale have been advanced, especially by Wilbourn *et al*. Differences in understanding such clinical and physiologic phenomenon (e.g. *double-crush*) are common in medical practice and often become controversial for a number of reasons. The challenge to the practicing clinician is to weigh the logical, clinical and experimental evidence that supports or refutes such concepts so that our reporting of results is most accurate and has the optimal chance of adequately guiding the referring practitioner in developing appropriate intervention. This literature review will identify clinical, anatomical and experimental research evidence that both comports with and, alternatively, challenges the *double-crush* hypothesis.

CONCLUSION: This exercise in exploring the evidence is designed to enhance the skill of interpreting relevant information that is useful for our patients in the process of making clinically important diagnostic decisions.

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37th Annual ENMG Symposium

March 27–28, 2026

Double Crush Syndrome:
Myth or Reality?

Anatomical Evidence and Review of Literature

Arthur J. Nitz, PT, PhD, ECS

Learning Objectives

- ▶ #1: Identify the key clinical and electrophysiologic findings in *support* of the double-crush syndrome diagnosis.
- ▶ #2: Describe the underlying anatomic and pathophysiologic evidence that *challenges* the double-crush syndrome designation.

Outline

I. Introduction

- a. Why do controversies exist in (ENMG) practice?
- b. How do we solve them?

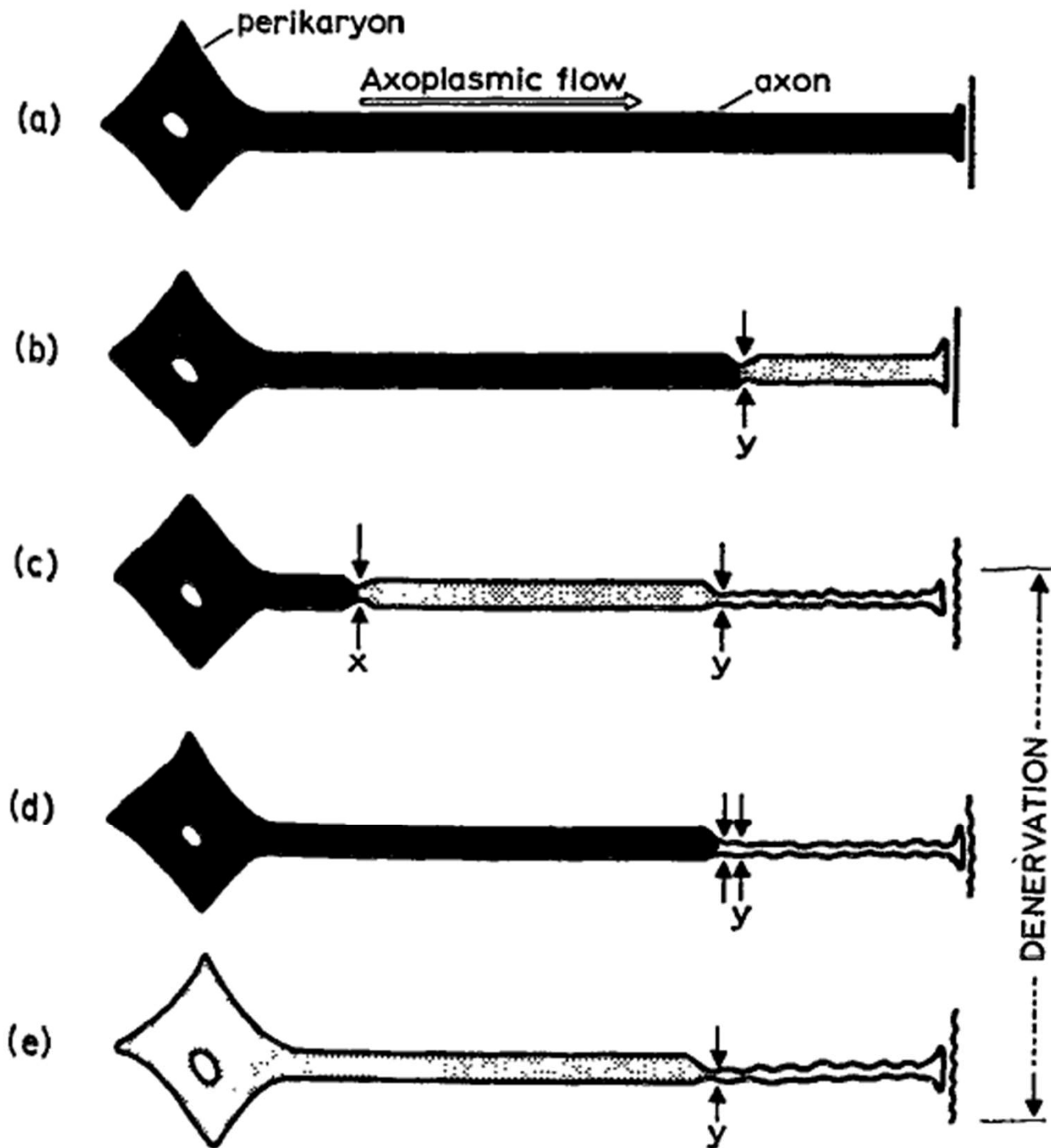
II. Clinical and electrophysiologic evidence *for* double-crush syndrome

III. Anatomical and logical evidence *against* double-crush syndrome

IV. Clinical implications and Conclusion

Upton AR, McComas AJ: The double crush in nerve entrapment syndromes. *Lancet*, 1973; 18(2):359–362

- ▶ Their basic observation
- ▶ The double crush hypothesis suggests that compression of a nerve at a proximal site renders it more likely that a contemporaneous compression on the same nerve at a distal site will have an additive effect on nerve function.
- ▶ The proposed explanation – serial constraint on axoplasmic flow



The different types of neural lesion which can give rise to denervation.

Table 1. *Common Double Crush Syndromes*

Median	Cervical radiculopathy and carpal tunnel syndrome
	Thoracic outlet and carpal tunnel syndrome
	Pronator syndrome and carpal tunnel syndrome
Ulnar	Cervical radiculopathy and cubital tunnel syndrome
	Thoracic outlet and cubital tunnel syndrome
	Cubital tunnel and Guyon's canal syndrome
Radial	Cervical radiculopathy and radial tunnel syndrome

(Typical) Double Crush Syndrome Case Presentation

- ▶ Clinical presentation
- ▶ *Expected* nerve conduction findings
- ▶ *Expected* needle EMG findings
- ▶ *Expected* interpretation

Electrophysiologic Controversies

- ▶ Electrophysiologic Clinical practice controversies
 - Over reliance on late responses for radiculopathy diagnosis (F wave, H-reflex)
 - Differentiating conduction block (intact nerve) vs. severed or degenerated axon
 - Waiting 21 days to perform EMG
- ▶ Conditions
 - Thoracic Outlet Syndrome
 - Tarsal Tunnel Syndrome
 - **Double-Crush Syndrome?**

What is the controversy for Double-crush Syndrome?

- ▶ Does it exist?
- ▶ What are the specific diagnostic criteria to establish it?
- ▶ If it exists, what is the pathophysiologic mechanism that explains its existence?

Evidence for Double Crush Syndrome

- ▶ Common sense/logic
- ▶ Clinical presentation
- ▶ Clinical studies
- ▶ Experimental (anatomical, pathophysiologic studies)

Table Some various combinations of nerve lesions reported in the literature as instances of double-crush syndrome

Proximal nerve	Distal nerve	Reference
Mental nerve	Inferior alveolar n.	18
C5, C6 myelopathy	Axillary n.	19
Cervical root	Brachial plexus (TOS)	20
	Long thoracic nerve	21
	Median n., wrist (CTS)	1,7,11,21-35
	Median n., elbow (AIN)	34,36
	Ulnar n., elbow	1,7,11,21,29-32,34,37-40
	Ulnar n., wrist	34,39
	Radial n., elbow (e.g., radial tunnel, PIN)	21,31,32,34
Brachial plexus (mostly TOS)	Median n., wrist (CTS)	11,20,25,28,29,31,32,34,41-49
	Median n., elbow	48-50
	Ulnar n., elbow	11,20,29-32,34,39,41,42,44,48,49,51-53
	Ulnar n., wrist	20,34,39,48
	Radial n., elbow (e.g., radial tunnel, PIN)	20,54
Median nerve, elbow (e.g., pronator syndrome)	Median n., wrist (CTS)	11,21,32,34,56-58
Ulnar nerve, elbow	Ulnar n., wrist	30,32,34,39,59
Radial nerve, elbow	Radial n., elbow	60
Lumbosacral root	Femoral n.	34
	Peroneal n., fibular head	29,32,34,61
	Tibial n., popliteal fossa	34
	Tibial n., foot (TTS)	32,34,62,63
Tibial nerve, leg	Tibial n., foot (TTS)	63

n. = nerve; TOS = thoracic outlet syndrome; CTS = carpal tunnel syndrome; AIN = anterior interosseous nerve; PIN = posterior interosseous nerve; TTS = tarsal tunnel syndrome.

Evidence for Double–Crush Syndrome

- ▶ Clinical and electrophysiologic studies
 - Upton and McComas, Lancet, 1973
 - Massey *et al*, South Med J, 1981
 - Hurst *et al*, J Hand Surg Br, 1985
 - Osterman, Orthop Clin North Am, 1983, 1988
 - Simpson and Fern, Orthop Clin North Am, 1996
 - Pohl *et al*, J Am Acad Orthop Surg, 2024
 - da Silva Magalhaes *et al*, Neurocir, 2019
 - Katsuura *et al*, Clin Med Insights: Arthritis and Musculoskeletal Disorders, 2020

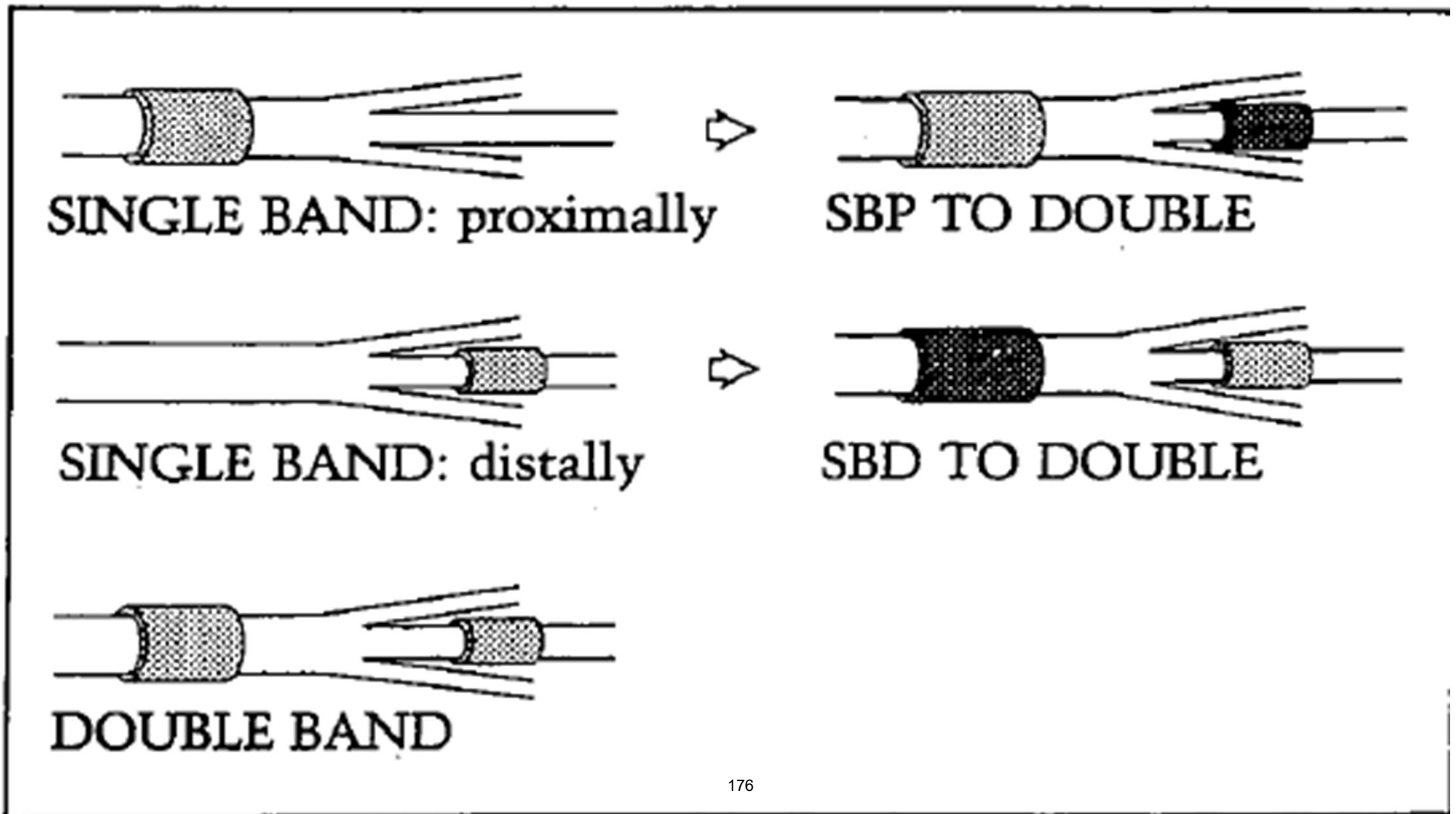
Evidence *for* Double Crush Syndrome

- ▶ Experimental/Anatomical studies
 - Nemoto *et al*, J Hand Surg Am, 1987
 - Seiler *et al*, Surg Forum, 1983
 - Mackinnon *et al*, Ann Plast Surg, 1984
 - Mackinnon and Dellon, Hand Clinic, 1986

Primary proposed explanation: Disruption of axoplasmic flow

- * anterograde
- * retrograde

Dellon, Mackinnon: Chronic nerve compression model for the Double Crush Hypothesis. Ann Plast Surg, 26(3), 1991



Evidence against Double Crush

- ▶ Logical
- ▶ Clinical presentation/clinical studies
- ▶ Anatomical
- ▶ Electrophysiologic

Challenges to the Double-Crush Hypothesis

- ▶ No standardized or validated criteria to define or diagnose DC syndrome
- ▶ No true consensus regarding prevalence or general epidemiology (10% to +70%)
 - Upton and McComas – 76% incidence of CR and CTS
 - Morgan and Wilbourn (12,000 patients) – CR concomitant with CTS or cubital tunnel syndrome – <1%
 - What accounts for that amount of variability?
 - Used rigid clinical, electrodiagnostic and anatomic criteria
 - Pre-existing bias?

Further challenges to the Double Crush Hypothesis

▶ Nomenclature

- Triple crush?
- Quadruple crush?
- “Reverse” double crush?

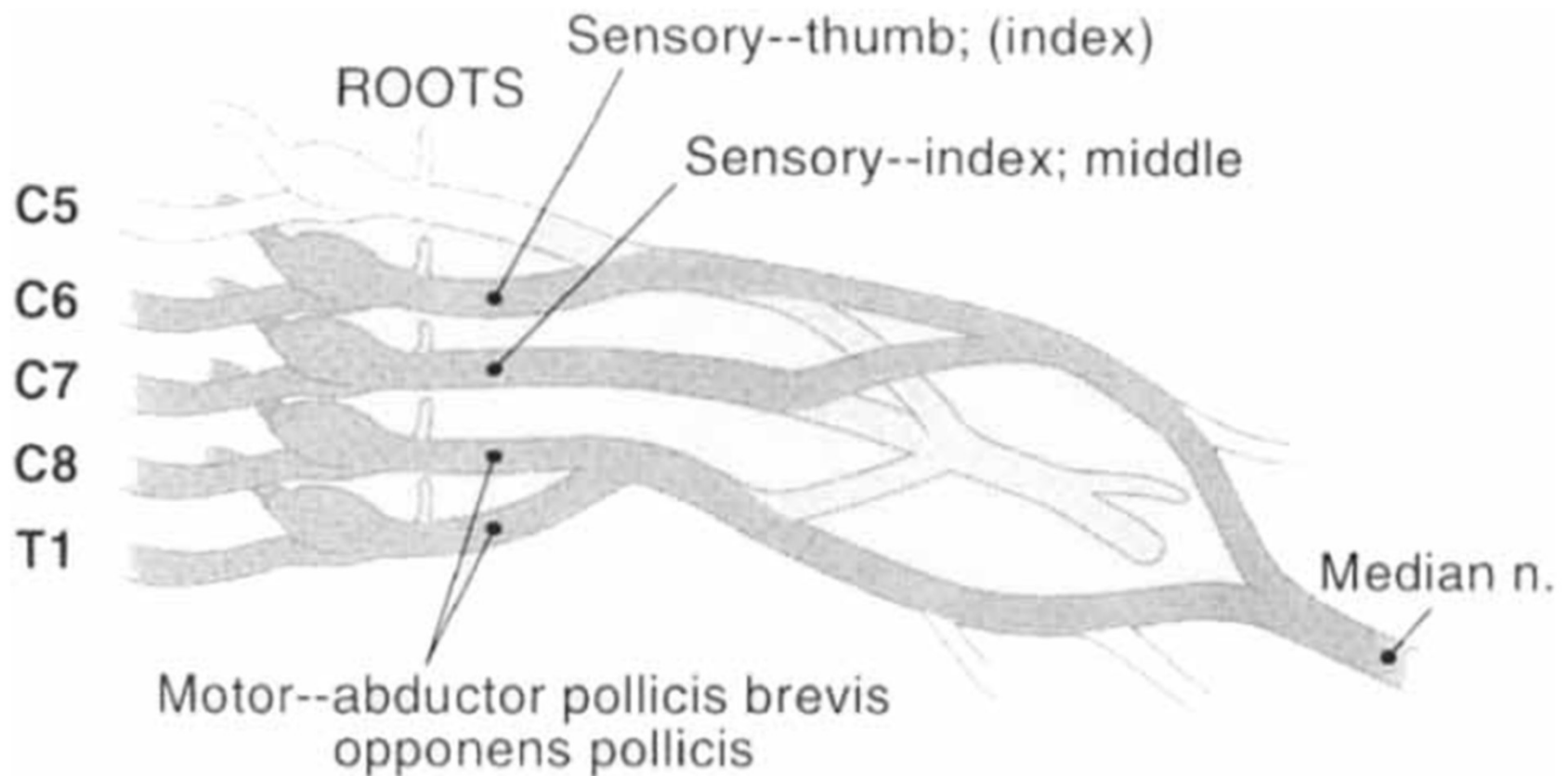
▶ Crush

- What about stretch?
- Non-mechanical – metabolic compromise

Additional challenges to Double Crush Hypothesis

- ▶ Electrophysiologic challenges
 - CTS – primary finding is evidence of demyelination
 - Proposed DC pathophysiology is related to altered axoplasmic flow of the axon and attended by needle EMG evidence of denervation
- ▶ Anatomic challenges
 - Distribution of median nerve motor and sensory fibers to the hand that traverse the brachial plexus
 - Sensory fibers primarily from C5, 6; motor fibers to the thumb primarily from C8, T1
 - This anatomic reality violates the stipulation that the proximal and distally compressed fibers be in continuity

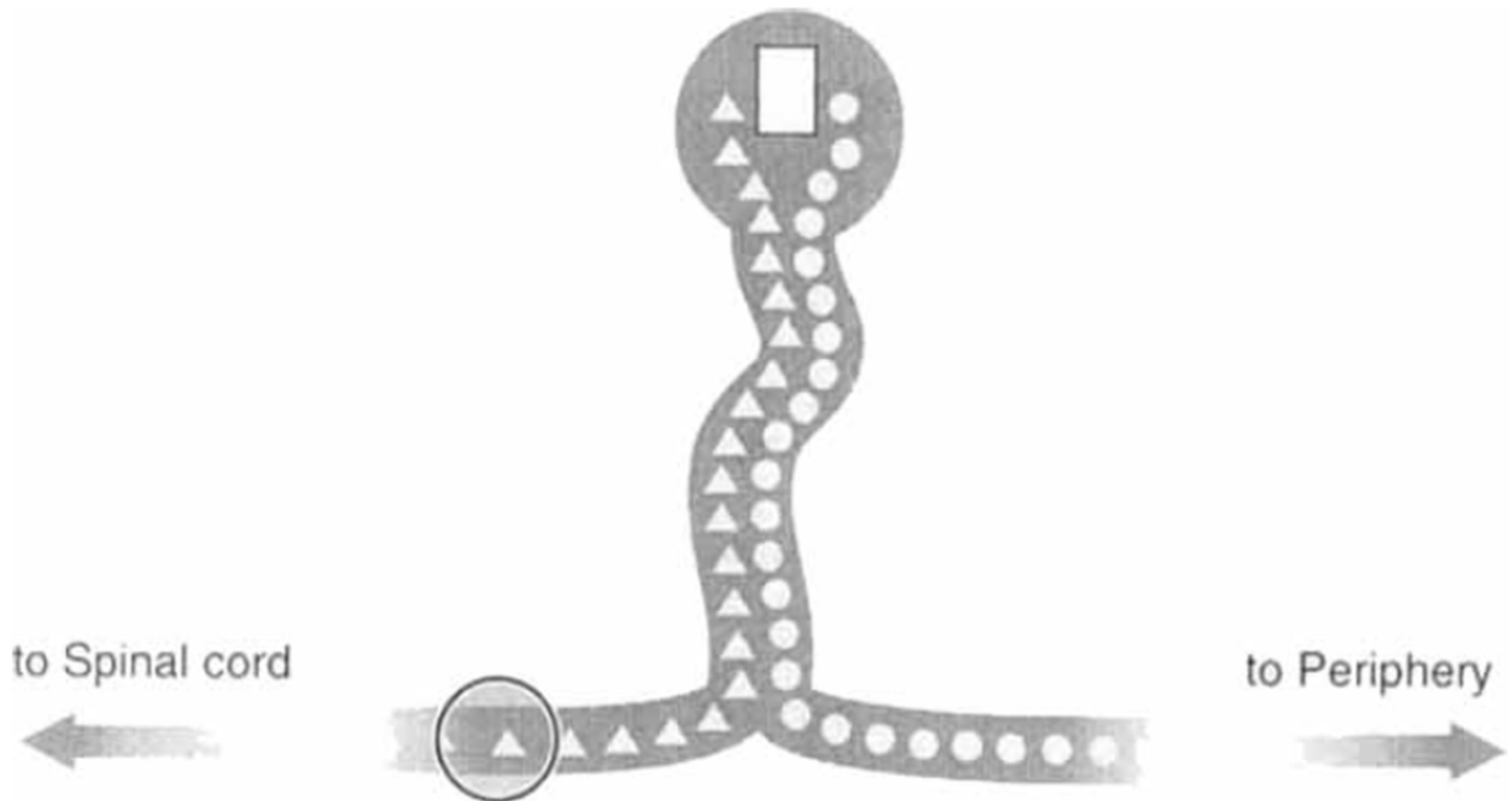
Challenges to *Double-Crush* Syndrome



Yet another challenge to Double Crush Hypothesis

- ▶ By “definition” DC syndrome requires a *proximal and distal* compression site along the same axons resulting in altered axoplasmic flow.
- ▶ Proximal compression (intra-spinal for CR) is proximal to the DRG cell body. The pre- and post-ganglionic sensory fibers are not anatomically continuous, even though they share the same cell body.
- ▶ This independence of the centrally and peripherally directed sensory fibers extends to their axoplasmic transport systems.

Challenges to *Double-Crush* Syndrome



So if its *not* Double-Crush Syndrome, then what is it?!

- ▶ Naam, AASN, 2016 Subclinical involvement of multiple nerve structures
- ▶ Novak and Mackinnon, J Hand Surg (Am), 1994 Posture related upper extremity pain
- ▶ Nirschl, Va Med Mon, 1969 “mesenchymal syndrome”
- ▶ Cohen *et al*, J Hand Surg (Am), 2016 Multifocal neuropathy
 - Note: Not multifocal motor neuropathy (autoimmune disorder)

Identifying the problem

DCS or MFN(?)

- ▶ Know the anatomy
 - Cutaneous distribution
 - Motor distributions
- ▶ Detailed physical exam
 - Ten-ten test for sensation
 - Direct palpation over entrapment sites
 - Careful motor exam –subtle, “fatiguing motor weakness”
 - Scratch collapse test

Conclusion; solutions

- ▶ Is Double Crush a genuine clinical entity?
- ▶ What is its definition?
- ▶ What are the electrophysiologic criteria?
- ▶ If it truly exists, what is the underlying pathophysiologic mechanism?
- ▶ If it does not exist, how do we explain the apparent ubiquitous acceptance of this diagnostic category?
- ▶ What do we do going forward?

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GENERALIZED PERIPHERAL POLYNEUROPATHY SECONDARY TO VITAMIN B12 DEFICIENCY: A CASE REPORT

Greathouse D

ABSTRACT

BACKGROUND: Polyneuropathy is an acquired or hereditary disease process affecting multiple peripheral nerves in multiple extremities.¹⁻³ Vitamin B12 deficiency is a risk factor for development of acquired peripheral polyneuropathy as it can cause demyelination of the motor and sensory large (Type A) fibers.⁴ This case details the subjective and objective clinical exams, electrophysiologic testing, and interventions for a 34-year-old female with insidious and progressive sensory and motor loss affecting the lower greater than upper extremities that rendered her incapable of ambulating. Initial electrophysiologic testing demonstrated a severe, generalized peripheral polyneuropathy (GPPN) in bilateral lower extremities (BLE) > bilateral upper extremities (BUE) that was characterized by both demyelinating and axonal features. Subsequent evaluation by a neurologist determined that a vitamin B12 deficiency caused her peripheral polyneuropathy.⁴

DISCUSSION: Polyneuropathy is a disease process involving a number of peripheral nerves (multiple nerves in multiple extremities) and consists of a triad of sensory changes in a glove or stocking distribution, distal weakness, and hyporeflexia. Electrophysiological testing (including nerve conduction studies and diagnostic EMG) is performed to confirm the findings of a thorough history and physical examination and may identify neurological injury associated with generalized peripheral polyneuropathy.¹⁻⁴ Vitamin B12 deficiency may cause demyelination or axonal loss of nerves in the peripheral and central nervous systems.⁷⁻⁹

CONCLUSIONS: Insidious onset of severe motor weakness with EMG/NCS findings suggesting a generalized peripheral polyneuropathic process warrants immediate referral to neurology for further evaluation and treatment.¹⁻⁴ Measuring vitamin B12 serum levels with additional testing of methylmalonic acid (MMA) is useful to diagnose metabolic vitamin B12 deficiency in patients with polyneuropathy.⁷

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Generalized Peripheral Polyneuropathy Secondary to Vitamin B12 Deficiency

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Generalized Peripheral Polyneuropathy

- Polyneuropathy is a hereditary or acquired disease process involving a number of peripheral nerves in multiple extremities and is often accompanied by sensory changes in a glove or stocking distribution, distal greater than (>) proximal weakness and hyporeflexia.¹⁻³
- The possible sources of acquired generalized peripheral polyneuropathy (GPPN) are numerous and can include vitamin deficiency.¹⁻⁴
- GPPN associated with vitamin B12 deficiency typically affects lower > upper extremities with both demyelinating and axonal features and most commonly causes sensory loss but can also cause motor weakness.⁴
- Vitamin B12 deficiency is more common with in older adults and can be caused by malabsorption, insufficient intake, history of gastric surgery, pernicious anemia and medications such as proton pump inhibitors and metformin.⁴

History (Subjective)

- A 34-year-old female was referred by her orthopaedic surgeon for electrophysiological testing of bilateral lower extremities (BLE) and bilateral upper extremities (BUE) with a referring diagnosis of **weakness BLE greater than BUE**.
- Two months prior to electrophysiological testing, the patient had an insidious onset of **weakness** in her feet (toe flexion, toe extension, and foot intrinsics), ankles and legs that progressed proximally to her thighs, hands, wrists and elbows.
- **The weakness progressed over the following month, rendering her unable to ambulate two months after the onset of symptoms.**

History

- One month after onset of symptoms, she also noted the development **numbness/tingling (N/T) and paresthesia** in the distal > proximal aspects of BUE > BLE without clear dermatomal or peripheral nerve pattern.
- Due to the severity and progressive nature of her weakness and diminishing functional ability, she sought care from her primary care provider (PCP), an emergency room physician, and an orthopaedic physician who had seen her for knee pain in the past. She was not given a diagnosis for the cause of her weakness by any of these practitioners.

History (Subjective)

- The patient reported mild neck pain but denied low back pain or radicular pain in either BUE or BLE. She had a history of mild left knee pain but otherwise denied any pain in BUE or BLE.
- The patient described N/T in the bilateral feet, legs and distal thighs in a stocking distribution and in hands, forearms and distal arms in a glove distribution.
- She complained of weakness in the distal > proximal BLE (including bilateral foot, ankle, knee and hip motions) and distal > proximal BUE (including hand grip and fine movements, wrist and elbow motions).
- She denied injury or trauma to the neck, lumbar spine, BUE and BLE. The patient denied bowel or bladder incontinence, problems with vision, chewing or swallowing or facial muscle weakness.

Review of Systems

- The patient reported that her general medical health was good.
- She denied diabetes, heavy metal exposure, thyroid disease, renal disease, alcohol abuse, or family history of neuromuscular disease.
- She also denied any recent surgeries or recent infection or acute illness in the past year.
- A review of systems was non-contributory for cardiovascular, pulmonary, gastrointestinal, genitourinary or endocrine disease.

Physical Exam

- The patient presented in a wheelchair and was completely dependent with all transfers to/from the exam table.
- Non-functional weakness (< 1/5) was noted in all distal muscles tested in BLE including the bilateral foot intrinsics, toe, forefoot, and ankle motions (Table 1). Bilateral knee and hip strength were decreased but functional (> 3-/5).
- Non-functional weakness (2/5) was noted in the bilateral thenar, hypothenar, and ulnar intrinsic muscles (Table 2). Bilateral long digit/thumb flexors and extensors, wrist and elbow motions were weak but functional (> 3-/5).

TABLE 1: Motor Testing of BLE (12-10-2024)

Hip		Left	Right
	Flexion	3-/5	3-/5
	Extension	3-/5	3-/5
	Abduction	3-/5	3-/5
	Adduction	3/5	3/5
	External rotation	3/5	3/5
	Internal rotation	3/5	3/5
Knee			
	Flexion	3-/5	3-/5
	Extension	3-/5	3-/5
Ankle			
	Dorsiflexion	0/5	0/5
	Plantar flexion	1/5	0/5
Forefoot			
	Eversion	0/5	0/5
	Inversion	0/5	0/5
Toe			
	Flexion (FDL and FHL)	1/5	1/5
	Extension (EHL and EDL)	0/5	0/5
	Extension (EDB)	0/5	0/5
Foot Intrinsic		0/5	0/5

Abbreviations: FDL, flexor digitorum longus; FHL, flexor hallucis longus; EHL, extensor hallucis longus; EDL, extensor digitorum longus; EDB, extensor digitorum brevis.

TABLE 2: Motor Testing of BUE (12-10-2024)

Shoulder		Left	Right
	Flexion	4/5	4/5
	Extension	4/5	4/5
	Abduction	4/5	4/5
	Adduction	4/5	4/5
	External rotation	4/5	4/5
	Internal rotation	4/5	4/5
Elbow			
	Flexion	3+/5	3+/5
	Extension	3/5	3/5
Forearm			
	Pronation	3/5	3/5
	Supination	3/5	3/5
Wrist			
	Flexion	3-/5	3-/5
	Extension	3-/5	3-/5
Digit 2-5			
	Flexion (FDS and FDP)	3/5	3/5
	Extension (ED, EDMB and EI)	3-/5	3-/5
Digit 1			
	Flexion (FPL)	3/5	3/5
	Extension (EPL and EPB)	3-/5	3-/5
	Thenar (APB and OP)	2/5	2/5
Digit 5			
	Flexion (FDMB)	2/5	2/5
	Abduction (ADM)	2/5	2/5
	Opposition (ODM)	2/5	2/5
Intrinsics		2/5	2/5

Abbreviations: FDS, flexor digitorum superficialis; FDP, flexor digitorum profundus; ED, extensor digitorum; EDMB, extensor digitorum minimi brevis; EI, extensor indicis; FPL, flexor pollicis longus; EPL, extensor pollicis longus; EPB, extensor pollicis brevis (EPB); APB, flexor pollicis brevis; OP, opponens pollicis; FDMB, flexor digiti minimi brevis; ADM, abductor digiti minimi; ODM, opponens digiti minimi; Intrinsics, ulnar innervated palmar and dorsal interossei and adductor pollicis.

Physical Exam

- Muscle stretch reflexes (MSRs) of the bilateral knee jerk (**L2-L3-L4**), ankle jerk (**S1**), brachioradialis (C5-**C6**-C7), biceps brachii (C5-**C6**), and triceps brachii (C6-**C7**-C8) were present and equal (2/4).
- The Babinski and Chaddock pathological reflexes were absent in BLE, and the Hoffman pathological reflex was absent in BUE.
- There was normal sensation to light touch and pain (pin prick) in all peripheral nerves and dermatomes in BUE and BLE.
- There was normal vibration testing (initial perception and time - 10 sec) on BUE (thumb – D1, radial styloid, and olecranon) and BLE (great toe – D1, medial malleolus, and tibial tuberosity).
- The bilateral pedal, posterior tibial, radial, and ulnar artery pulses were palpable and equal.

Physical Exam

- The patient had mild decreased cervical motion in all planes, but she denied neck pain or radicular pain in BUE.
- Provocative testing of the cervical and lumbar spine with Spurling's test and straight leg raise tests were normal.
- Motor and sensory testing of cranial nerves III, IV, V, VI, VII, IX, X, XI and XII was normal.

Nerve Conduction Studies - Summary

- There were **no recordable sensory responses** in the bilateral superficial fibular and right sural nerves (Table 3).
- There were **prolonged distal sensory latencies** in the bilateral median and ulnar, left superficial radial, and left sural nerves.
- Bilateral deep fibular nerves, recorded at both the extensor digitorum brevis and tibialis anterior muscles, and the left tibial nerve, recorded from the abductor hallucis, had **no recordable motor response** at distal or proximal stimulation sites.
- There were **prolonged distal motor latencies, decreased compound motor action potential amplitudes and slow distal conduction velocities** recorded in the bilateral median and ulnar nerves and right tibial nerve.
- The F wave responses were **absent** in the right tibial nerve and prolonged in the bilateral median and ulnar nerves (Table 3).
- Extremity temperature was maintained throughout NCS testing (wrist at or above 32° C; ankle at or above 29° C).

TABLE 3: Sensory, Motor and Central Conduction Nerve Conduction Studies in BUE and BLE (12-10-2024)

Nerve Conduction Studies

Anti Sensory Summary Table

Site	NR	Peak (ms)	Norm Peak (ms)	P-T Amp (μV)	Norm P-T Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left SFN Anti Sensory (Ant Lat Mall)											
12 cm	NR		<4.0		>5.0	12 cm	Ant Lat Mall		12.0		>32
Right SFN Anti Sensory (Ant Lat Mall)											
12 cm	NR		<4.0		>5.0	12 cm	Ant Lat Mall		12.0		>32
Left Superficial Radial Anti Sensory (Base 1st Digit)											
D1		3.2	<2.7	20.2	>	D1	Base 1st Digit	3.2	10.0	31	
Right Superficial Radial Anti Sensory (Base 1st Digit)											
D1		2.7	<2.7	20.2	>	D1	Base 1st Digit	2.7	10.0	37	
Left Sural Anti Sensory (Lat Mall)											
Calf		6.8	<4.3	3.7	>5.0	Calf	Lat Mall	6.8	14.0	21	>32
Right Sural Anti Sensory (Lat Mall)											
Calf	NR		<4.3		>5.0	Calf	Lat Mall		14.0		>32

Ortho Sensory Summary Table

Site	NR	Peak (ms)	Norm Peak (ms)	P-T Amp (μV)	Norm P-T Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Ortho Sensory (Wrist)											
Palm		2.6	<2.2	115.6	>15	Palm	Wrist	2.6	8.0	31	
D2		4.4	<3.6	28.3	>15	D2	Wrist	4.4	14.0	32	
Right Median Ortho Sensory (Wrist)											
Palm		2.5	<2.2	120.5	>15	Palm	Wrist	2.5	8.0	32	
D2		4.6	<3.6	22.1	>15	D2	Wrist	4.6	14.0	30	
Left Ulnar Ortho Sensory (Wrist)											

Knee	27.9		0.3						
Left Ulnar Motor (Abd Dig Minimi)									
Wrist	5.1	<3.6	4.1	>5	B Elbow	Wrist	5.7	24.0	42 >50
B Elbow	10.8		2.9		A Elbow	B Elbow	2.5	13.0	52 >50
A Elbow	13.3		2.9						
Right Ulnar Motor (Abd Dig Minimi)									
Wrist	5.7	<3.6	4.7	>5	B Elbow	Wrist	6.4	25.0	39 >50
B Elbow	12.1		4.2		A Elbow	B Elbow	2.4	14.0	58 >50
A Elbow	14.5		4.2						

F Wave Studies

NR	F-Lat (ms)	Lat Norm (ms)	L-R F-Lat (ms)	L-R Lat Norm
Left Median (Mrkrs) (Abd Poll Brev)				
	41.68	<32	0.00	<2.2
Right Median (Mrkrs) (Abd Poll Brev)				
	41.68	<32	0.00	<2.2
Right Tibial (Mrkrs) (Abd Hallucis)				
NR		<58		<5.7
Left Ulnar (Mrkrs) (Abd Dig Min)				
	40.10	<32	0.83	<2.5
Right Ulnar (Mrkrs) (Abd Dig Min)				
	39.27	<32	0.83	<2.5

Abnormal values highlighted in red.

Abbreviations: Amp, amplitude; Anti, antidromic; BUE, both upper extremities; cm, centimeter; D1, digit 1 (thumb); D2, digit 2 (index finger); D5, digit 5 (little finger); dist, distance; ms, millisecond; m/sec, meter/second; Mrkrs, markers; NR, no response; norm, normal; O-P, onset to peak; Ortho, orthodromic; P-T, peak to trough; vel, velocity; μ V, microvolt;

Palm	2.5	<2.2	10.2	>10	Palm	Wrist	2.5	8.0	32
D5	4.2	<3.5	9.1	>10	D5	Wrist	4.2	14.0	33
Right Ulnar Ortho Sensory (Wrist)									
Palm	2.4	<2.2	27.1	>10	Palm	Wrist	2.4	8.0	33
D5	4.4	<3.5	12.1	>10	D5	Wrist	4.4	14.0	32

Motor Summary Table

Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Deep Fibular Motor (Ext Dig Brev)											
Ankle	NR		<6.6		>2	B Fib	Ankle		0.0		>40
B Fib	NR										
Right Deep Fibular Motor (Ext Dig Brev)											
Ankle	NR		<6.6		>2	B Fib	Ankle		0.0		>40
B Fib	NR										
Left DFN TA Motor (Tib Ant)											
Fib Head	NR		<4.2								
Right DFN TA Motor (Tib Ant)											
Fib Head	NR		<4.2								
Left Median Motor (Abd Poll Brev)											
Wrist		7.6	<4.2	4.5	>5	Elbow	Wrist	9.2	29.0	32	>50
Elbow		16.8		2.5							
Right Median Motor (Abd Poll Brev)											
Wrist		7.3	<4.2	3.7	>5	Elbow	Wrist	8.3	29.0	35	>50
Elbow		15.6		1.2							
Left Tibial Motor (Abd Hall Brev)											
Ankle	NR		<6.8		>2	Knee	Ankle		0.0		>40
Knee	NR										
Right Tibial Motor (Abd Hall Brev)											
Ankle		12.6	<6.8	0.4	>2	Knee	Ankle	15.3	41.0	27	>40

EMG - Summary

- Increased insertional activity and abnormal spontaneous electrical activity (fibrillation potentials and positive waves) at rest were noted in all the upper and lower extremity muscles tested except for the bilateral deltoid and left triceps brachii muscles (Table 4).
- Fibrillation potentials observed were large amplitude ($> 100 \mu\text{V}$), suggesting acute denervation.¹⁻³
- Normal motor unit morphology (shape, duration and amplitude) was present in all muscles tested during voluntary contraction in BUE and BLE.
- The recruitment and interference patterns were decreased during maximal voluntary contraction in the distal $>$ proximal muscles of BLE $>$ BUE including no recordable motor unit potentials (MUP) in the bilateral fibularis longus and EHL, and right gastrocnemius and 1 MUP in the bilateral tibialis anterior and left gastrocnemius (Table 4.)

TABLE 4: Needle Electromyography Results in Muscles Tested in BUE and BLE (12-10-2024)

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	1stDorInt	Ulnar	C8-T1	Incr	1+*	2+	Nml	Nml	0	Reduced	50%	
Right	Abd Poll Brev	Median	C8-T1	Incr	1+*	2+	Nml	Nml	0	Reduced	50%	
Right	PronatorTeres	Median	C6-7	Incr	2+*	2+	Nml	Nml	0	Reduced	75%	
Right	Biceps	Musculocut	C5-6	Incr	2+*	2+	Nml	Nml	0	Reduced	75%	
Right	Triceps	Radial	C6-7-8	Incr	1+*	2+	Nml	Nml	0	Reduced	75%	
Right	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	1stDorInt	Ulnar	C8-T1	Incr	1+*	2+	Nml	Nml	0	Reduced	50%	
Left	Abd Poll Brev	Median	C8-T1	Incr	1+*	2+	Nml	Nml	0	Reduced	50%	
Left	PronatorTeres	Median	C6-7	Incr	2+*	2+	Nml	Nml	0	Reduced	50%	
Left	Biceps	Musculocut	C5-6	Incr	2+*	2+	Nml	Nml	0	Reduced	75%	
Left	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	AntTibialis	Deep Fibular	L4-5	Incr	2+*	2+	Nml	Nml	0	Reduced		1 MUP
Right	Fibularis Longus	Super Fibular	L5-S1	Incr	2+*	3+	Nml	Nml	0	Reduced		No MUPs
Right	ExtHallLong	Deep Fibular	L5, S1	Incr	2+*	2+	Nml	Nml	0	Reduced		No MUPs
Right	RectFemoris	Femoral	L2-4	Incr	2+*	2+	Nml	Nml	0	Reduced	25%	
Left	AntTibialis	Deep Fibular	L4-5	Incr	2+*	3+	Nml	Nml	0	Reduced		1 MUP
Left	Fibularis Longus	Super Fibular	L5-S1	Incr	2+*	3+	Nml	Nml	0	Reduced		No MUPs
Left	ExtHallLong	Deep Fibular	L5, S1	Incr	2+*	3+	Nml	Nml	0	Reduced		No MUPs
Left	RectFemoris	Femoral	L2-4	Incr	2+*	2+	Nml	Nml	0	Reduced	25%	
Left	Gastroc	Tibial	S1-2	Incr	2+*	3+	Nml	Nml	0	Reduced		1 MUP
Left	BicepsFemL	Tibial	L5-S2	Incr	1+*	2+	Nml	Nml	0	Reduced	50%	
Right	Gastroc	Tibial	S1-2	Incr	2+*	3+	Nml	Nml	0	Reduced		No MUPs
Right	BicepsFemL	Tibial	L5-S2	Incr	2+*	2+	Nml	Nml	0	Reduced	75%	

Conclusion – Electrophysiological Testing

- There was electrophysiological evidence of a generalized, symmetrical, length dependent, sensorimotor, peripheral polyneuropathy in BLE > BUE with both significant axonal loss and demyelination. **Acute denervation** with reduced recruitment and interference patterns was noted in all muscles tested in BLE and BUE except for the bilateral deltoid and left triceps brachii muscles.
- Although the findings on the history and physical exam did not suggest focal cervical or lumbosacral radiculopathy, the involvement of proximal upper and lower extremity muscles on the initial EMG examination might be suggestive of **polyradiculoneuropathy** (involvement of both nerve roots and peripheral nerves) which can be seen in chronic inflammatory demyelinating polyneuropathy (CIDP) and B12 related neuropathies.
- Given the diffuse EMG abnormalities and motor weakness, it was difficult to fully exclude **motor neuron disease**. However, the abnormal sensory findings on nerve conduction testing and the absence of fasciculation potentials on EMG, as well as the absence of cranial nerve involvement or upper motor neuron signs suggested that polyneuropathic disease was more likely.
- There was no electrophysiological evidence on this exam suggestive of **myopathic** disease in the muscles tested in BUE and BLE.

Interventions

- One month following the electrophysiological testing, the patient was evaluated by a **neurologist who specialized in neuromuscular disease**.
- She was subsequently **admitted to a large specialty hospital for additional evaluation** including magnetic resonance imaging, lumbar puncture and blood work.
- Electrophysiological testing of BUE and BLE was not repeated.
- An initial diagnosis of **chronic inflammatory demyelinating polyneuropathy (CIDP)** was explored, but after a 6-day intervention of intravenous immunoglobulin fusion therapy (IVIG) without significant improvement, the neurologist determined the GPPN was not due to CIDP.
- The patient was diagnosed with a **vitamin B12 deficiency** due to her **low serum B12 and elevated methylmalonic acid (MMA) levels**.
- The neurologist confirmed that the generalized peripheral polyneuropathy in BLE > BUE was secondary to vitamin B12 deficiency.
 - **The patient was treated with vitamin B12 supplementation with mild improvement noted in strength in BLE and BUE.**
 - After a 2-week hospitalization, the patient was admitted to a rehabilitation facility for two weeks.

Outcome/Follow-Up

- **Three months** following the initial electrophysiological evaluation, the patient reported she “has continued to improve slowly” with some increase in strength in BLE and BUE.
 - She still uses a wheelchair for community mobility but was independent with transfers.
 - She was using a walker for home ambulation and noted that “my balance is diminished”.
 - She was awaiting bilateral ankle foot orthoses for persistent foot drop.
 - The patient was scheduled to begin outpatient physical therapy soon.
- **Seven months** following the initial EMG and NCS testing, the patient was referred by her PCP for repeat electrophysiological testing.
 - She reported gradual improvements in function and strength in BUE > BLE since the inpatient rehab and continued outpatient physical therapy and a home exercise program.
 - She was independent with transfers and has been using a walker for ambulation for the past two months.
 - The patient had a pulmonary embolus during her inpatient rehab and was on Plavix and Eliquis.
 - She has been referred to a neuromuscular disease clinic at a nearby university hospital.
 - The electrophysiologic evaluation performed seven months following the initial EMG and NCS testing is found in Appendix 1.

Discussion

- This case details the initial presentation and subsequent neurological recovery over a nine-month period in a healthy 34-year female with GPPN attributed to vitamin B12 deficiency.
- The patient's severe and insidious strength loss and notable functional impairment upon initial exam suggested a systemic neuromuscular disease process rather than focal radiculopathy or mononeuropathy.
- The sensory impairment on clinical exam and absent sensory nerve conduction studies suggested that a motor neuron disease or muscle disease, such as myopathy, was less likely.
- Given the severe GPPN demonstrated on electrophysiological testing and the absence of family history of neuromuscular disease, a hereditary polyneuropathic process was less likely and possible sources of acquired polyneuropathy were considered.

Discussion

- The neuromuscular disease expert initially treated this patient for **suspected chronic inflammatory demyelinating polyradiculopathy (CIDP)**.
- This patient had a presentation that suggested CIDP, to include progressive, symmetric, proximal and distal motor weakness in the upper and lower extremities and sensory involvement in at least two limbs.⁷
- In addition, the patient's weakness had developed over at least 8 weeks, making Guillain Barre less likely.⁷
- Nerve conduction study findings also met criteria for suspected CIDP⁷, to include prolonged distal motor latencies >50% above the upper limit of normal (right tibial, bilateral ulnar and bilateral median nerves), decreased motor nerve conduction velocity >30% below the lower limit of normal (right tibial, bilateral median nerves), prolonged F waves >20% above the upper limit of normal (bilateral ulnar and median nerves) and sensory nerve conduction abnormalities in at least two nerves (noted in most BUE and BLE sensory nerves tested).
- An objective treatment response to immunomodulatory agents, such as IVIg, supports the diagnosis of CIDP.⁷ **This patient reported no significant change in her function with IVIg, suggesting another possible cause for the GPPN and leading to the patient's diagnosis of a vitamin B12 deficiency.**
- **Improvement of neurological impairments after B12 supplementation further supported this as the cause for her GPPN.**

Discussion

- [Leishear et al⁴](#) studied the relationship of vitamin B12 and sensory and motor peripheral nerve function in asymptomatic older adults.
 - This multi-center study concluded that B12 deficiency or low serum B12 was associated with worse sensory and motor peripheral nerve function as measured by diminished amplitude and nerve conduction velocity in the fibular motor nerve, diminished vibratory sense or diminished monofilament detection.⁴
- In addition to measuring vitamin B12 serum levels to identify absolute deficiency, [additional testing of methylmalonic acid \(MMA\), a functional marker of B12 deficiency, can help diagnose metabolic B12 deficiency.](#)⁸
 - In a retrospective cohort study of 331 patients, measuring vitamin B12 serum levels in addition to MMA, was useful in diagnosing metabolic vitamin B12 deficiency in patients with polyneuropathy.⁸
 - At the time of the second electrophysiological testing, the patient stated her “MMA values were elevated,” a finding consistent with metabolic vitamin B12 deficiency.

Discussion

- [Huang et al⁹](#) published a case report that included serial nerve conduction studies of a 22-year-old vegetarian with acute, severe polyneuropathy secondary to vitamin B12 deficiency.
 - This patient presented with weakness and N/T of the distal limbs, and results of the electrophysiological testing showed an axonal type sensory-motor polyneuropathy.
 - The patient received both oral and intramuscular injection of vitamin B12, and after 14.5 months, the patient demonstrated improved strength of the distal muscles in BLE and BUE and improved motor and sensory amplitudes on nerve conduction studies.⁹
- On follow-up, our patient had similar improvements in nerve conduction testing in the BUE, as well as decreased evidence of axonopathy on EMG examination of proximal extremity muscles.

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APPENDIX 1: EMG/NCS Reevaluation (7 months following initial EMG/NCS testing)

Subjective:

- The patient denied neck pain, lower back pain or radicular pain in BUE or BLE.
- The patient had bilateral wrist pain but denied other pain in BUE and BLE.
- She reported that the previous N/T in BUE and BLE had resolved.
- Her strength and function have improved in the proximal > distal BUE and BLE, but she continued to have non-functional strength in bilateral ankle, forefoot and toe motions.
- She denied problems with bowel/bladder incontinence, breathing, chewing, swallowing or vision.

APPENDIX 1: EMG/NCS Reevaluation (7 months following initial EMG/NCS testing)

Physical Examination:

Motor strength improvements were demonstrated in BLE with normal strength in the bilateral hip and knee motions but continued non-functional strength in the bilateral ankle, forefoot and toe motions (Table 5).

Significant strength improvements were demonstrated in BUE with normal strength in the bilateral shoulder, elbow, forearm, wrist and finger flexor motions with improved strength in the thenar, hypothenar, and intrinsic ulnar innervated muscles in both hands (Table 6).

The ankle jerk (S1) MSRs were now absent in BLE.

MSRs of the bilateral brachioradialis, biceps brachii, triceps brachii, and knee jerk were present and equal.

Hoffman (BUE) and Babinski (BLE) pathological reflexes were absent.

Light touch and pain (pinprick) sensation remains normal in BUE and BLE.

TABLE 5: Motor Testing of BLE (6-30-2025)

Hip		Left	Right
	Flexion	5/5	5/5
	Extension	5/5	5/5
	Abduction	5/5	5/5
	Adduction	5/5	5/5
	External rotation	5/5	5/5
	Internal rotation	5/5	5/5
Knee			
	Flexion	5/5	5/5
	Extension	5/5	5/5
Ankle			
	Dorsiflexion	0/5	0/5
	Plantar flexion	2/5	2/5
Forefoot			
	Eversion	0/5	0/5
	Inversion	2/5	2/5
Toe			
	Flexion (FDL and FHL)	1/5	1/5
	Extension (EHL and EDL)	0/5	0/5
	Extension (EDB)	0/5	0/5
Foot Intrinsic		0/5	0/5

Abbreviations: FDL, flexor digitorum longus; FHL, flexor hallucis longus; EHL, extensor hallucis longus; EDL, extensor digitorum longus; EDB, extensor digitorum brevis.

TABLE 6: Motor Testing of BUE (6-30-2025)

Shoulder		Left	Right
	Flexion	5/5	5/5
	Extension	5/5	5/5
	Abduction	5/5	5/5
	Adduction	5/5	5/5
	External rotation	5/5	5/5
	Internal rotation	5/5	5/5
Elbow			
	Flexion	5/5	5/5
	Extension	5/5	5/5
Forearm			
	Pronation	5/5	5/5
	Supination	5/5	5/5
Wrist			
	Flexion	5/5	5/5
	Extension	5/5	5/5
Digit 2-5			
	Flexion (FDS and FDP)	5/5	5/5
	Extension (ED, EDMB and EI)	3-/5	3/5
Digit 1			
	Flexion (FPL)	5/5	5/5
	Extension (EPL and EPB)	3-/5	3-/5
	Thenar (APB and OP)	2/5	2/5
Digit 5			
	Flexion (FDMB)	3/5	4/5
	Abduction (ADM)	3/5	4/5
	Opposition (ODM)	3/5	4/5
Intrinsics		3/5	4/5

Abbreviations: FDS, flexor digitorum superficialis; FDP, flexor digitorum profundus; ED, extensor digitorum; EDMB, extensor digitorum minimi brevis; EI, extensor indicis; FPL, flexor pollicis longus; EPL, extensor pollicis longus; EPB, extensor pollicis brevis (EPB); APB, flexor pollicis brevis; OP, opponens pollicis; FDMB, flexor digiti minimi brevis; ADM, abductor digiti minimi; ODM, opponens digiti minimi; Intrinsics, ulnar innervated palmar and dorsal interossei and adductor pollicis.

APPENDIX 1: EMG/NCS Reevaluation (7 months following initial EMG/NCS testing)

- ***Nerve Conduction Studies:*** Summary
- The sensory NCS studies of the bilateral median and superficial radial and right ulnar nerves were now **normal** (Table 7).
- The bilateral median and ulnar DMLs **were prolonged and the CMAP amplitudes were decreased** but were **improved from the initial examination**.
- The bilateral median forearm and ulnar forearm and elbow MNCVs were now **normal**.
- The bilateral median and ulnar F waves remain **prolonged but were improved** from the initial examination.
- **The right tibial nerve motor study demonstrated no recordable response in the right abductor hallucis when stimulated at the ankle and knee.**
- Otherwise, the motor (bilateral deep fibular and left tibial) and sensory (bilateral superficial fibular and sural) NCS studies in BLE remain **unchanged** from the initial examination (Table 7).

TABLE 7: Sensory, Motor and Central Conduction Nerve Conduction Studies in BUE and BLE (6-30-2025)

Nerve Conduction Studies

Anti Sensory Summary Table

Site	NR	Peak (ms)	Norm Peak (ms)	P-T Amp (μV)	Norm P-T Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left SFN Anti Sensory (Ant Lat Mall)											
12 cm	NR		<4.0		>5.0	12 cm	Ant Lat Mall		12.0		>32
Right SFN Anti Sensory (Ant Lat Mall)											
12 cm	NR		<4.0		>5.0	12 cm	Ant Lat Mall		12.0		>32
Left Superficial Radial Anti Sensory (Base 1st Digit)											
D1		2.5	<2.7	34.1	>	D1	Base 1st Digit	2.5	10.0	40	
Right Superficial Radial Anti Sensory (Base 1st Digit)											
D1		2.3	<2.7	22.8	>	D1	Base 1st Digit	2.3	10.0	43	
Left Sural Anti Sensory (Lat Mall)											
Calf	NR		<4.3		>5.0	Calf	Lat Mall		14.0		>32
Right Sural Anti Sensory (Lat Mall)											
Calf		4.9	<4.3	4.4	>5.0	Calf	Lat Mall	4.9	14.0	29	>32

Ortho Sensory Summary Table

Site	NR	Peak (ms)	Norm Peak (ms)	P-T Amp (μV)	Norm P-T Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Ortho Sensory (Wrist)											
Palm		2.2	<2.2	114.7	>15	Palm	Wrist	2.2	8.0	36	
D2		3.6	<3.6	31.8	>15	D2	Wrist	3.6	14.0	39	
Right Median Ortho Sensory (Wrist)											
Palm		2.2	<2.2	88.0	>15	Palm	Wrist	2.2	8.0	36	
D2		3.1	<3.6	20.5	>15	D2	Wrist	3.1	14.0	45	
Left Ulnar Ortho Sensory (Wrist)											

Palm	2.1	<2.2	23.8	>10	Palm	Wrist	2.1	8.0	38
D5	4.0	<3.5	12.8	>10	D5	Wrist	4.0	14.0	35
Right Ulnar Ortho Sensory (Wrist)									
Palm	1.9	<2.2	22.0	>10	Palm	Wrist	1.9	8.0	42
D5	3.3	<3.5	14.9	>10	D5	Wrist	3.3	14.0	42

Motor Summary Table

Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Deep Fibular Motor (Ext Dig Brev)											
Ankle	NR		<6.6		>2	B Fib	Ankle		0.0		>40
B Fib	NR										
Right Deep Fibular Motor (Ext Dig Brev)											
Ankle	NR		<6.6		>2	B Fib	Ankle		0.0		>40
B Fib	NR										
Left DFN TA Motor (Tib Ant)											
Fib Head	NR		<4.2			Poplit	Fib Head		0.0		>40.5
Poplit	NR		<5.7								
Right DFN TA Motor (Tib Ant)											
Fib Head	NR		<4.2			Poplit	Fib Head		0.0		>40.5
Poplit	NR		<5.7	0.0							
Left Median Motor (Abd Poll Brev)											
Wrist		5.5	<4.2	3.2	>5	Elbow	Wrist	6.1	31.0	51	>50
Elbow		11.6		2.4							
Right Median Motor (Abd Poll Brev)											
Wrist		4.9	<4.2	0.5	>5	Elbow	Wrist	5.6	28.0	50	>50
Elbow		10.5		0.1							
Left Tibial Motor (Abd Hall Brev)											
Ankle	NR		<6.8		>2	Knee	Ankle		0.0		>40
Knee	NR										

Right Tibial Motor (Abd Hall Brev)									
Ankle	NR	<6.8	>2	Knee	Ankle	0.0	>40		
Knee	NR								
Left Ulnar Motor (Abd Dig Minimi)									
Wrist	4.4	<3.6	2.1	>5	B Elbow	Wrist	4.3	22.5	52 >50
B Elbow	8.7		0.9		A Elbow	B Elbow	2.3	16.0	70 >50
A Elbow	11.0		0.7						
Right Ulnar Motor (Abd Dig Minimi)									
Wrist	4.2	<3.6	5.4	>5	B Elbow	Wrist	4.6	25.0	54 >50
B Elbow	8.8		5.6		A Elbow	B Elbow	2.1	12.0	57 >50
A Elbow	10.9		5.8						

F Wave Studies

NR	F-Lat (ms)	Lat Norm (ms)	L-R F-Lat (ms)	L-R Lat Norm
Left Median (Mrkrs) (Abd Poll Brev)				
	33.52	<32	0.16	<2.2
Right Median (Mrkrs) (Abd Poll Brev)				
	33.36	<32	0.16	<2.2
Left Ulnar (Mrkrs) (Abd Dig Min)				
	34.70	<32	1.49	<2.5
Right Ulnar (Mrkrs) (Abd Dig Min)				
	33.21	<32	1.49	<2.5

Abnormal values highlighted in red.

Abbreviations: Amp, amplitude; Anti, antidromic; BUE, both upper extremities; cm, centimeter; D1, digit 1 (thumb); D2, digit 2 (index finger); D5, digit 5 (little finger); dist, distance; ms, millisecond; m/sec, meter/second; Mrkrs, markers; NR, no response; norm, normal; O-P, onset to peak; Ortho, orthodromic; P-T, peck to trough; vel, velocity; μ V, microvolt;

APPENDIX 1: EMG/NCS Reevaluation (7 months following initial EMG/NCS testing)

- ***Electromyography:*** Summary
- An abbreviated EMG study of BUE and BLE was performed (Table 8).
- EMG studies of the bilateral biceps brachii, pronator teres, rectus femoris, long head biceps femoris, right extensor digitorum (ED) and 1st DI were now **normal**.
- There was chronic denervation in the bilateral APB, tibialis anterior, fibularis longus, lateral gastrocnemius and left 1st DI compared to the acute denervation in the initial examination.
- No MUPs were observed during voluntary contraction of the bilateral tibialis anterior and right fibularis longus.
- However, three low-amplitude, polyphasic MUPs were now observed in the left fibularis longus and MUPs with decreased recruitment and interference pattern (25%) were present in the bilateral lateral gastrocnemius muscles.
 - Both findings are compatible with reinnervation of these muscles from the initial examination.
- There was decreased MUP recruitment and interference patterns in the bilateral APB and left 1st DI, and this finding is unchanged from the initial examination.

TABLE 8: Needle Electromyography Results in Muscles Tested in BUE and BLE (6-30-2025)

EMG

Side	Muscle	Nerve	Root	Ins Act	Fib s	Ps w	Am p	Dur	Pol y	Recrt	Int Pat	Commen
Left	1stDorInt	Ulnar	C8-T1	Incr	1+	2+	Nml	Nm 	0	Reduced	75%	
Left	Abd Poll Brev	Median	C8-T1	Incr	1+	2+	Nml	Nm 	2+	Reduced	50%	
Left	PronatorTeres	Median	C6-7	Nm 	Nm 	Nm 	Nml	Nm 	0	Nml	Nml	
Left	Biceps	Musculocut	C5-6	Nm 	Nm 	Nm 	Nml	Nm 	0	Nml	Nml	
Righ t	1stDorInt	Ulnar	C8-T1	Nm 	Nm 	Nm 	Nml	Nm 	0	Nml	Nml	
Righ t	Abd Poll Brev	Median	C8-T1	Incr	1+	2+	Nml	Nm 	0	Reduced	50%	
Righ t	PronatorTeres	Median	C6-7	Nm 	Nm 	Nm 	Nml	Nm 	0	Nml	Nml	
Righ t	Biceps	Musculocut	C5-6	Nm 	Nm 	Nm 	Nml	Nm 	0	Nml	Nml	
Righ t	AntTibialis	Deep Fibular	L4-5	Incr	2+	3+				Reduced		NO MUPs
Righ t	Fibularis Longus	Super Fibular	L5-S1	Incr	2+	3+				Reduced		NO MUPs
Righ t	RectFemoris	Femoral	L2-4	Nm 	Nm 	Nm 	Nml	Nm 	0	Nml	Nml	
Left	AntTibialis	Deep Fibular	L4-5	Incr	2+	3+				Reduced		NO MUPs
Left	Fibularis Longus	Super Fibular	L5-S1	Incr	2+	3+	Nml	Nm 	2+	Reduced	Nml	3 MUPs
Left	RectFemoris	Femoral	L2-4	Nm 	Nm 	Nm 	Nml	Nm 	0	Nml	Nml	
Left	Gastroc	Tibial	S1-2	Incr	2+	2+	Nml	Nm 	0	Reduced	25%	
Left	BicepsFemL	Tibial	L5-S2	Nm 	Nm 	Nm 	Nml	Nm 	0	Nml	Nml	
Righ t	Gastroc	Tibial	S1-2	Incr	2+	2+	Nml	Nm 	2+	Reduced	75%	

Righ t	BicepsFemL	Tibial	L5-S2	Nm 	Nm 	Nm 	Nml	Nm 	0	Nml	Nml
Left	ExtDigCom	Radial (Post Int)	C7-8	Incr	2+	2+	Nml	Nm 	0	Reduced	75%
Righ t	ExtDigCom	Radial (Post Int)	C7-8	Nm 	Nm 	Nm 	Nml	Nm 	0	Nml	Nml

Abnormal values highlighted in red.

Fibrillation potential amplitudes were < 100 µV.

Abbreviations: Abd hall, abductor hallucis; Amp, amplitude; Ant, anterior; ADM, abductor digiti minimi; APB, abductor pollicis brevis; BUE, both upper extremities; BLE, both lower extremities; Dur, duration; fibs, EDB, extensor hallucis brevis; EHL, extensor hallucis longus; Ext, extensor; Fem, femoris; fibrillation potentials; Fib, fibular; FCU, flexor carpi ulnaris; Gastroc, gastrocnemius; Hall, hallucis; Ins Act, 1st DI, first dorsal interosseous; insertional activity; Incr, increased; Int Pat, interference pattern; ms, L, longus; Long, longus; Lum pvm, lumbar paravertebral muscle; Med, medius; MPN, medial plantar nerve; millisecond; MUP, motor unit potential; Nml, normal; PVM, paravertebral muscles; Parasp, paraspinal/vertebral muscle; Poly, polyphasic potentials; PPR, posterior primary rami; Psw, positive sharp waves; Rect, rectus; Recrt, recruitment;

APPENDIX 1: EMG/NCS Reevaluation (7 months following initial EMG/NCS testing)

- ***Conclusion (Electrophysiological Testing):***
- There was electrophysiological evidence of a generalized, symmetrical, length dependent, sensorimotor, peripheral polyneuropathy in BLE > BUE with both significant axonal loss and demyelination.
- In comparison with the initial EMG and NCS studies,
 - There was **improvement** noted in the NCS testing in BUE with improved motor and sensory amplitudes and distal latencies.
 - EMG testing in BUE and BLE was also **improved** with better motor unit recruitment and interference patterns in proximal muscles.
 - **However, there was still chronic, severe loss of motor units in the bilateral ankle, forefoot, and toe muscles.**

Journal of Clinical Electrophysiology and Wound Management

Generalized Peripheral Polyneuropathy Secondary to Vitamin B12 Deficiency: A Case Report

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Keywords: polyneuropathy, nerve conduction studies, needle electromyography, Vitamin B12

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RELATIONSHIP BETWEEN ELECTRODIAGNOSTIC AND SONOGRAPHIC CATEGORIZATIONS OF ULNAR NEUROPATHY AT THE ELBOW

Savage N

ABSTRACT

PURPOSE: This presentation will focus on the relationship between electrodiagnostic (EDX) and sonographic assessments of ulnar neuropathy at or near the elbow (UNE), highlighting their diagnostic agreement, areas of divergence, and the influence of demographic and clinical factors within a large patient cohort.

MATERIALS AND METHODS: 299 elbows (54% left) from 262 patients (52.6±18.0 years; 55% male) undergoing EDX testing and ultrasound imaging (USI) examinations between August 2018 and October 2025 were included for analysis. EDX classification of UNE as defined by Padua (2001) and Zeidman (2020) was evaluated and compared to ulnar nerve cross-sectional area (CSA) at or near the medial epicondyle.

RESULTS: EDX evidence of UNE was present in 176 elbows (59%) with an average ulnar motor conduction across the elbow of 41.1 m/s compared to 60.8 m/s in patients without UNE. USI evidence of UNE was present in 151 elbows (51%) with an average CSA of 14.2 mm² compared to 6.4 mm² in patients without UNE. Chi Square analysis revealed significant differences between the EDX and USI impressions of UNE ($\chi^2=76.1$, $P<.001$) with 75% agreement. Ulnar nerve subluxation was observed in 24% of elbows and was significantly associated with the EDX ($\chi^2=6.5$, $P=.011$) but not the USI ($\chi^2=2.5$, $P=.111$) impression of UNE. Additional analysis was performed evaluating the relationship between age, sex, limb tested, and past medical history with EDX and USI findings.

DISCUSSION: EDX and USI assessments demonstrated substantial but incomplete agreement, reflecting their measurement of distinct functional and structural aspects of UNE. The stronger association between EDX findings and ulnar nerve subluxation highlights the dynamic nature of the condition, while sonographic enlargement reflects chronic structural change. These results support a complementary, multimodal approach to improve diagnostic accuracy and clinical management of UNE.

CONCLUSIONS: This presentation discussed integrating EDX and sonographic perspectives to enhance evaluation and understanding of UNE highlighting the value of a complementary, multimodal approach to improve diagnostic precision and guide clinical decision-making.

Nathan J. Savage, PhD, DPT, OCS, ECS, RMSK

RELATIONSHIP BETWEEN ELECTRODIAGNOSTIC AND SONOGRAPHIC CATEGORIZATIONS OF ULNAR NEUROPATHY AT THE ELBOW

Dr. Nathan J. Savage, PhD, DPT, ECS, OCS, RMSK



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Learning Objectives

- Define and discuss EDX-based classifications for UNE
- Evaluate and discuss relationship b/w EDX and sonographic categorizations of UNE
- Discuss value of combining EDX and sonographic evaluations of common peripheral nerve injuries in upper extremity



Manuscript: *Ulnar neuropathy at the elbow:
evaluation of EDX-based severity
classifications in 299 elbows*

Journal of the Peripheral Nervous System (under review)



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Introduction

- UNE second most common entrapment neuropathy
 - Prevalence up to 6% in general population,
 - Incidence as high as 30/100K person-years
 - Direct surgical treatment costs over \$500 million/year in U.S.
- Classifications exist that categorize relative severity of UNE based on specific EDX-based criteria
 - Padua et al (2001); Greathouse et al (2017); Zeidman and Pandey (2020)



Introduction

- Padua only considers nerve conduction data
- Greathouse and Zeidman consider nerve conduction data and needle EMG findings
- Greathouse requires evaluation of ulnar sensory conduction across elbow in each proposed severity categories
 - Because not routinely performed in our EDX laboratory (and presumably others) excluded from further analysis



Padua (2001)

Mild

- Slow NCV across elbow (<39 m/s)
- Normal DSA* (>8.0 μ V)
*Doubled 4.0 μ V value based on orthodromic study to approximate an antidromic study

Moderate

- Slow NCV across elbow
- Low DSA (≤ 8.0 μ V)

Severe

- Slow NCV across elbow
- Absent distal sensory response

Extreme

- Absent distal motor response
- Absent distal sensory response

Zeidman (2020)

Mild

- Slow NCV across elbow (<50 m/s) $\pm$ low DMA (<5.6 mV or $\geq 10\%$ drop)
- Normal DSA (≥ 17 μ V; ≥ 10 μ V in patients >60 years)
- Normal EMG

Moderate

- Slow NCV across elbow $\pm$ low DMA
- Low/absent DSA (<17 μ V; <10 μ V in patients >60 years) OR abnormal EMG (resting or volitional)

Severe

- Slow NCV across elbow $\pm$ low or absent DMA
- Absent distal sensory responses
- Abnormal EMG

Introduction

- Previous studies have investigated relationship b/w clinical symptoms and UNE severity
- Authors found no studies investigating ability of existing classifications to discriminate b/w consecutive categories of UNE severity based on specific EDX-based criteria
- Savage and McKell recently demonstrated that consolidated categorizations of median neuropathy at wrist (i.e., “Mild”, “Moderate”, or “Severe”) was superior to multiple severity categories



Introduction

- **Purpose:** Evaluate EDX findings across consecutive categories of UNE severity within existing EDX-based classifications
- **Hypotheses:**
 - EDX findings would poorly discriminate b/w consecutive categories of UNE severity
 - Binary categorizations would improve discrimination
 - Distribution of UNE within *binary* categorizations would be significantly different



Methods

- Retrospective study of cross-sectional data from patients undergoing EDX testing and USI for suspected UNE
- **Primary outcome:** categorization of UNE severity
- IRB approval Winston-Salem State University (IRB-FY2023-37)
- Final EDX impression determined for all elbows (“Negative” or “UNE”) based on normative data by Buschbacher



Methods

- Elbows categorized according to UNE severity based on criteria defined in Padua (5 levels) and Zeidman (4 levels) classifications
- Classifications consolidated to *binary* categorizations for statistical comparison (i.e., “Negative” and “UNE”)
- MANOVA used to compare EDX findings across UNE severity categories within complete and *binary* categorizations
- Chi Square analysis used to compare *binary* categorizations



Results

- Data collected August 2018 through October 2025
- 262 patients (52.6 ± 18.0 years; 67.8 ± 3.9 inches; 55% male)
- 299 elbows (53% left)
- **Final EDX Impression:**
 - 140 elbows “Negative” (47%)
 - 159 elbows “UNE” (53%)



Results

- **Strict application of criteria:**

- Padua categorized 292 of 299 elbows (98%)
 - Zeidman categorized 248 of 299 elbows (83%)
- For purposes of analysis, and consistent w/ clinical practice, elbows not meeting strict criteria placed in category best fitting EDX findings



Padua

UNE Severity	N	Category Comparison	NCV (m/s)	Mean Δ	P value	DSA (μ V)	Mean Δ	P value
Negative	237		53.89 $\pm$ 10.24			24.89 $\pm$ 18.47		
Mild	21	Mild v Neg	34.33 $\pm$ 3.61	-19.56	<.001	18.08 $\pm$ 9.74	-6.81	.761
Moderate	14	Mod v Mild	33.57 $\pm$ 4.78	-0.76	1.00	4.99 $\pm$ 1.72	-13.09	.246
Severe	22	Sev v Mod	30.73 $\pm$ 6.64	-2.84	1.00	0.00 $\pm$ 0.00	-4.99	1.00
Extreme	5	Ext v Sev	0.00 $\pm$ 0.00	-30.73	<.001	5.28 $\pm$ 11.81	5.28	1.00
UNE Severity	N	Category Comparison	DMA (mV)	Mean Δ	P Value	Abnormal EMG*	Count	%
Negative	237		9.48 $\pm$ 2.74			Negative	91	31.7
Mild	21	Mild v Neg	9.75 $\pm$ 2.26	0.27	1.00	Mild	16	5.6
Moderate	14	Mod v Mild	7.57 $\pm$ 2.29	-2.18	.223	Moderate	10	3.5
Severe	22	Sev v Mod	4.07 $\pm$ 3.18	-3.50	.002	Severe	17	5.9
Extreme	5	Ext v Sev	4.32 $\pm$ 3.86	0.25	1.00	Extreme	5	1.7

p<.0125 for post hoc comparisons

*Not a component of the Padua classification system

Zeidman

UNE Severity	N	Category Comparison	NCV (m/s)	Mean Δ	P value	DSA (μ V)	Mean Δ	P value
Negative	112		61.64 $\pm$ 7.95			33.38 $\pm$ 19.35		
Mild	31	Mild v Neg	51.32 $\pm$ 9.22	-10.32	<.001	30.78 $\pm$ 17.00	-2.59	1.00
Moderate	150	Mod v Mild	40.59 $\pm$ 7.69	-10.73	<.001	11.05 $\pm$ 9.64	-19.73	<.001
Severe	6	Sev v Mod	9.33 $\pm$ 22.86	-31.26	<.001	4.40 $\pm$ 10.78	-6.65	1.00
UNE Severity	N	Category Comparison	DMA (mV)	Mean Δ	P value	Abnormal EMG	Count	%
Negative	112		10.15 $\pm$ 2.50			Negative	0	0.0
Mild	31	Mild v Neg	10.54 $\pm$ 1.75	0.39	1.00	Mild	15	5.2
Moderate	150	Mod v Mild	7.84 $\pm$ 3.25	-2.70	<.001	Moderate	118	41.1
Severe	6	Sev v Mod	4.87 $\pm$ 3.70	-2.97	.081	Severe	6	2.1

p<.0167 for post hoc comparisons

Final EDX Impression

UNE Present?	N	Category Comparison	NCV (m/s)	Mean Δ	P value	DSA (μ V)	Mean Δ	P value
No	140	UNE v Neg	59.36 $\pm$ 8.92	-19.55	<.001	30.78 $\pm$ 18.75	-17.78	<.001
Yes	159		39.81 $\pm$ 11.14			12.99 $\pm$ 13.52		
UNE Present?	N	Category Comparison	DMA (mV)	Mean Δ	P value	Abnormal EMG	Count	%
No	140	UNE v Neg	10.05 $\pm$ 2.54	-2.12	<.001	Negative	11	3.8
Yes	159		7.93 $\pm$ 3.30			UNE	128	44.6

Binary Padua

UNE Present?	N	Category Comparison	NCV (m/s)	Mean Δ	P value	DSA (μ V)	Mean Δ	P value
No	237	UNE v Neg	53.89 $\pm$ 10.24	-23.78	<.001	24.89 $\pm$ 18.47	-17.22	<.001
Yes	62		30.11 $\pm$ 10.38			7.68 $\pm$ 10.08		
UNE Present?	N	Category Comparison	DMA (mV)	Mean Δ	P value	Abnormal EMG*	Count	%
No	237	UNE v Neg	9.48 $\pm$ 2.74	-2.68	<.001	Negative	91	31.7
Yes	62		6.80 $\pm$ 3.68			UNE	48	16.7

*Not a component of the Padua classification system

Binary Zeidman

UNE Present?	N	Category Comparison	NCV (m/s)	Mean Δ	P value	DSA (μ V)	Mean Δ	P value
No	112	UNE v Neg	61.64 $\pm$ 7.95	-20.27	<.001	33.38 $\pm$ 19.35	-19.27	<.001
Yes	187		41.37 $\pm$ 11.19			14.10 $\pm$ 13.46		
UNE Present?	N	Category Comparison	DMA (mV)	Mean Δ	P value	Abnormal EMG	Count	%
No	112	UNE v Neg	10.15 $\pm$ 2.50	-1.96	<.001	Negative	0	0.0
Yes	187		8.19 $\pm$ 3.27			UNE	139	48.4

Results

- Significant differences found in distribution of “Negative” and “UNE” across *binary* categorizations
 - Padua vs Zeidman ($\chi^2 = 46.8$, $df = 1$, $P < .001$)
 - All discrepancy found in “UNE” categorization
 - Padua vs Final EDX Impression ($\chi^2 = 68.9$, $df = 1$, $P < .001$)
 - All discrepancy found in “UNE” categorization
 - Zeidman vs Final EDX Impression ($\chi^2 = 196.6$, $df = 1$, $P < .001$)
 - Nearly all discrepancy found in “Negative” categorization



Results

- 287 limbs (96%) underwent needle EMG of ulnar innervated muscles
 - 148 negative (52%)
 - 139 positive (38%)
 - i.e., presence of abnormal resting activity and/or neuropathic MUPs



Results

- Significant moderate correlations found b/w:
 - Age and ulnar DSL ($r = .52$, $P < .001$) and ulnar DSA ($r = .61$, $P < .001$)
 - Ulnar DSA and motor NCV across elbow ($r = .51$, $P < .001$)
 - Ulnar DSL and DML ($r = .59$, $P < .001$)
 - Ulnar DSA and DMA ($r = .54$, $P < .001$)
 - Ulnar DML and DMA ($r = .54$, $P < .001$)
- Sex (but not age category) effect modifier



Conclusions

- Findings demonstrate that *binary* categorizations offer superior discrimination capability compared to classifications that use multiple categories of UNE severity
- Given prevalence, economic burden, and clinical consequences of UNE, simplified and pragmatic categorizations most likely to enhance diagnostic consistency and clinical decision-making



Manuscript: *Sonographic measurement of ulnar nerve CSA to determine severity of ulnar neuropathy at the elbow: analysis of EDX-based classifications*

Journal of Ultrasound in Medicine (under review)



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Introduction

- EDX testing long considered gold standard for diagnosing neuropathic changes in PNS
- USI increasingly used to assist in diagnosis of peripheral nerve disorders including UNE, shown to be convenient, safe, and cost-effective for a variety of NMSK conditions
 - Experts agreed all investigations of UNE should include nerve conduction studies and USI
 - Should assess CSA and nerve mobility (*Clinical Neurophysiology, 2021*)



Introduction

- Criteria for determining presence of UNE based on USI have been recommended
 - Jacobson *Fundamentals of Musculoskeletal Ultrasound (3ed)*:
 - Ulnar nerve CSA $>9 \text{ mm}^2$
 - Meta-Analysis (*Arch Phys Med Rehabil, 2018*):
 - Rarely exceeds 10 mm^2 in healthy participants



Introduction

- **Purpose:** Evaluate diagnostic accuracy of sonographic measurements of ulnar nerve CSA to determine UNE severity across EDX-based classifications
- **Hypotheses:**
 - CSA would poorly discriminate b/w consecutive categories of UNE severity in complete classifications
 - *Binary* categorizations would provide best overall diagnostic accuracy



Methods

- **Primary outcome:** ulnar nerve CSA
- Final USI impression determined for all elbows using CSA cutoff values ≤ 9 mm² for “Negative” and >9 mm² for “UNE”
- Evaluated complete and *binary* EDX-based categorizations



Methods

- Chi Square analysis of final USI impression vs *binary* EDX-based categorizations of UNE severity
- ROC analysis to establish CSA cutoff values and associated diagnostic LRs
 - **Youden's index** [i.e., $(Sn + Sp) - 1$] and **optimized LRs** (i.e., first value to achieve conclusive shift in diagnostic probability)
 - **Diagnostic probability depends on disease prevalence**
 - For illustrative purposes, pre-test probabilities for UNE:
 - 25% (**unlikely**) vs 50% (**toss-up**) vs 75% (**likely**)



Results

- **Final USI impression:**

- 150 elbows “Negative” (50%)
 - 149 elbows “UNE” (50%)
- Prevalence of ulnar nerve subluxation/dislocation 24% (72 of 299 elbows)
- Prevalence of diabetes 15% (45 of 299 elbows)



Results

- Chi Square analysis
 - Moderate to good agreement b/w all *binary* EDX-based categorizations and final USI impression of UNE
 - Ranging from 60% (Padua) to 80% (Zeidman) in “Negative” category
 - Ranging from 68% (Zeidman) to 89% (Padua) in “UNE” category
 - Final EDX Impression $\chi^2 = 76.6$, $P < .001$
 - Padua, $\chi^2 = 47.3$, $P < .001$
 - Zeidman, $\chi^2 = 163.1$, $P < .001$



EDX-Based Classification	N	Ulnar Nerve CSA Mean $\pm$ SD
Final EDX Impression		
Negative	140	7.69 $\pm$ 3.42
UNE	159	12.66 $\pm$ 5.47
Padua et al ¹⁴		
Negative	237	9.09 $\pm$ 4.00
Mild	21	14.33 $\pm$ 7.77
Moderate	14	13.64 $\pm$ 3.46
Severe	22	16.50 $\pm$ 5.85
Extreme	5	16.20 $\pm$ 10.76
Zeidman and Pandey ¹¹		
Negative	112	7.39 $\pm$ 3.48
Mild	31	9.90 $\pm$ 3.54
Moderate	150	12.44 $\pm$ 5.30
Severe	6	14.83 $\pm$ 10.19

Results

- No significant differences in ulnar nerve CSA found:
 - Ulnar subluxation/dislocation
 - 11.0 vs 10.1 mm², P = .198
 - Patients w/ diabetes
 - 11.2 vs 10.2 mm², P = .255
- Sex and age category (i.e., <50 vs ≥50 years) effect modifiers



Results

- Ulnar nerve CSA cutoff values across all *binary* EDX-based categorizations revealed AUC values w/ excellent discrimination
 - .807 to .810
- Youden's index established CSA cutoff values resulting in small shifts in diagnostic probability to discriminate b/w “UNE” and “Negative” categorizations
 - +LRs and -LRs 2.77 to 4.10 and 0.39 to 0.29, respectively



(A)							Optimized Cutoff Values	
Padua Classification	N	Category Comparison	AUC	CSA (mm ²)	+LR	-LR	CSA (mm ²)	+LR & -LR
Negative	237							
Mild	21	Mild v Negative	.760*	9.5	2.16	0.24	19.5 & 5.5	9.52 & 0.00
Moderate	14	Mod v Mild	.573	12.5	1.83	0.37	NA	NA
Severe	22	Severe v Mod	.649	16.5	2.86	0.69	NA	NA
Extreme	5	Ext v Severe	.477	18.5	2.20	0.73	NA	NA
Zeidman Classification	N	Category Comparison	AUC	CSA (mm ²)	+LR	-LR	CSA (mm ²)	+LR & -LR
Negative	112							
Mild	31	Mild v Negative	.739*	8.5	2.58	0.47	NA & 5.5	NA & 0.08
Moderate	150	Mod v Mild	.660*	12.5	3.26	0.67	NA	NA
Severe	6	Severe v Mod	.519	18.5	3.33	0.74	25.0 & NA	8.35 & NA

*p<.05

Results

- Optimized LR_s with CSA cutoff values resulting in large shifts in diagnostic probability were found in all *binary* EDX-based categorizations
 - +LR_s and -LR_s 7.74 to 9.00 and 0.15 to 0.11, respectively



Results

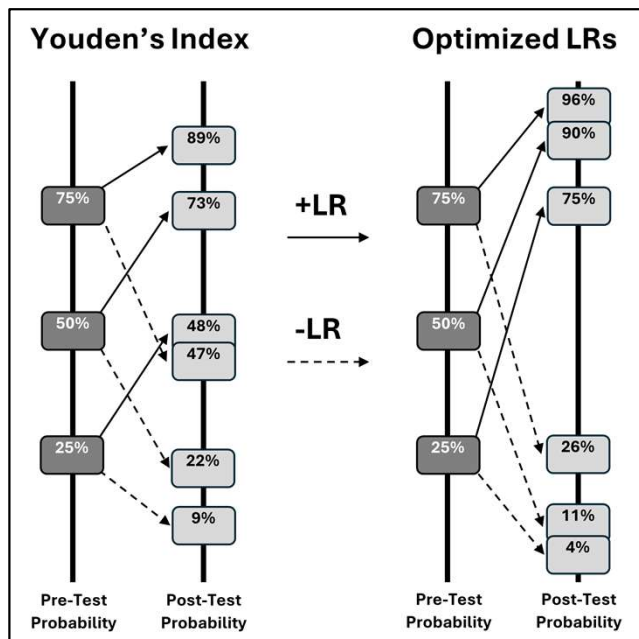
- Overall, CSA cutoff values based on optimized LRs to rule out UNE were consistent across all *binary* EDX-based categorizations
 - $<6 \text{ mm}^2$
- CSA cutoff values based on optimized LRs to rule in UNE were most consistent with EDX-based categorizations that included needle EMG findings (i.e., final EDX impression and Zeidman)
 - $>15 \text{ mm}^2$ and $>16 \text{ mm}^2$, respectively



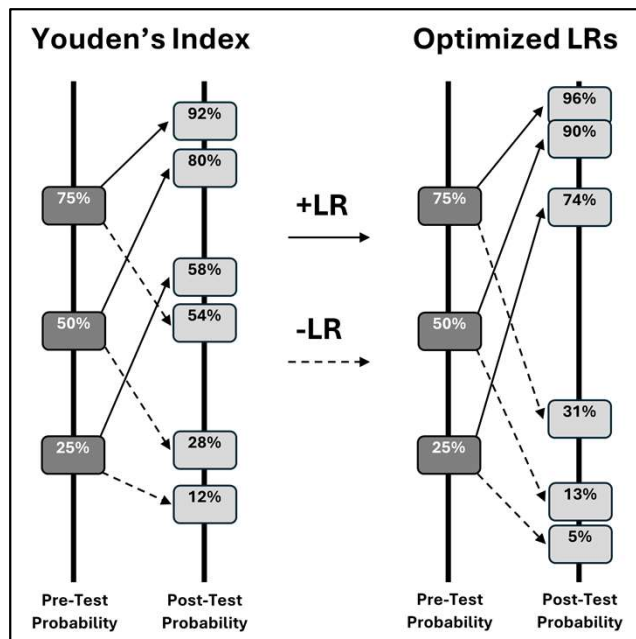
(B)							Optimized Cutoff Values	
Final EDX Impression	N	Category Comparison	AUC	CSA (mm ²)	+LR	-LR	CSA (mm ²)	+LR & -LR
Negative	140	UNE v Negative	.810*	8.5	2.77	0.29	16.5 & 5.5	9.00 & 0.12
UNE	159							
<i>Binary Padua</i>	N	Category Comparison	AUC	CSA (mm ²)	+LR	-LR	ΔCSA (mm ²)	+LR & -LR
Negative	237	UNE v Negative	.809*	12.5	4.10	0.39	20.5 & 5.5	8.69 & 0.15
UNE	62							
<i>Binary Zeidman</i>	N	Category Comparison	AUC	CSA (mm ²)	+LR	-LR	ΔCSA (mm ²)	+LR & -LR
Negative	112	UNE v Negative	.807*	8.5	2.95	0.35	15.5 & 5.5	7.74 & 0.11
UNE	187							

*p<.05

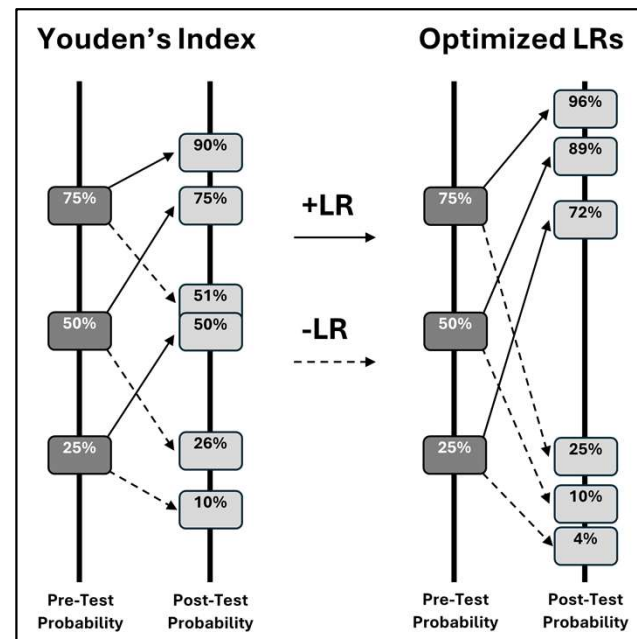
Final EDX Impression



Binary Padua



Binary Zeidman



Conclusions

- Using ulnar nerve CSA to classify UNE severity is not recommended based on lack of consistent and meaningful shifts in diagnostic probability
- *Binary* categorizations to rule out or rule in UNE based on proposed CSA cutoff values provides conclusive shifts in diagnostic probability for EDX-based categorizations examined



		Diagnosis of UNE	Shift in Diagnostic Probability
Optimized LRs	Count (%)		
CSA			
<6 mm ²	51 (17%)	Rule out	Conclusive
>15 mm ²	42 (14%)	Rule in	Conclusive
Unclassified	206 (69%)	Equivocal	NA
Youden's Index	Count (%)		
CSA			
≤8 mm ²	133 (44%)	Rule out	Small
≥9 mm ²	166 (56%)	Rule in	Small

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SEGMENTAL ZOSTER PARESIS IN THE UPPER EXTREMITY

Painter E

ABSTRACT

BACKGROUND: Herpes zoster refers to a viral infection characterized by skin lesions, paresthesia and pain along a dermatome. It is caused by reactivation of the varicella zoster virus in the dorsal root ganglia and is associated with diminished immune system capacity. Segmental zoster paresis is a rare complication of herpes zoster and causes focal, asymmetric weakness in the same in myotome that corresponds to the dermatomal rash distribution. The virus can affect the spinal cord, nerve roots, plexus or peripheral nerves, creating a complex neurological picture.

DISCUSSION: A 79-year-old man was referred for electrophysiologic testing with a primary complaint of motor weakness and pain throughout the right upper extremity (RUE). He reported a history of herpes zoster nine months prior that was characterized by skin lesions affecting the RUE from the right shoulder to the hand and affecting all aspects of the hand. Since that time, he has had severe pain and motor weakness throughout the distal greater than proximal RUE. Electrophysiologic testing demonstrated diffuse denervation potentials and mild to severely reduced recruitment and interference patterns in the RUE muscles representative of the right C6-T1 myotome. Additionally, the lack of recordable sensory nerve responses in the RUE suggested additional post-ganglionic pathology, consistent with segmental zoster paresis.

CONCLUSION: Electrophysiological testing is an important tool in evaluating the patient with neurological complaints following herpes zoster infection. It can help characterize the neurological structures involved, evaluate for more common spinal nerve root and peripheral nerve lesions and provide insight on prognosis.

Elizabeth E. Painter, PT, DPT, DSc, ECS

Segmental Zoster Paresis in the Upper Extremity

Elizabeth E. Painter, PT, DPT, DScPT



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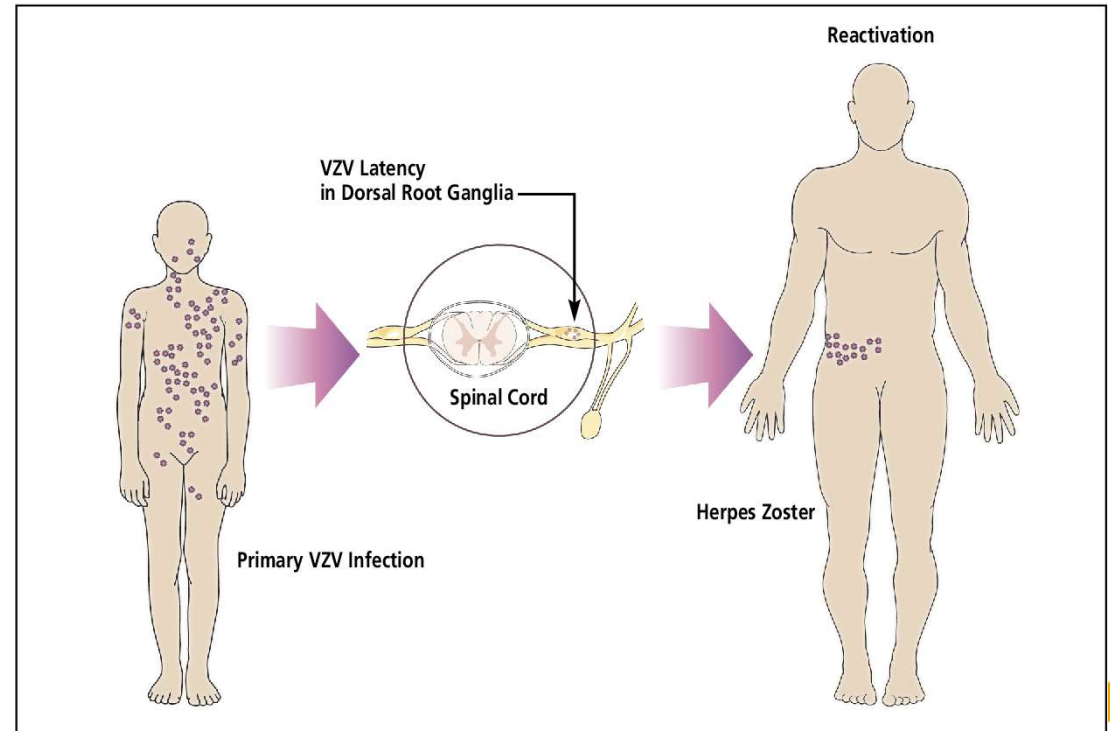
Objectives

- Identify findings from the subjective exam that suggest neurological lesion related to herpes zoster infection
- Identify findings from the clinical exam that suggest neurological lesion related to herpes zoster infection
- Plan an electrophysiologic exam (EMG/NCS) to evaluate a patient with new onset motor weakness and sensory disturbance related to herpes zoster infection
- Using clinical and electrophysiological data, distinguish new onset motor and sensory loss from herpes zoster from other sources of neurological pathology



Herpes Zoster

- Acute, infectious skin disease caused by re-activation of varicella zoster virus (VZV)
- Shingles
- VZV infection often occurs in childhood (chicken pox)
- Remains in the dorsal root ganglion after infection or vaccination
- VZV reactivation occurs with ageing, immunosuppression



<https://www.semanticscholar.org/paper/Herpes-Zoster-Overview%3A-Natural-History-and-Weaver/b9873be6b14d0175e5e7425824048c60737e4370/figure/0>

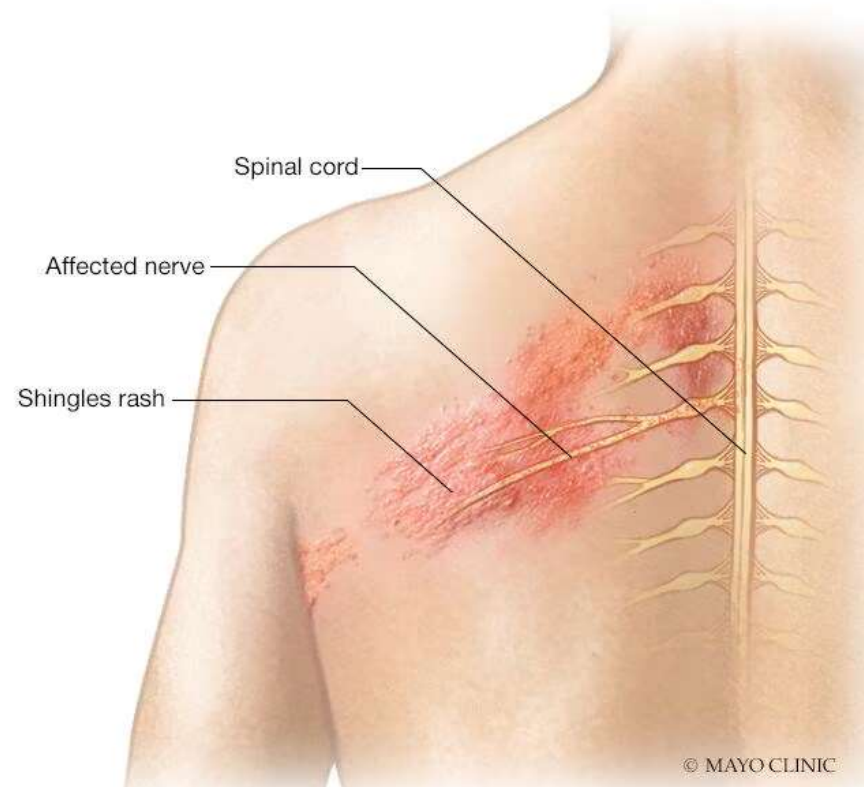
Meng 2021



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Herpes Zoster

- Reactivation of VZV mostly affects sensory nerves and produces pain and skin lesions unilaterally along the affected dermatome
- Most common nerve segments affected are thoracic (>50%), lumbar and trigeminal nerve



Meng 2021, Chen 2020

<https://www.mayoclinic.org/diseases-conditions/shingles/symptoms-causes/syc-20353054>

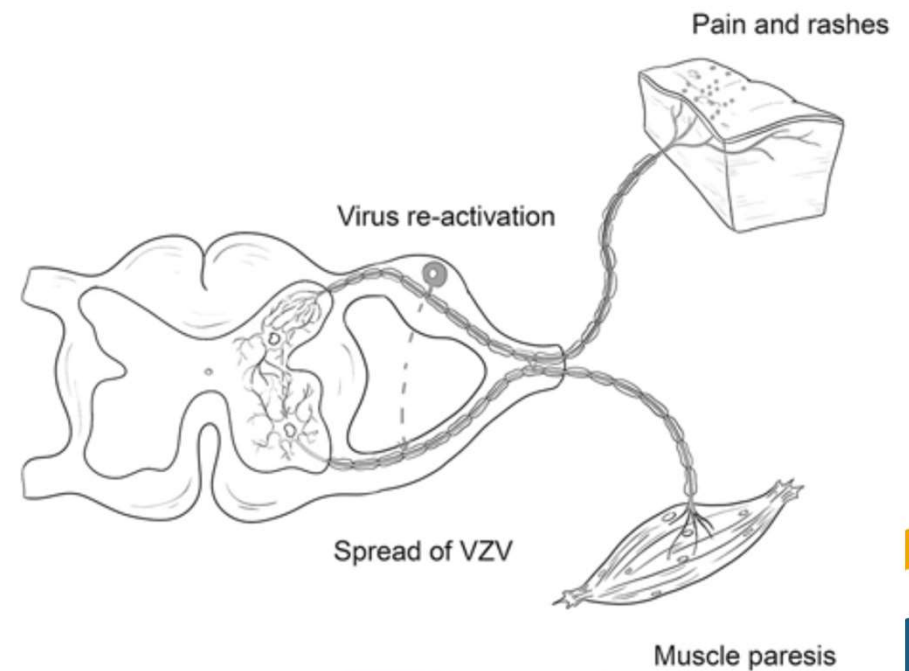


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Segmental Zoster Paresis (SZP)

- Motor involvement is rare (0.5-5%)
- Occurs 2-3 weeks after skin lesions but variable
- Affects myotome corresponding to rash distribution but variable
- Possible spread from dorsal root ganglia to anterior horn cells or root

Meng 2021 (image), Chen 2020



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Segmental Zoster Paresis: Diagnosis

- Diagnosis can be challenging
 - Weakness onset before lesions
 - Myotomal/dermatomal mismatch
 - Pre-existing spinal pathology
- Imaging may show enhancement of dorsal horn, nerve roots or peripheral nerves
- EMG/NCS: excludes other conditions, localize lesion(s), determine prognosis

Kang 2013, Chen 2021, Aykac 2024



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Segmental Zoster Paresis: EMG/NCS

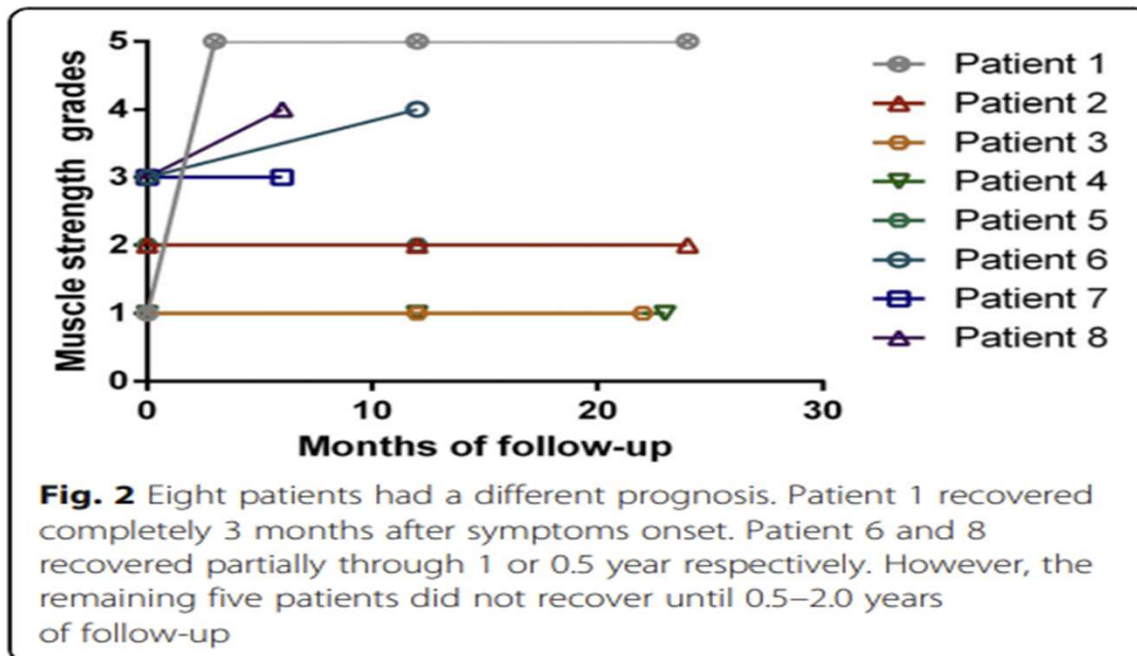
- Pathophysiology: axonal degeneration with secondary segmental demyelination
- EMG/NCS of 8 patients with SZP from 1393 with VZV (0.57%)
 - EMG/NCS showed primarily axonopathy with severely decreased or absent CMAP and/or SNAP, PSWs/Fibs
 - 2/8: radiculopathy
 - 2/8: plexopathy
 - 3/8: radiculoplexopathy
 - 1/8: combined radiculopathy and mononeuropathy

Liu 2018, Aykac 2024



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Prognosis of Segmental Zoster Paresis



Liu 2018



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Segmental Zoster Paresis: Treatment

- No study on effective treatment for improving functional recovery
 - Mostly case studies
- 50-70% of cases recover fully
- Recovery occurs over 1-2 years
- Early initiation of antivirals (within 72 hrs) and corticosteroids

Aykac 2024



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Case: Subjective Exam

- RHD 79-year-old male
- Weakness in R hand > arm that limits his ability to grip or extend his fingers
- Pain throughout the RUE and hand
- Limited R shoulder ROM due to pain and weakness
- Denies N/T in RUE
- Near constant swelling the R hand
- Episode of shingles 9 months prior with lesions from the R shoulder distally to the hand, affecting all aspects of the hand
- Weakness started shortly after development of skin lesions
- No improvement in strength over time
- Denies neck pain or LUE or BLE symptoms



Case: Subjective Exam

- Reports history of shingles vaccine but unsure when or type
- Treatment: anti-viral medication at time of lesions
 - Cervical ESI and PT without improvement
- EMG/NCS 9 months ago with “nerve damage” but no specifics or report
- Has seen neurology and orthopedics
 - Diagnosed with post-herpetic neuralgia
- Imaging of both cervical spine and brachial plexus
 - Multi-level DDD, worst at C5-6 with moderate to severe canal stenosis
- Referred to neurosurgery for further evaluation



Case: Subjective Exam

- Prior medical history:
 - Diabetes (x 3 yrs, controlled with medication)
 - CAD s/p stent x 7
 - HTN
 - High cholesterol
 - Non-Hodgkins lymphoma (in remission x 10-11 years)
 - Peripheral vascular disease RLE
- Takes baby aspirin and Brilinta daily for anticoagulation
- Prior surgical history:
 - Splenectomy
 - Cardiac stent x 7
 - Cardiac valve replacement 2023



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Case: Clinical Exam

- Cautious, slow movements of RUE
- Wears a wrist brace
- Trophic changes and mild swelling in the R hand
- Severe atrophy over the R FDI, ADM, APB, medial forearm
- R shoulder ROM limited to 90 deg with discomfort and compensatory movement



Case: Clinical Exam

- 1/5 R FDI, APB, ADM
- 2+/5 R finger extension (EDC, EI)
- 3+/5 R wrist flex/ext with some discomfort
- 4/5 R shoulder girdle muscles, elbow flex/ext
- MSR: 1+ bilateral biceps reflex, triceps reflex and brachioradialis reflex
- Pathological Reflex: Hoffman reflex absent bilaterally
- Decreased sensation to pinprick in the R palmar 5th digit
- Hypersensitivity to pin prick throughout the remainder of the RUE
- Special Tests: Neg Tinels at B wrist, B elbow



Sensory Nerve Conduction Studies

Stim Site	NR	Peak (ms)	Norm Peak (ms)	O-P Amp (µV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median 2nd digit Anti Sensory (2nd Digit)											
Palm		1.9	<2.2	34.4	>15	Palm	2nd Digit	1.9	7.0	37	
Wrist		3.5	<3.6	34.2		Wrist	Palm	1.6	7.0	44	
Right Median 2nd digit Anti Sensory (2nd Digit)											
Palm	NR		<2.2		>15	Palm	2nd Digit		7.0		
Wrist	NR		<3.6		>15	Wrist	Palm		7.0		
Left Radial Anti Sensory (Base 1st Digit)											
Wrist		2.2	<2.8	28.8	>10	Wrist	Base 1st Digit	2.2	10.0	45	
Right Radial Anti Sensory (Base 1st Digit)											
Wrist	NR		<2.8		>10	Wrist	Base 1st Digit		10.0		
Left Ulnar 5th digit Anti Sensory (5th Digit)											
Wrist		3.4	<3.5	13.1	>10.0	Wrist	5th Digit	3.4	14.0	41	
Right Ulnar 5th digit Anti Sensory (5th Digit)											
Wrist	NR		<3.5		>10.0	Wrist	5th Digit		14.0		



Motor Nerve Conduction Studies

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Motor (Abd Poll Brev)											
Wrist		4.1	<4.2	4.6	>5	Elbow	Wrist	4.9	24.5	50	>50
Elbow		9.0		4.5							
Right Median Motor (Abd Poll Brev)											
Wrist	NR		<4.2		>5	Elbow	Wrist		0.0		>50
Elbow	NR										
Right Radial Motor (Ext Ind Prop)											
8cm		5.5	<3.4	0.1	>5.0	Up Arm	8cm		0.0		>50
Up Arm	NR										
Left Ulnar Motor (Abd Dig Minimi)											
Wrist		3.1	<3.6	7.5	>3	B Elbow	Wrist	3.9	20.0	51	>50
B Elbow		7.0		6.9		A Elbow	B Elbow	2.1	12.0	57	>50
A Elbow		9.1		6.1							
Right Ulnar Motor (Abd Dig Minimi)											
Wrist	NR		<3.6		>3	B Elbow	Wrist		0.0		>50
B Elbow	NR					A Elbow	B Elbow		0.0		>50
A Elbow	NR										



EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	>12ms	2+	Reduced	75%	
Right	Biceps	Musculocut	C5-6	Incr	1+	2+	Nml	>12ms	2+	Nml	Nml	
Right	FlexCarRad	Median	C6-7	Incr	2+	2+	Nml	>12ms	2+	Reduced	75%	
Right	ExtDigCom	Radial (Post Int)	C7-8	Incr	1+	2+	Nml	>12ms	2+	Reduced	50%	
Right	1stDorInt	Ulnar	C8-T1	Incr	2+	2+				Reduced	None	no MUPs
Right	Abd Poll Brev	Median	C8-T1	Incr	2+	2+				Reduced	None	no MUPs
Left	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Biceps	Musculocut	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	FlexCarRad	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	ExtDigCom	Radial (Post Int)	C7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Abd Poll Brev	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	FlexDigProf	Ulnar	C8,T1	Incr	2+	2+				Reduced	None	no MUP
Right	ExtIndicis	Radial (Post Int)	C7-8	Incr	2+	2+	Nml	Nml	0	Reduced	Discr	2-3 MUP
Right	ABD Dig Min	Ulnar	C8-T1	Incr	2+	2+				Reduced	None	no MUP



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Interpretation

- Diffuse denervation potentials and mild to severely reduced recruitment and interference patterns were noted in the RUE in muscles representative of the right C6-T1 myotome. The right C8-T1 myotome is most severely affected with no recordable motor units in any of the right C8-T1 muscles tested in the hand and forearm. The C5 nerve root appears relatively spared. Nerve conduction studies of the RUE demonstrated no recordable sensory nerve responses or median or ulnar motor nerve responses. The right radial motor response had a very small amplitude response with prolonged distal motor latency and no recordable proximal response. Collectively, these findings suggest spinal nerve root pathology affecting the right C6-T1 (C8-T1>C6-7) nerve roots with abnormalities largely following a myotomal pattern. However, the lack of recordable sensory nerve responses suggests additional post-ganglionic pathology. These findings may be consistent with the patient's history of shingles/herpes varicella zoster virus affecting the RUE.



Management

- No surgical intervention recommended for the cervical spine
- Cause of weakness determined to be related to herpes zoster infection
- Prognosis for neurological recovery was poor
 - 9 month duration of weakness
 - Absent motor and sensory responses on NCS
- Referred to pain management



Discussion

- Segmental zoster paresis or post-herpetic neuralgia?
 - Weakness in affected dermatomes following HZ infection
 - Severe pain
 - Pre and post-ganglionic features on EMG/NCS
 - Axonal pathology



Risk Factors for SZP

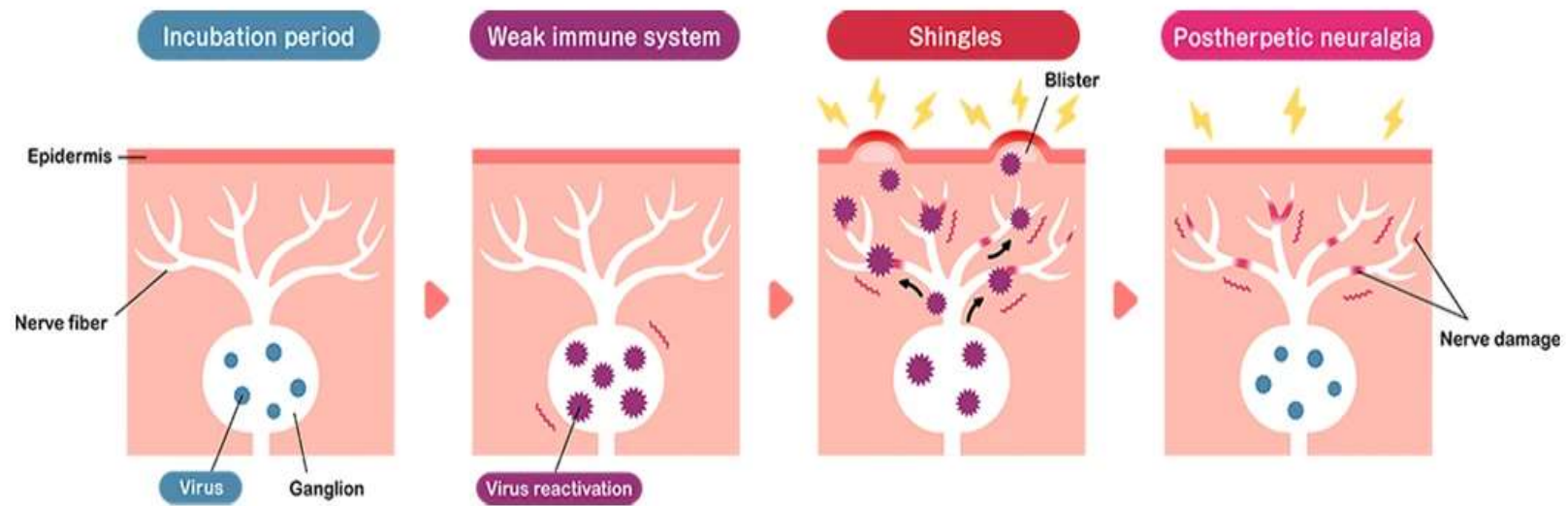
- Li & Feng: nomogram-based prediction of risk of limb weakness based on 314 patients
 - Higher age, high initial VAS, involvement of C6 or C7 nerve roots are most predictive
- Wang: Retrospective case control study (21 with SZP, 66 controls)
 - Presence and grade of cervical neuroforaminal stenosis (NFS) is an independent risk factor for SZP
 - NFS and baseline strength are independent prognostic factors

Li & Feng 2023, Wang 2026



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Post-Herpetic Neuralgia



<https://www.advocatehealth.com/health-services/brain-spine-institute/brain-care-center/conditions-treatments/neuromuscular-disorders/postherpetic-neuralgia>



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Post-Herpetic Neuralgia

- Inflammation leads to demyelination, Wallerian degeneration, sclerosis
 - Loss of myelin and axons in peripheral nerves and sensory roots on autopsy (Watson 1988, 1991)
 - Skin biopsy: 20-40% reduction of cutaneous nerve fiber (Fetell 2023)
 - Some reduction in nerve fiber density noted on the contralateral side
 - Dorsal root ganglia: inflammation, hemorrhagic necrosis, neuronal loss (Gilden 2000)
 - Persistence of inflammation for 1-2 years
 - Atrophy of dorsal horn on autopsy (Kost 1996)



Post-Herpetic Neuralgia

- Impairs all sensory fiber groups: large and small myelinated, unmyelinated C fibers
- Axonal pathology
- EMG/NCS: reduced or absent SNAPs (60%)
 - CMAP reduction 18%
 - Abnormal spontaneous activity is possible, more likely with SZP
- Clinical diagnosis: EMG/NCS is not routinely indicated
 - Small fiber pathology predominates
- EMG/NCS is recommended for SZP

Johnson 2014, Mondelli 2022



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Risk Factors for Post-Herpetic Neuralgia

- Age>60
- Severe rash manifestations or high viral load
- Smoking history
- Alcohol Abuse
- Immunosuppressive status
- COPD
- Diabetes
- HTN
- Malignant tumors
- CKD

Wang 2025



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Reduction of Risk

- Recombinant zoster vaccine (Shingrix/RZV): 87-91% effectiveness against post-herpetic neuralgia
- Older live attenuated vaccine (Zostavax): 65-67% effectiveness
- Means of protection: prevent herpes zoster
- Duration of protection lasts at least 3 years
 - No data to recommend revaccination
- No published evidence that specifically addresses risk of SZP

Zerbo 2025, de Oliveira Gomes 2023



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Segmental Zoster Paresis

- Profound motor weakness without recovery
- Absent CMAPs
- Absent SNAPs
- Cervical stenosis
- Age 79
- Involvement of C6-7 roots

Post-Herpetic Neuralgia

- Severe pain
- Absent SNAPs
- Multiple co-morbidities
- Extensive initial rash



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Conclusions

- Patients with segmental zoster paresis present a complex electrophysiological picture
- EMG/NCS helps exclude other conditions, localize the lesion, determine prognosis
- Vaccination with recumbent zoster vaccine is best means of prevention



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ATYPICAL MEDIAN NEUROPATHY: IT'S NOT ALWAYS CARPAL TUNNEL SYNDROME

McKibben R

ABSTRACT

BACKGROUND: Carpal tunnel syndrome (CTS) with focal compression of the median nerve at the wrist is the most common suspected diagnosis referred for electrodiagnostic testing (EDX). However, other conditions can present with mimicking clinical signs and symptoms and may include alternative etiologies such as proximal nerve compression, trauma, anatomic variability, acquired inflammatory processes, or hereditary neuropathies.

DISCUSSION: Median nerve anatomy and common sites of entrapment will be reviewed followed by presentation and discussion of clinical case examples with accompanying EDX of atypical median neuropathy resulting from various etiologies. It is important to expand clinical testing protocols to assure appropriate nerve conduction (NCS) and needle electromyography (EMG) selections recognizing that median neuropathy does not always equal carpal tunnel syndrome.

Rick McKibben, PT, DSc, ECS



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Atypical Median Neuropathy

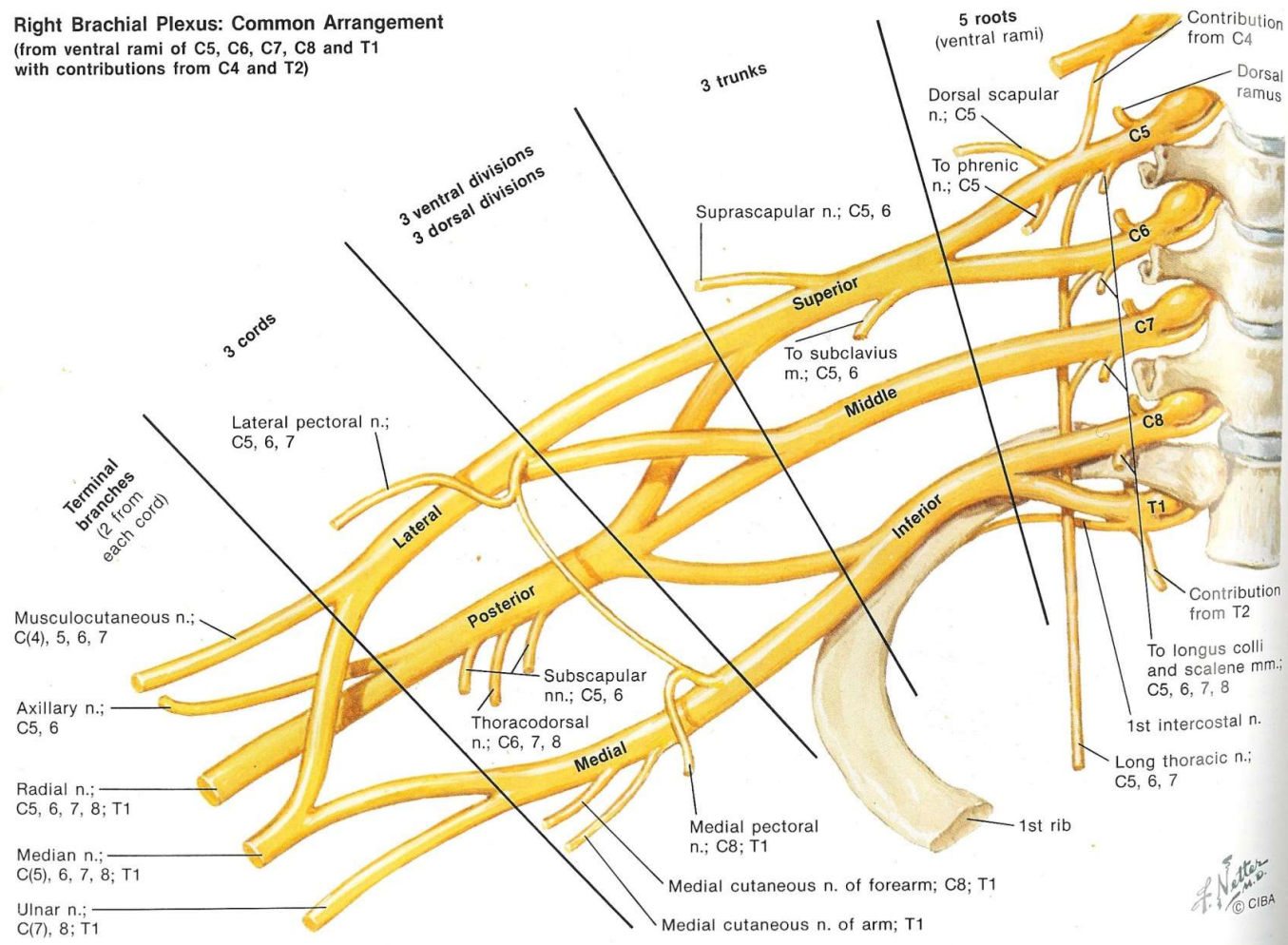
It's not always Carpal Tunnel

Rick McKibben, PT, DSc, ECS
2026 EMG Symposium





Right Brachial Plexus: Common Arrangement
(from ventral rami of C5, C6, C7, C8 and T1
with contributions from C4 and T2)





Median Nerve Anatomy and Entrapment

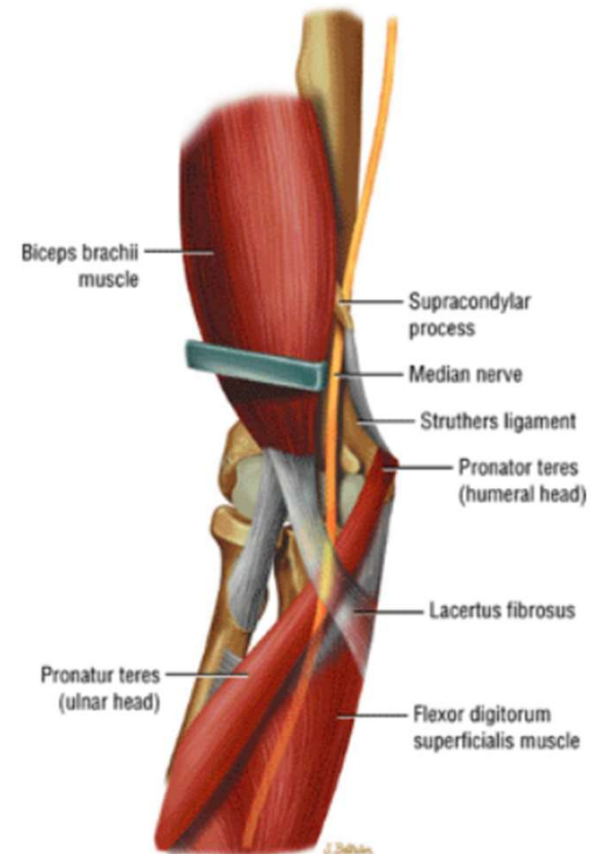
- Axilla
 - C6-T1
 - Upper, middle, lower trunks
 - Lies with axillary artery and vein under pec major
 - Entrapment in axilla is rare
- Arm
 - Common NVB
 - Brachial artery
 - Uln, MBC, MABC N's
 - Near Coracobrachialis the ulnar nerve leaves NVB
 - Median N & Brachial A move anterior near brachialis and intramuscular septum under biceps
 - Entrapment rare (rifle, tourniquet)
 - 1% Ligament of Struthers- bony spur from distal humerus to med epicondyle

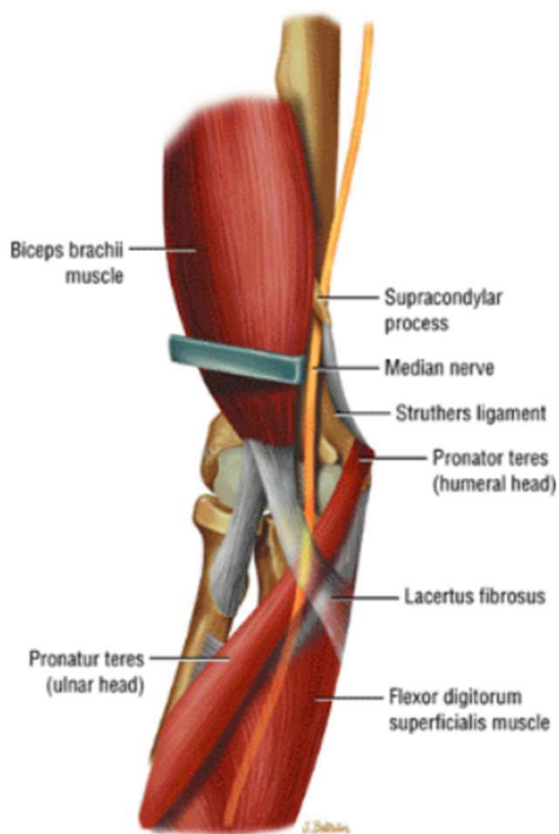




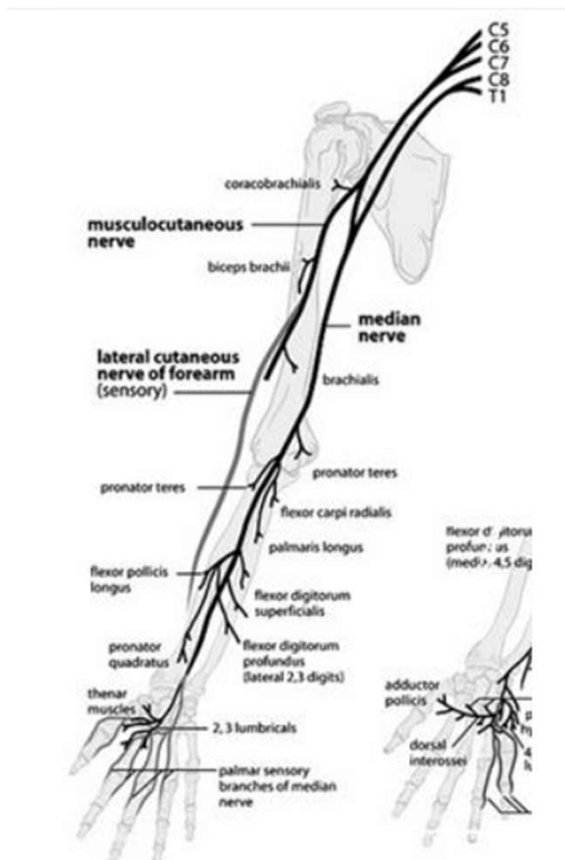
Median Nerve Anatomy and Entrapment

- Elbow
 - Medial to brachial artery and biceps, posterior to lacertus fibrosis
 - Entrapment not uncommon @ lacertus fibrosis or between 2 heads of pronator teres.
- Forearm
 - Innervates PT
 - Passes deep to PT
 - Under FDS with anterior interosseus artery (branch of ulnar artery)
 - Approx 5 cm proximal to flexor retinaculum, emerges behind lateral edge of FDS
 - Innervates FCR, PL, FDS
 - Entrapment @ PT or arch of FDS





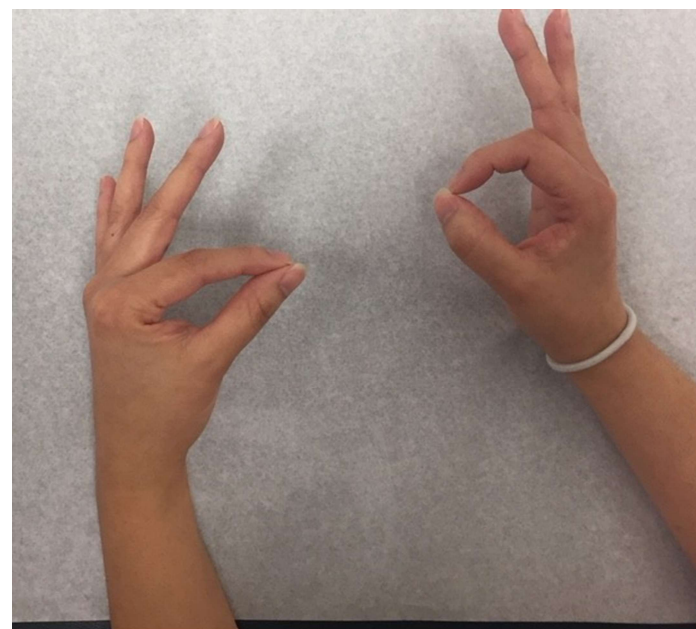
<https://musculoskeletalkey.com/entrapment-neuropathies-of-the-upper-extremity/>



<https://www.orthobullets.com/anatomy/10106/median-nerve>

• AIN

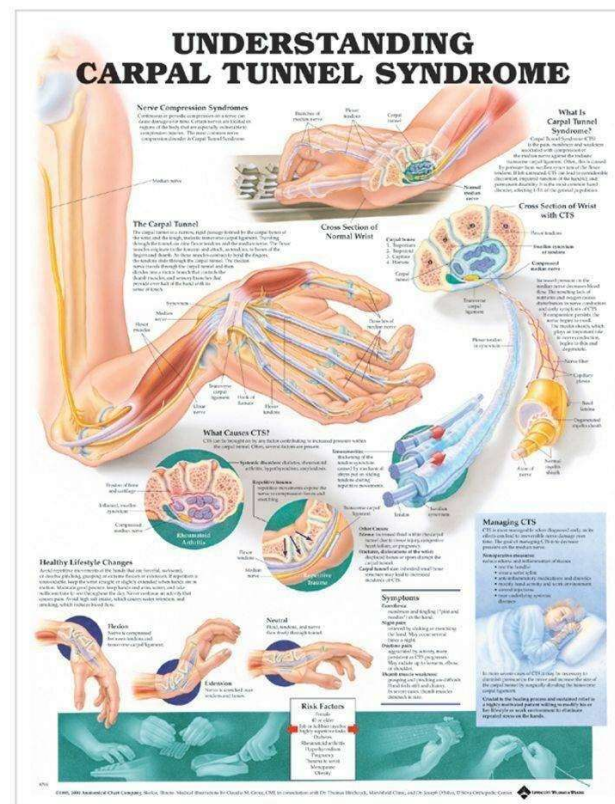
- Branches after exiting PT (pure motor)
- Innervates FDP, FPL, PQ
- Common entrapment
 - Lacertus fibrosus
 - Deep head PT
 - Kiloh-Nevin Syndrome





Median Nerve Anatomy and Entrapment

- Wrist
 - Lies b/w FCR and PL/FDS
 - Palmar cutaneous branch arises medially about 5 cm proximal to radial styloid innervates palm and thenar eminence
 - Under flexor retinaculum in CT with other contents
 - Of course, entrapment common...CTS
- Hand
 - Lateral Branch
 - Recurrent Motor - APB, FPB, OP
 - 1st lumbrical, skin of radial/ulnar digit I and radial digit II
 - Intermediate Branch
 - 2nd lumbrical, skin of adjacent sides digits II-III
 - Medial Branch
 - Communicates with superficial ulnar nerve to innervate adjacent sides of digits III-IV



<https://anatomywarehouse.com/understanding-carpal-tunnel-syndrome-anatomical-chart-a-102731?srltid=AfmBOoq9Rd10AIVOReZilZQgjXK2cZ1X0ltdpQK6iZwFKAWpPuKUh7cU>



Electrodiagnostic Testing – Median Nerve

**YOU'VE GOT A
FEW OPTIONS!**



AAEM PRACTICE TOPIC IN ELECTRODIAGNOSTIC MEDICINE

American Association of Electrodiagnostic Medicine
421 First Avenue S.W., Suite 300 East, Rochester, MN 55902 (507/288-0100)

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Muscle Nerve 25: 918–922, 2002

PRACTICE PARAMETER FOR ELECTRODIAGNOSTIC STUDIES IN CARPAL TUNNEL SYNDROME: SUMMARY STATEMENT*

AMERICAN ASSOCIATION OF ELECTRODIAGNOSTIC MEDICINE, AMERICAN ACADEMY OF NEUROLOGY,
and AMERICAN ACADEMY OF PHYSICAL MEDICINE AND REHABILITATION



Atypical CTS?

- 71yom – retired psychiatrist
- Referred to r/o CTS
- c/o isolated L paresthesias medial ½ digit III
- Worse when brushing teeth, leans on open palm
- Light touch dysesthesia only to medial ½ digit III
- Provocation with Durkan's
- All other neuromotor screening very normal.



Nerve Conduction Studies

Anti Sensory Summary Table

Site	NR	Peak (ms)	Norm Peak (ms)	O-P* Amp (μV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Anti Sensory (3rd Digit)											
Palm		1.9	<2.2	41.3	>10	Palm	3rd Digit	1.9	7.0	37	
Wrist		3.5	<3.6	30.7		Wrist	Palm	1.6	7.0	44	>39
Left Ulnar Anti Sensory (5th Digit)											
Wrist		3.4	<3.4	24.2	>10	Wrist	5th Digit	3.4	12.0	35	

Comparison Summary Table

Site	NR	Peak (ms)	O-P* Amp (μV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)
Left Median/Radial Comparison (Digit 1)							
Median 10cm		2.8	16.6	>7	Median 10cm	Radial 10 cm	0.3
Radial 10 cm		2.5	11.6				
Left Median/Ulnar Comparison (Digit 4)							
Median Wr		3.7	13.9		Median Wr	Ulnar Wr	0.2
Ulnar Wr		3.9	17.1				
Left Median/Ulnar Palm Comparison (Lumbrical/2nd Palmar Interosseus)							
Median		3.9			Median	Ulnar	0.1
Ulnar		3.8					

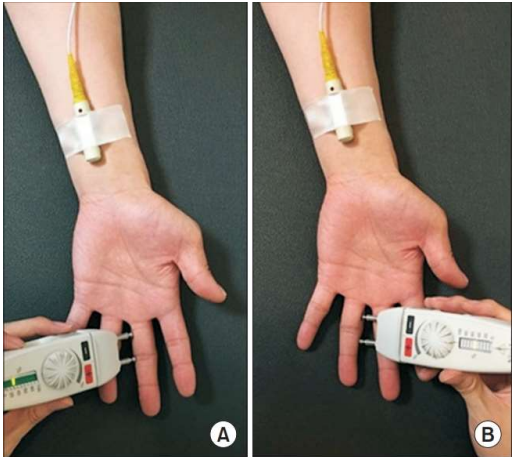
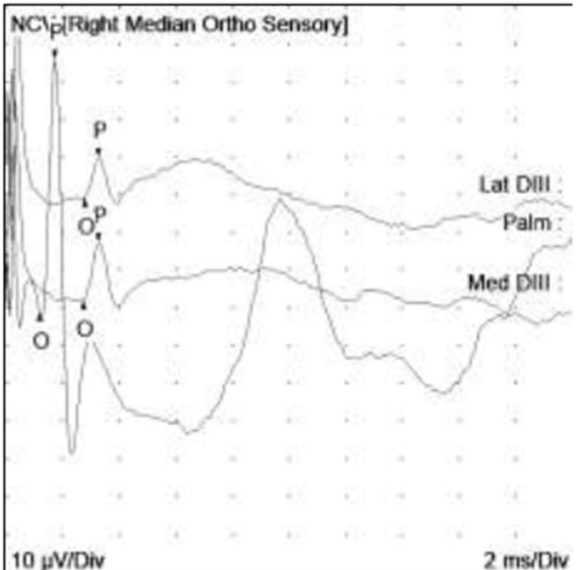
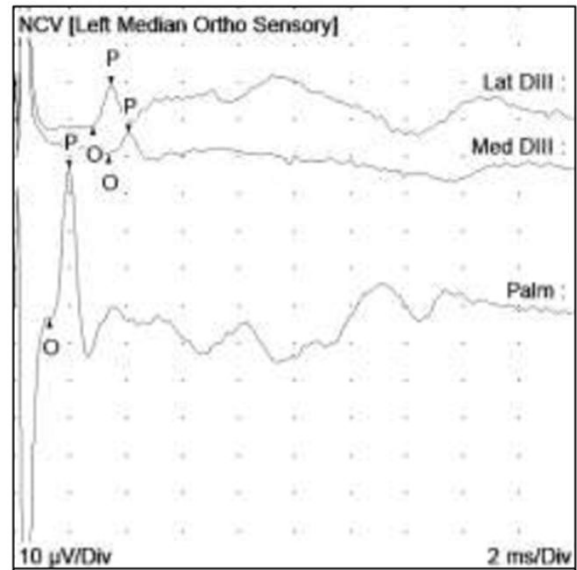
Motor Summary Table

Site	NR	Onset (ms)	Norm Onset (ms)	O-P* Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Motor (Abd Poll Brev)												
Wrist		3.8	<4.2	8.0	>2.8	6.41	Wrist	Elbow	4.8	26.0	54	>50
Elbow		8.6		8.8		6.72						



Ortho Sensory Summary Table

Site	NR	Peak (ms)	Norm Peak (ms)	O-P* Amp (µV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Ortho Sensory (Wrist)											
Lat DIII		3.5		10.9		Lat DIII	Wrist	3.5	14.0	40	>39
Med DIII		4.1		5.4		Med DIII	Wrist	4.1	14.0	34	>39
Palm		2.2		40.1		Wrist	Palm	2.2	7.0	32	>39
Right Median Ortho Sensory (Wrist)											
Lat DIII		3.3		10.5		Lat DIII	Wrist	3.3	14.0	42	>39
Med DIII		3.3		15.3		Med DIII	Wrist	3.3	14.0	42	>39
Palm		1.7		67.3		Wrist	Palm	1.7	14.0	41	>39



Case Report
Palmar Digital Neuropathy With Anatomical Variation of Median Nerve: Usefulness of Orthodromic Technique: A Case Report
Jun Soo Noh, MD¹, Jong Woong Park, MD, PhD², Hee-Ryu Kwon, MD, PhD³
Annals of Rehabilitation Medicine 2019;42(3):341-346. DOI: <https://doi.org/10.5535/arm.2019.42.3.341>
Published online: June 28, 2019



Atypical CTS?

Impression: (Abnormal Study)

1. There is evidence to suggest transcarpal sensory demyelination **with partial axonopathy** involving the L median sensory nerve that involves the medial digital branch. This correlates very well with primary complaints. All remaining studies involving the L median sensory and motor nerves are well WNL so this seems to involve only the fascicles destined for the medial digital branch to digit III.
2. There are no findings to suggest any other neuropathic processes involving the L upper.

Is the low amplitude related to:

- A. axonopathy as written above
- B. **partial sensory conduction block at the wrist**
- C. **we don't really know?**

Advised patient not to lean on open hand, symptoms resolved



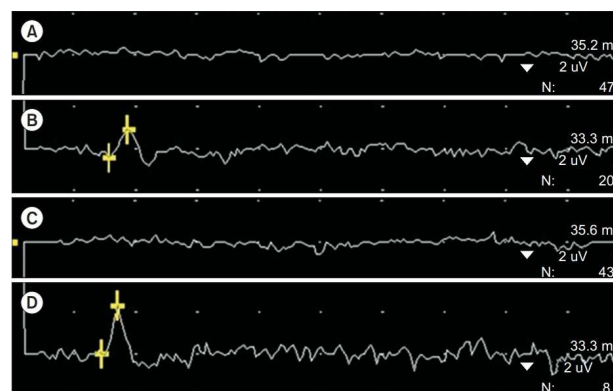
Atypical CTS?

Case Report

Palmar Digital Neuropathy With Anatomical Variation of Median Nerve: Usefulness of Orthodromic Technique: A Case Report

Jun Soo Noh, MD¹ , Jong Woong Park, MD, PhD² , Hee-Kyu Kwon, MD, PhD¹ 

Annals of Rehabilitation Medicine 2019;43(3):341-346. DOI: <https://doi.org/10.5535/arm.2019.43.3.341>
Published online: June 28, 2019





Atypical CTS?

- 58yof 6 weeks s/p distal radius Fx
- Underwent ORIF 4 weeks after Fx
- Post-op swelling, pain, paresthesias developed digits I > II
- Weakness in the APB/OP, very guarded

Motor Summary Table

Site	NR	Onset (ms)	Norm Onset (ms)	O-P* Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Anterior Interosseus Motor (Flex Poll Long)												
Right Elbow		3.8		1.4		9.84						
Left Median Motor (Abd Poll Brev) severe partial conduction block at wrist												
Palm		2.5		1.1	>2.8	3.59	Wrist	Palm	4.4	5.0	11	>29
Wrist		6.9	<4.2	0.2	>2.8	3.75	Wrist	Elbow	3.9	23.0	59	>50
Elbow		10.8		0.2		4.22						
Left Ulnar Motor (Abd Dig Minimi)												
Wrist 8cm		2.5	<3.6	4.1	>4.0	6.09	B Elbow	Wrist 8cm	3.1	19.0	61	>49
B Elbow		5.6		4.1		6.09	A Elbow	B Elbow	1.4	8.0	57	>44
A Elbow		7.0		4.0		6.09						



Nerve Conduction Studies

Anti Sensory Summary Table

Site	NR	Peak (ms)	Norm Peak (ms)	O-P* Amp (μV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Anti Sensory (3rd Digit)											
Palm		1.8	<2.2	31.8	>10	Palm	3rd Digit	1.8	7.0	39	
Wrist		3.8	<3.6	24.7		Wrist	Palm	2.0	7.0	35	>39
Left Median Anti Sensory Run #2 (2nd Digit) partial sensory conduction block at wrist											
Palm		1.6	<2.2	22.5	>10	Palm	2nd Digit	1.6	7.0	44	
Wrist		3.7	<3.6	10.7		Wrist	Palm	2.1	7.0	33	>39
Left Radial Anti Sensory (Base 1st Digit)											
Wrist		2.0	<2.5	14.4		Wrist	Base 1st Digit	2.0	10.0	50	
Left Ulnar Anti Sensory (5th Digit)											
Wrist		2.8	<3.4	21.5	>10	Wrist	5th Digit	2.8	12.0	43	

Comparison Summary Table

Site	NR	Peak (ms)	O-P* Amp (μV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)
Left Median/Radial Comparison (Digit 1)							
Median 10cm	NR			>7	Median 10cm	Radial 10 cm	
Radial 10 cm		2.6	6.5				

EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Left	BrachioRad	Radial	C5-6	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	FlexCarRad	Median	C6-7	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	FlexPolLong	Median (Ant Int)	C7-8	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Ext Poll Long	Radial (Post Int)	C7-8	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Abd Poll Brev	Median	C8-T1	Nml	2+	2+	Nml	Distorted	>12ms	3+	Rapid	Reduced	poor activation
Left	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	



Impression: (Abnormal Study)

- 1. Evidence suggests severe L focal median neuropathy at the wrist.**
 - a. This is an unusual pattern of fascicular involvement most involving the recurrent median motor and median digital branch to digit I much more so than the digital branches to digits II > III.**
 - b. There is motor/sensory axonopathy with demyelination and severe partial transcarpal conduction block.**
- 2. There are no findings to suggest notable superimposed neuropathic processes involving the L upper.**



> J Hand Surg Glob Online. 2022 Aug 31;4(6):324-327. doi: 10.1016/j.jhsg.2022.08.001.
eCollection 2022 Nov.

Incidence of Carpal Tunnel Syndrome after Distal Radius Fracture

Margaret E Cooke ¹, Alex Gu ², Lauren E Wessel ³, Alexander Koo ⁴, Daniel A Osei ⁵,
Duretti T Fufa ⁵

- *+23,000 patients with DRF, CTS incidence after 6 month*
- *9.2% diagnosed within 6 months*
 - *NO ORIF 6.3%*
 - ***ORIF 19.8%***
- ***Higher incidence of CTS with ORIF – reports up to 30%***
- ***Prophylactic CTR?***



► J Wrist Surg. 2022 Nov 9;12(3):211–217. doi: [10.1055/s-0042-1756501](https://doi.org/10.1055/s-0042-1756501)

Carpal Tunnel Release Performed during Distal Radius Fracture Surgery

[Alyssa Rothman](#)^{1,✉}, [Aneesh V Samineni](#)², [David C Sing](#)¹, [Joanne Y Zhang](#)³, [Andrew B Stein](#)¹

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PMCID: PMC10202570 PMID: [37223388](https://pubmed.ncbi.nlm.nih.gov/37223388/)

► J Wrist Surg. 2019 Apr 22;8(5):366–373. doi: [10.1055/s-0039-1685203](https://doi.org/10.1055/s-0039-1685203)

Surgical Decision-Making in Median Neuropathy Associated with Distal Radius Fractures

[Sezai Özkan](#)^{1,2}, [Chaitanya S Mudgal](#)^{1,✉}, [Brady T Evans](#)³, [Colyn J Watkins](#)³, [Marilyn M Heng](#)⁴, [Frank W Bloemers](#)²

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PMCID: PMC6773573 PMID: [31579544](https://pubmed.ncbi.nlm.nih.gov/31579544/)

Not sufficient evidence
Not recommended without preoperative Sx

► J Hand Surg Glob Online. 2024 Jul 14;6(5):631–635. doi: [10.1016/j.jhsg.2024.05.010](https://doi.org/10.1016/j.jhsg.2024.05.010).
eCollection 2024 Sep.

Prophylactic Carpal Tunnel Release During Volar Plating of Distal Radius Fractures: A Review

[Harin B Parikh](#)¹, [Violette C Simon](#)¹, [Stuart H Kuschner](#)¹

Affiliations + expand

PMID: 39381388 PMCID: [PMC11456658](https://pubmed.ncbi.nlm.nih.gov/PMC11456658/) DOI: [10.1016/j.jhsg.2024.05.010](https://doi.org/10.1016/j.jhsg.2024.05.010)



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Predictors of Acute Carpal Tunnel Syndrome Following ORIF of Distal Radius Fractures: A Matched Case–Control Study

[Brandon E. Earp, MD](#) · [Ariana N. Mora, BA](#) · [W. Emerson Floyd, IV, BA](#) · [Philip E. Blazar, MD](#)

Affiliations & Notes Article Info

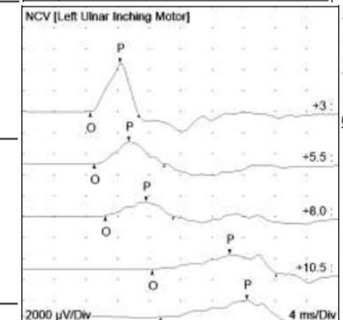
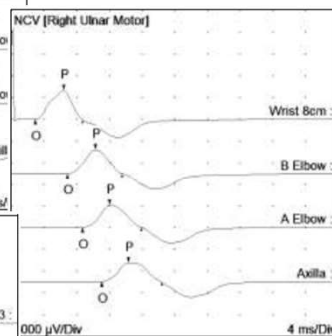
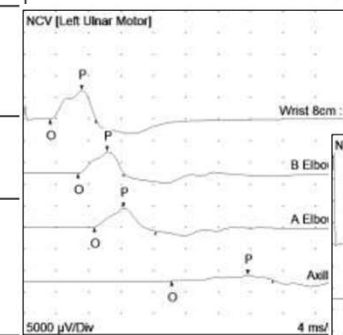
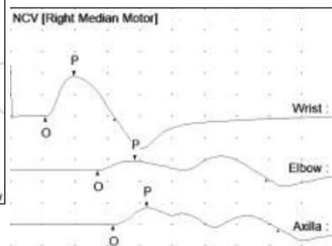
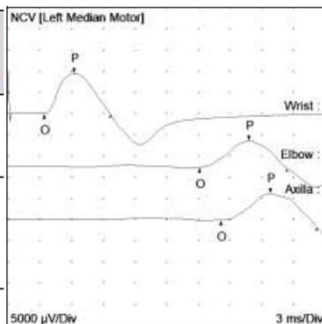


Atypical CTS?

- 19yof presented with objective L median paresthesias
- Prior R CTS at age 15, did well with CTR
- No comorbidities
- No objective findings on clarifying assessment
- EDX showed 22m/s median motor NCV in forearm
- Med/Rad D1, Med DIII, Ulnar motor/sensory, superficial radial sensory all WNL, EMG normal
- Underwent CTR – No relief
- Postoperatively - MRI L forearm
 - 3cm neural/perineural cystic change median nerve in the distal 1/3rd forearm
- Developed subtle L ulnar paresthesias, sent for another EDX

Motor Summary Table

Site	NR	Onset (ms)	Norm Onset (ms)	O-P* Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Anterior Interosseus Motor (Flex Poll Long)												
Right Elbow		3.4		4.5		9.38	Right Elbow	Left Elbow	0.0	0.0		
Left Elbow		3.4		4.7		10.78						
Left Median Motor (Abd Poll Brev)												
Wrist		3.4	<4.2	9.5	>2.8	6.25	Wrist	Elbow	14.7	24.5	17	>50
Elbow		18.1		6.3		8.28	Elbow	Axilla	2.1	10.0	48	>50
Axilla		20.2		6.3		9.06						
Right Median Motor (Abd Poll Brev) MGA unreliable forearm NCV												
Wrist		3.3	<4.2	9.3	>2.8	6.41	Wrist	Elbow	4.8	24.0	50	>50
Elbow		8.1		2.2		15.63	Elbow	Axilla	1.4	8.0	57	>50
Axilla		9.5		3.9		15.31						
Right Peroneal Motor (Ext Dig Brev)												
Ankle 8cm		5.5	<6.2	7.8	>0.700	6.41	B Fib	Ankle 8cm	6.7	30.0	45	>38
B Fib		12.2		7.5		6.56	Poplt	B Fib	1.4	8.0	57	>39
Poplt		13.6		7.4		6.72						
Right Tibial Motor (Abd Hall Brev)												
Ankle 8cm		5.2	<6.1	12.5	>2.8	8.13	Knee	Ankle 8cm	8.7	38.0	44	>38
Knee		13.9		8.3		9.22						
Left Ulnar Motor (Abd Dig Minimi)												
Wrist 8cm		3.3	<3.6	6.6	>4.2	5.63	B Elbow	Wrist 8cm	3.4	19.0	56	>49
B Elbow		6.7		4.9		5.63	A Elbow	B Elbow	2.1	8.0	38	>44
A Elbow		8.8		4.3		7.50	Axilla	A Elbow	9.5	8.0	8	>50
Axilla		18.3		1.3		12.50						
Right Ulnar Motor (Abd Dig Minimi)												
Wrist 8cm		3.0	<3.6	6.7	>4.2	5.78	B Elbow	Wrist 8cm	3.9	19.5	50	>49
B Elbow		6.9		5.6		6.72	A Elbow	B Elbow	1.9	8.0	42	>44
A Elbow		8.8		5.2		7.19	Axilla	A Elbow	2.3	8.0	35	>50
Axilla		11.1		4.3		7.34						
Left Ulnar Inching Motor (ADM)												
+3		8.6		4.6		6.09	+3	+5.5	0.5	2.5	50	>35
+5.5		9.1		2.1		8.91	+5.5	+8.0	1.4	2.5	18	>35
+8.0		10.5		1.3		8.59	+8.0	+10.5	5.9	2.5	4	>35
+10.5		16.4		1.4		15.78	+10.5	+13.0	1.1	2.5	23	>35
+13.0		17.5		1.6		15.31						





Nerve Conduction Studies

Anti Sensory Summary Table

Site	NR	Peak (ms)	Norm Peak (ms)	O-P* Amp (μV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Anti Sensory (3rd Digit)											
Palm		1.6	<2.2	61.9	>10	Palm	3rd Digit	1.6	7.0	44	
Wrist		3.0	<3.6	49.2		Wrist	Palm	1.4	7.0	50	>39
Elbow		6.9		12.1		Elbow	Wrist	3.9	24.5	63	
Right Median Anti Sensory (3rd Digit)											
Palm		1.7	<2.2	57.8	>10	Palm	3rd Digit	1.7	7.0	41	
Wrist		3.0	<3.6	33.7		Wrist	Palm	1.3	7.0	54	>39
Elbow		7.1		7.8		Elbow	Wrist	4.1	24.0	59	
Right Sup Peron Anti Sensory (Ant Lat Mall)											
14 cm		4.0	<4.2	6.5	>2.0	14 cm	Ant Lat Mall	4.0	14.0	35	
Right Sural Anti Sensory (Lat Mall)											
Calf		3.7	<4.2	13.4	>5.0	Calf	Lat Mall	3.7	12.0	32	
Left Ulnar Anti Sensory (5th Digit)											
Wrist		2.9	<3.4	33.3	>10	Wrist	5th Digit	2.9	12.0	41	



Comparison Summary Table

Site	NR	Peak (ms)	O-P* Amp (µV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)
Left Median/Radial Comparison (Digit 1)							
Median 10cm		2.5	37.3	>7	Median 10cm	Radial 10 cm	0.1
Radial 10 cm		2.4	7.7				

Mixed Onset Summary Table

Site	Onset (ms)	O-P* Amp (µV)	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)
Left Mixed Median Forearm Mixed Onset (Medial Elbow)								
L Wrist	3.7	2.1	1.56					
R Wrist	3.7	5.9	1.97					

F Wave Studies

NR	F-Lat (ms)	Lat Norm (ms)	L-R F-Lat (ms)
Right Peroneal (Mrkrs) (EDB)			
	49.60	<60	

H Reflex Studies

NR	H-Lat (ms)	Lat Norm (ms)	L-R H-Lat (ms)	L-R Lat Norm
Right Tibial (Gastroc)				
NR		<36		2.0

EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Left	Abd Poll Brev	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	FlexCarRad	Median	C6-7	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	BrachioRad	Radial	C5-6	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	



Impression: (Abnormal Study with Complex Findings)

1. There is unusual segmental motor slowing and temporal dispersion most consistent with demyelination involving several nerve segments not typically involved in focal mononeuropathies that include:
 - a. The L median motor nerve in the forearm.
 - b. The L ulnar motor nerve in the axilla. Short segment testing confirms this occurs within 8-11cm proximal to the medial epicondyle.
 - c. Less notably is mild segmental slowing in the R ulnar motor across elbow and axillary segments but without the temporally dispersed waveforms.
 - d. Recommend imaging of these nerve segments.
2. Despite the primary complaint of paresthesias in the L median > ulnar distributions, the distal median and ulnar sensory responses are very well preserved.
3. There are no EMG changes to suggest primary motor axonopathy.
4. Given the several segments noted in the uppers, a brief screening from the lower was performed. While there was an absent R tibial H reflex, there were no abnormalities in other standard motor/sensory nerve conduction studies to suggest more diffuse polyneuropathic changes.
5. Recommend neuromuscular neurology referral to assist in differentiating acquired/inflammatory vs mechanical etiology.



MRI's

- L Brachial Plexus
 - Normal
- L Humerus
 - 4cm fusiform enlargement of ulnar nerve 6cm proximal to elbow
 - 2cm fusiform enlargement of median nerve mid humeral shaft
- R Humerus
 - Undulation and fusiform thickening of ulnar nerve at the distal humerus and median nerve at mid humeral area, less notable than on the L
- Differential for neurofibromas vs multifocal inflammatory neuropathy



Diagnosis?

- Saw local NM specialist – Dx variant CIDP, recommended IVIg
- Went to Mayo Clinic – Same Dx initiated IVIg treatments
- Sx eventually included the feet, never developed any weakness
- MADSAM or unusual CIDP variant.



- My brother
- 67yom 8 days s/p dog bite to the R proximal forearm
- Paresthesias digits II>I>III/IV and LABC
- Frank weakness FCR, APB, FPL, FDP median ½
- History of bilateral nocturnal hand paresthesias, also worse riding mountain bike and motorcycle
- + Familial Hx CTS (mother, brother)





8 days post dog bite

Motor Summary Table

Site	NR	Onset (ms)	Norm Onset (ms)	O-P* Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Anterior Interosseus Motor (Flex Poll Long)												
Right Elbow		4.4		3.7		12.97						
Right Anterior Interosseus Motor (Flex Poll Long) 75% deficit R CMAP												
Right Elbow		5.6		0.9		8.75						
Left Median Motor (Abd Poll Brev)												
Wrist		4.2	<4.2	6.5	>2.8	7.81	Wrist	Elbow	5.0	26.0	52	>50
Elbow		9.2		5.3		8.44						
Right Median Motor (Abd Poll Brev) 65% deficit R CMAP												
Wrist		5.0	<4.2	2.3	>2.8	7.66	Wrist	Elbow	6.1	25.0	41	>50
Elbow		11.1		2.0		7.50	Elbow	Axilla	1.4	8.0	57	>50
Axilla		12.5		2.2		7.19						
Left Radial Motor (Ext Ind Prop)												
Forearm 8cm		2.3	<3.0	6.5		7.66						
Right Radial Motor (Ext Ind Prop)												
Forearm 8cm		3.0	<3.0	4.3		10.16	Brachium	Forearm 8cm	4.2	23.0	55	>50
Brachium		7.2		5.1		9.69						
Left Ulnar Motor (Abd Dig Minimi)												
Wrist 8cm		3.3	<3.6	8.9	>4.2	6.09						
Right Ulnar Motor (Abd Dig Minimi)												
Wrist 8cm		3.4	<3.6	7.0	>4.2	5.78	B Elbow	Wrist 8cm	4.4	23.0	52	>49
B Elbow		7.8		6.8		8.75	A Elbow	B Elbow	1.7	10.0	59	>44
A Elbow		9.5		7.0		9.06						



Nerve Conduction Studies

Anti Sensory Summary Table

Site	NR	Peak (ms)	Norm Peak (ms)	O-P* Amp (uV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Lat Ante Brach Cutan Anti Sensory (Lat Forearm)											
Lat Biceps		2.5		6.6		Lat Biceps	Lat Forearm	2.5	0.0		
Right Lat Ante Brach Cutan Anti Sensory (Lat Forearm) approx. 50% deficit R SNAP											
Lat Biceps		3.1		3.6		Lat Biceps	Lat Forearm	3.1	0.0		
Left Median Anti Sensory Run #1 (3rd Digit)											
Palm		1.9	<2.2	17.2	>10	Palm	3rd Digit	1.9	7.0	37	
Wrist		4.0	<3.6	20.1		Wrist	Palm	2.1	7.0	33	>39
Left Median Anti Sensory Run #2 (2nd Digit)											
Palm		1.9	<2.2	33.2	>10	Palm	2nd Digit	1.9	7.0	37	
Wrist		4.0	<3.6	30.0		Wrist	Palm	2.1	7.0	33	>39
Right Median Anti Sensory Run #1 (3rd Digit)											
Palm		1.9	<2.2	24.9	>10	Palm	3rd Digit	1.9	7.0	37	
Wrist		4.4	<3.6	15.1		Wrist	Palm	2.5	7.0	28	>39
Right Median Anti Sensory Run #2 (2nd Digit) 75% deficit R SNAP											
Palm		1.9	<2.2	11.1	>10	Palm	2nd Digit	1.9	7.0	37	
Wrist		4.2	<3.6	9.8		Wrist	Palm	2.3	7.0	30	>39
Left Radial Anti Sensory (Base 1st Digit)											
Wrist		2.3	<2.5	20.2		Wrist	Base 1st Digit	2.3	10.0	43	
Right Radial Anti Sensory (Base 1st Digit)											
Wrist		2.2	<2.5	16.2		Wrist	Base 1st Digit	2.2	10.0	45	
Left Ulnar Anti Sensory (5th Digit)											
Wrist		3.3	<3.4	16.4	>10	Wrist	5th Digit	3.3	12.0	36	
Right Ulnar Anti Sensory (5th Digit)											
Wrist		3.4	<3.4	22.5	>10	Wrist	5th Digit	3.4	12.0	35	

Comparison Summary Table

Site	NR	Peak (ms)	O-P* Amp (uV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)
Left Median/Radial Comparison (Digit 1)							
Median 10cm		3.3	11.4	>7	Median 10cm	Radial 10 cm	0.6
Radial 10 cm		2.7	5.9				
Right Median/Radial Comparison (Digit 1) 70% deficit R median SNAP							
Median 10cm		3.5	3.4	>7	Median 10cm	Radial 10 cm	0.7
Radial 10 cm		2.8	7.3				



Impression: (Abnormal Study)

- 1. Evidence suggests severe proximal median neuropathy most likely occurring at the elbow proximal to the bifurcation of the median and anterior interosseus nerves.
 - a. This involves severe sensorimotor axonopathy with less significant demyelination.**
 - b. Prognosis is good but expected to be protracted given the severity of axonopathy.****
- 2. There is also partial sensory axonopathy involving the R lateral antebrachial cutaneous nerve.**
- 3. Likely unrelated to the dog bite is evidence of moderate R and mild L median focal neuropathies at the wrists.**
- 4. Recommend repeat study to complete needle EMG in the next month and estimate interval changes should there be any further concerns or issues during recovery.**



MRI

- Neuritis median nerve with partial thickness tear at distal elbow joint
- Hyperintensity of ulnar nerve in cubital tunnel
- Nondisplaced fracture anterior trochlea of medial epicondyle
- Partial thickness biceps tendon with prominent edema and partial tearing involving muscles of the anterior medial arm and elbow.
- Full thickness tear of proximal radial collateral ligament and lateral ulnar collateral ligament
- Partial thickness tear of common flexor tendon



4 months post dog bite – Significantly improved AIN, median APB not appreciably changed

Motor Summary Table

Site	NR	Onset (ms)	Norm Onset (ms)	O-P* Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Anterior Interosseus Motor (Flex Poll Long)												
Right Elbow		5.0		3.2		9.53						
Right Median Motor (Abd Poll Brev)												
Wrist		4.8	<4.2	1.4	>2.8	5.31	Wrist	Elbow	6.1	27.5	45	>50
Elbow		10.9		1.2		5.78	Elbow	Axilla	1.6	8.0	50	>50
Axilla		12.5		1.2		5.63						
Right Ulnar Motor (Abd Dig Minimi)												
Wrist 8cm		2.8	<3.6	8.6	>4.2	5.94	B Elbow	Wrist 8cm	4.2	23.5	56	>49
B Elbow		7.0		8.8		6.41	A Elbow	B Elbow	1.8	10.0	56	>44
A Elbow		8.8		9.0		6.09						



4 months post dog bite – Median Sensory DII/III worse, LABC a little better
Confirmed Median/AIN axonopathy with EMG.

Nerve Conduction Studies

Anti Sensory Summary Table

Site	NR	Peak (ms)	Norm Peak (ms)	O-P* Amp (µV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Lat Ante Brach Cutan Anti Sensory (Lat Forearm)											
Lat Biceps		2.8		4.4		Lat Biceps	Lat Forearm	2.8	14.0	50	
Right Median Anti Sensory (3rd Digit)											
Palm		2.1	<2.2	8.3	>10	Palm	3rd Digit	2.1	7.0	33	
Wrist		4.1	<3.6	8.0		Wrist	Palm	2.0	7.0	35	>39
Right Median Anti Sensory Run #2 (2nd Digit)											
Wrist		4.2	<3.6	4.6	>10	Wrist	2nd Digit	4.2	14.0	33	>39
Right Ulnar Anti Sensory (5th Digit)											
Wrist		2.9	<3.4	17.1	>10	Wrist	5th Digit	2.9	12.0	41	

Comparison Summary Table

Site	NR	Peak (ms)	O-P* Amp (µV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)
Right Median/Radial Comparison (Digit 1)							
Median 10cm		3.6	5.6	>7	Median 10cm	Radial 10 cm	0.9
Radial 10 cm		2.7	7.1				

EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	Biceps	Musculocut	C5-6	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	BrachioRad	Radial	C5-6	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	FlexCarRad	Median	C6-7	Nml	2+	2+	Nml	Nml	>12ms	2+	Rapid	Reduced	
Right	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	FlexPolLong	Median (Ant Int)	C7-8	Nml	2+	2+	Nml	Nml	>12ms	1+	Rapid	Reduced	
Right	ExtDigCom	Radial (Post Int)	C7-8	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Abd Poll Brev	Median	C8-T1	Nml	2+	2+	Nml	Nml	>12ms	1+	Rapid	Reduced	



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ALS (MOTOR NEURON DISORDERS), OFTEN THE GREAT MONONEUROPATHY IMITATOR

Whipple K

ABSTRACT

BACKGROUND: With an incidence rate of fewer than 10 per 100,000 people in the US, it is not uncommon for an EMG practitioner to have an entire career without seeing a single patient with a motor neuron disorder. However, even though it might be a rare patient, EMG is often the diagnostic test that first identifies motor neuron disorders and those providing EMG studies need to be able to recognize those disorders when they are present.

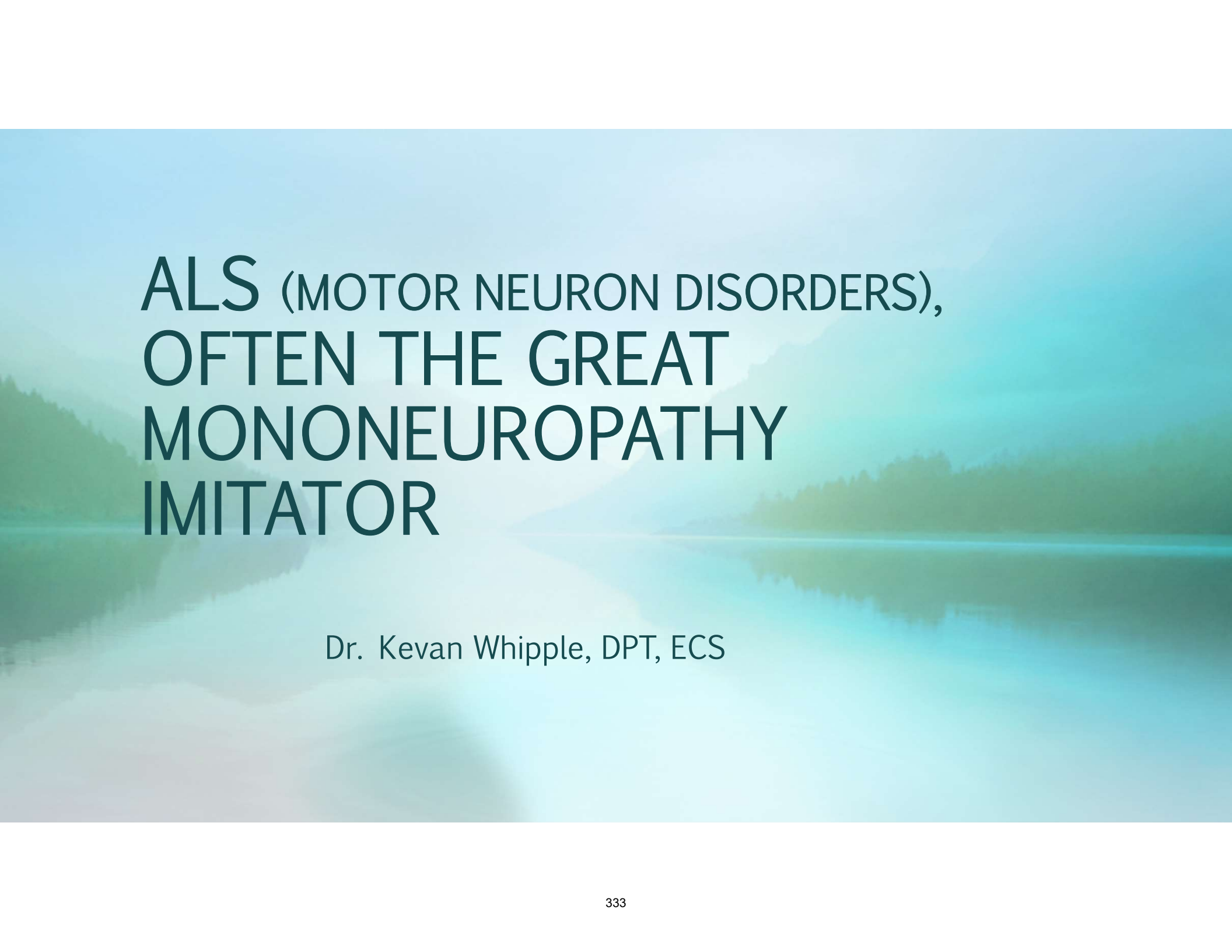
Although there are some hallmark characteristics that are typically associated with EMGs when a motor neuron disorder is present, those hallmark characteristics are often not obvious and can also be overshadowed by more common, everyday findings. In fact, studies have shown that ALS can often be disguised as a common mononeuropathy.

DISCUSSION: This presentation will discuss motor neuron disorders and what EMG providers should know regarding the disease process and how it typically manifests in electrodiagnostic studies.

This presentation will also provide a few personal case examples where patients were initially thought to have mononeuropathies by their referring providers and where the person performing the EMG study also thought the patients had mononeuropathies based on their findings. We will explore how it was later discovered that the patients had ALS, what key steps were missed during EMG with these patients and what EMG providers can learn from these examples in their own clinical practice.

CONCLUSIONS: Though rare, motor neuron disorders are something that every EMG provider needs to understand and be able to recognize when performing electrodiagnostic studies. This presentation will discuss those key elements of the disease process, how those elements manifest in EMG studies and how to distinguish between variants, like mononeuropathies, that are often misclassified when a motor neuron process is present.

Kevan Whipple, DPT, ECS



ALS (MOTOR NEURON DISORDERS), OFTEN THE GREAT MONONEUROPATHY IMITATOR

Dr. Kevan Whipple, DPT, ECS



Introduction

Shout out to Dr Mark Brooks for giving me the tools to assess the handful of Baxter's nerves I see each year...

...the inspiration to provide instruction on topics about difficult and rare cases where it makes all the difference to be the particular person with the particular tool that particular patient needs.

What do you suspect?

HISTORY:

The patient reports in October 2025 he started noticing changes with his tongue, throat and voice. He then started noticing difficulty with shortness of breath with minimal activity. He is experiencing fasciculations throughout in his body and he feels shaky, weak and fatigued throughout. He reports having multiple tests since October to rule out “everything from autoimmune to myasthenia gravis”, all of which he says are normal. He also has a longstanding fracture in the right foot/ankle that has not healed and requires a scooter to be non-weightbearing for several more months. He denies DM, thyroid and adrenal dysfunction.

What do you suspect?

- NCV/EMG all normal

- **EMG**

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	Deltoid	Axillary	C5-6	Nml	1+	1+				Reduced	Nml	fasciculations
Right	Triceps	Radial	C6-7-8	Nml	1+	Nml				Reduced	Nml	fasciculations
Right	Biceps	Musculocut	C5-6	Nml	1+	1+				Reduced	Nml	fasciculations
Right	FlexCarRad	Median	C6-7	Nml	2+	2+				Reduced	Nml	fasciculations
Right	1stDorInt	Ulnar	C8-T1	Incr	2+	2+				Reduced	Nml	fasciculations
Right	Abd Poll Brev	Median	C8-T1	Incr	2+	2+				Reduced	Nml	fasciculations
Left	Deltoid	Axillary	C5-6	Nml	1+	1+				Reduced	Nml	fasciculations
Left	Triceps	Radial	C6-7-8	Nml	1+	1+				Reduced	Nml	fasciculations
Left	Biceps	Musculocut	C5-6	Nml	1+	1+				Reduced	Nml	fasciculations
Left	FlexCarRad	Median	C6-7	Nml	2+	2+				Reduced	Nml	fasciculations
Left	1stDorInt	Ulnar	C8-T1	Incr	2+	2+				Reduced	Nml	fasciculations
Left	Abd Poll Brev	Median	C8-T1	Incr	2+	2+				Reduced	Nml	fasciculations
Right	Orbic Oris	Facial	CN VII	Nml	1+	Nml				Reduced	Nml	fasciculations
Right	Orbic Oculi	Facial	CN VII	Nml	1+	Nml				Reduced	Nml	fasciculations
	Tongue	Bulbar		Incr	2+	2+				Reduced	Nml	fasciculations

What do you suspect?

- 75 yo male complains about significant weakness, especially in his hands. He is also noticing that his voice sounds weaker when he talks.
- NCV testing is normal for sensory and motor fibers bilaterally.
- EMG shows 2 to 3+ fibrillations and positive sharp waves throughout in all muscles sampled (including the tongue), along with fasciculations.
- Referring provider is contacted and report also describes findings of a likely motor neuron disorder

What do you suspect? Continued

- Patient cannot be seen by neurology for at least 3-6 months
- PCP sends him in for another EMG/NCV of the lower extremities because of weakness and falling
- Patient states, “I have gotten so weak since seeing you just a week ago, I don’t think I’ll even be around by the time neurology sees me. I’m the only help my wife has. Without me, she won’t survive either.”
- Similar findings to previous UE study. Normal NCV. EMG shows fibrillations, positive sharp waves and fasciculations throughout
- Was able to call in a favor with a neurologist who saw him a couple of days later. Said it was the most difficult conversation of his career.

What do you suspect? Patient A

Patient A is a 43-year-old male with generally good health. He works as a supervisor at a large chicken egg farm where they collect about a million eggs per day.

In the course of about 2-3 months, he has been noticing progressive weakness in the left hand. There are no complaints in the right hand. The weakness mostly affects fine motor activities, such as writing with a pen, buttoning a shirt and tying shoes.

Nerve conduction studies show mild slowing of the ulnar nerve across the elbow. There is also mildly diminished CMAP amplitude above and below the elbow, normal at the wrist. All sensory findings are normal. All EMG findings are normal (sampled flexor carpi ulnaris and 1st dorsal interosseous for the ulnar nerve and multiple other muscles in bilateral upper extremities).

What does *YOUR* EMG/NCV summary look like for this patient?

To be continued.....

How Likely to See ALS in EMG/NCV?

Incidence Rates in the US:

Carpal tunnel 5-12%

Cubital tunnel 2-5%

Peroneal nerve: 1-2% with LE trauma

Motor neuron disorder: 1 to as high as 9/100,000

Almost 18,000 times more likely to see any of these three nerve entrapment issues **combined** than a motor neuron disorder when performing EMG/NCV

Thank you AI for help with the math :)



What is ALS?

- First described by Charcot in 1869
- Amyotrophic lateral sclerosis (ALS) also known as Lou Gehrig's disease is the most common motor neuron disease in adults
- Will use terms of “ALS” and “motor neuron disorder” interchangeably in this presentation for simplicity, but there are multiple motor neuron disorders
- Rapidly progressive, fatal neurodegenerative disorder of motor neurons
- Dysfunction of upper **and** lower motor neurons (UMNs and LMNs)

Demographics

- Incidence: 1–3 cases/100,000 persons annually
- Prevalence: 5.5 per 100,000 in 2017
 6.6 per 100,000 in 2018
 9.1 per 100,000 in 2023
- Military veterans are up to twice as likely to be diagnosed with the disease as the general public
- 100% fatal, with an average life span of three to five years. More than half live more than three years
- The disease can strike at any age. Symptoms most commonly develop between the ages of 55 and 75.

Pathogenesis and Risk Factors

- Pathogenesis remains largely unknown, likely multiple triggers
 - Glutamate and medications
- ~10% of cases are familial ALS
 - More than 30 genes have been linked to ALS
- ~90% are insidious/sporadic onset
- Possible risk factors include military service, smoking, extreme physical activity, infectious agents, genetics, environmental and occupational exposures, nutritional intake, and trauma

Upper Motor Neuron Signs

- Upper motor neuron (UMN): originates in the brain motor cortex and travels down the spinal cord in the lateral spinal tracts
- Spasticity, hyperreflexia, and pathologic reflexes such as Clonus and the Babinski sign
- Bulbar: exaggerated gag reflex, swallowing, forced yawns, slurred and nasal speech, difficulty chewing, swallowing, or breathing

Lower Motor Neuron Signs

- Lower motor neuron (LMN): originates in the anterior horn of the spinal cord
 - Weakness
 - Atrophy
 - Fasciculations and twitching in the muscles
 - Difficulty walking (tripping, stumbling, etc.)
 - Unexplained difficulty participating in enjoyable activities (running, hiking, musical instruments, singing)
 - Wasting of the hand muscles, usually first noticed in the thenar and intrinsic muscles

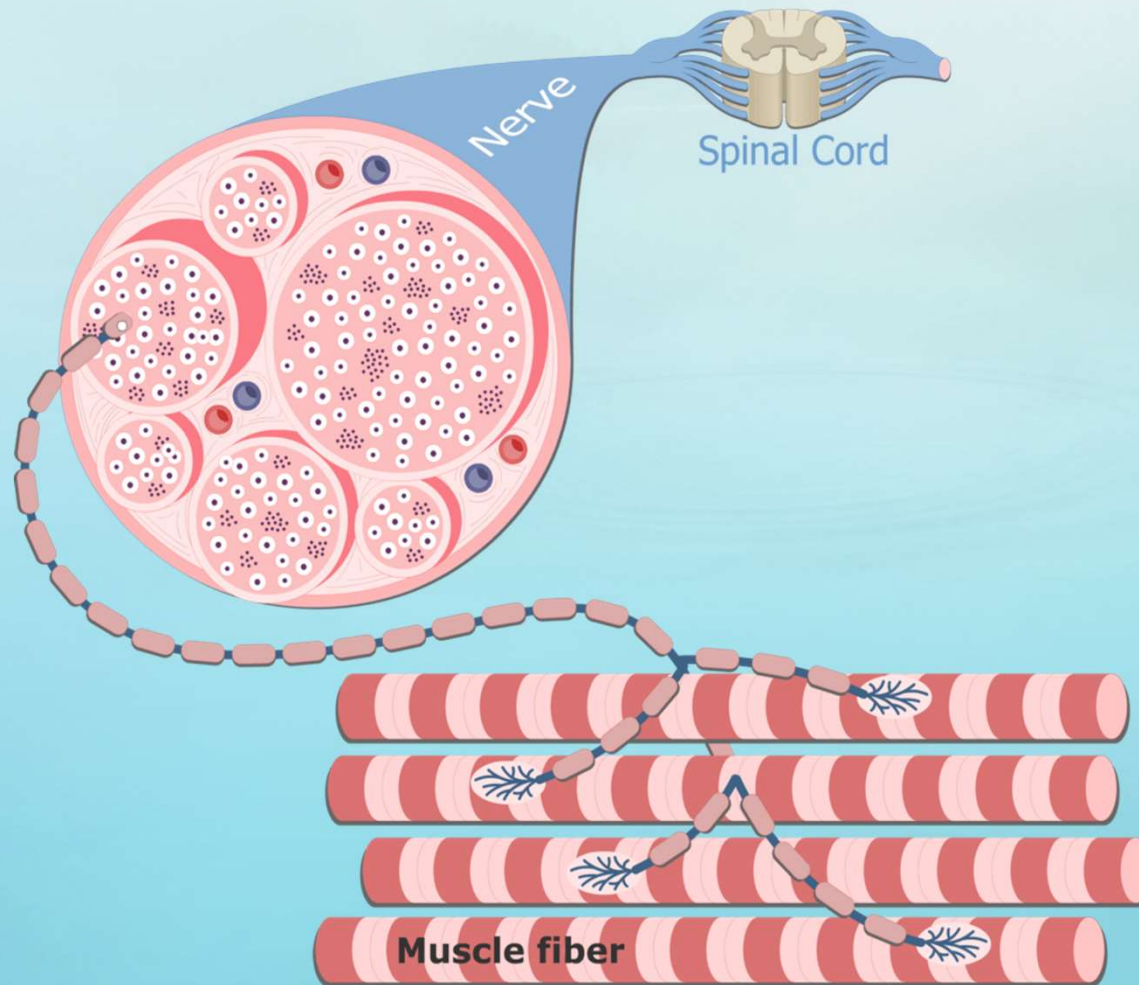
Lower Motor Neuron Signs with NCV/EMG

- NCV: Can see NCV changes in both sensory and motor. However, are often normal, especially if early in the disease progression (remember previous examples had very normal NCV findings).
- EMG: Will show fibrillations and/or positive sharp waves and/or fasciculations
- Why?

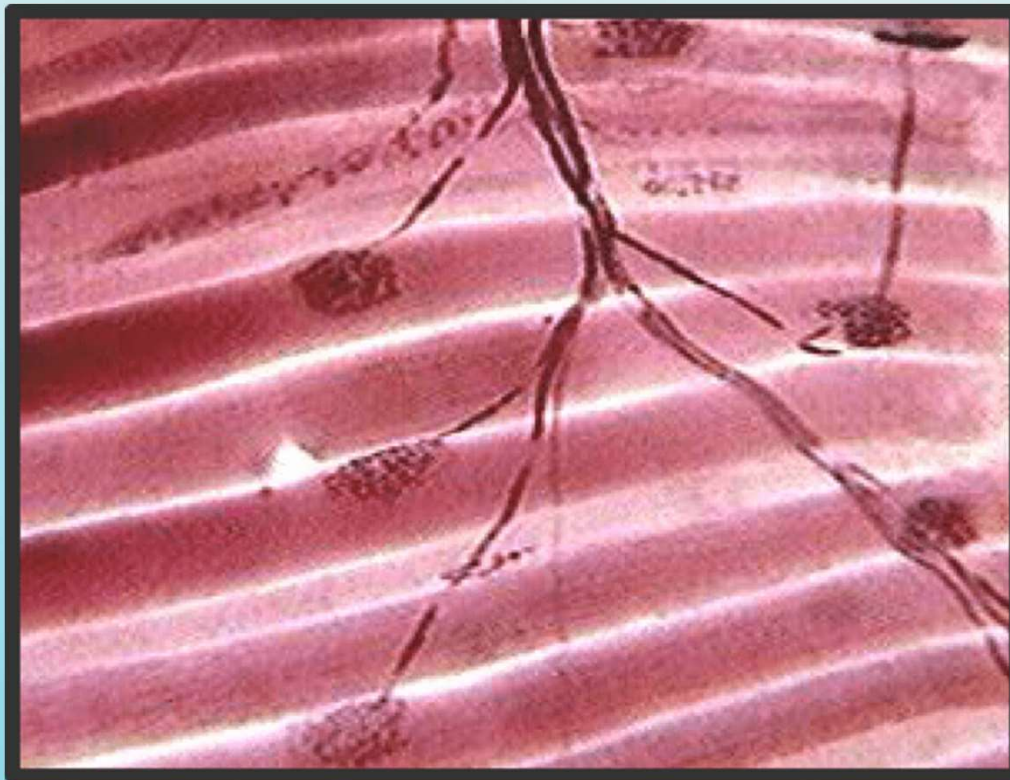
Anterior Horn Cell

- Single anterior horn cell consists of its motor axon and nerve branches, their neuromuscular junctions, and **all of their innervated muscle fibers**
- Significance on EMG is fasciculations
- However, does not mean that spontaneous denervation signs such as fibrillations and positive sharp waves are not also present

Anterior Horn Cell



Motor Unit Microscopy



Diagnosis

- Lower motor neurons (LMN)
 - Electromyography
 - Nerve conduction velocity
- Upper motor neurons (UMN)
 - Clinical examination
- Ruling out of other diseases
- Progressive spread of signs/symptoms

Awaji-shima Criteria 2007

- Electrodiagnostic and clinical signs are of utmost importance, with early EMG being essential
- Blood work for inclusion and exclusion tests
- Imaging (though Awaji-shima criteria says MRI are almost of no help), mostly for exclusion criteria (MS, tumor, white matter disease, etc.)
- Genetic Testing
- Muscle Biopsy
- Disease Progression Monitoring
 - weight changes
 - Rasch Overall ALS Disability Scale

Types of ALS

- Classical ALS: two-thirds of ALS diagnoses
 - Although the average life span is 3–5 years, 10% to 20% can live longer than 10 years
- Bulbar ALS: one-third of ALS diagnoses (dysphagia, dysarthria, difficulty breathing)
 - IX (glossopharyngeal: taste, somatosensory information from tongue, muscles for swallowing)
 - XII (hypoglossal nerve: muscles of tongue)
 - Spinal roots C3–C5: diaphragm
- Primary lateral sclerosis is also very rare type. UMN involvement only with the hallmark symptom of spasticity and weakness
 - Can live >10 years (suspected type for Stephen Hawking)

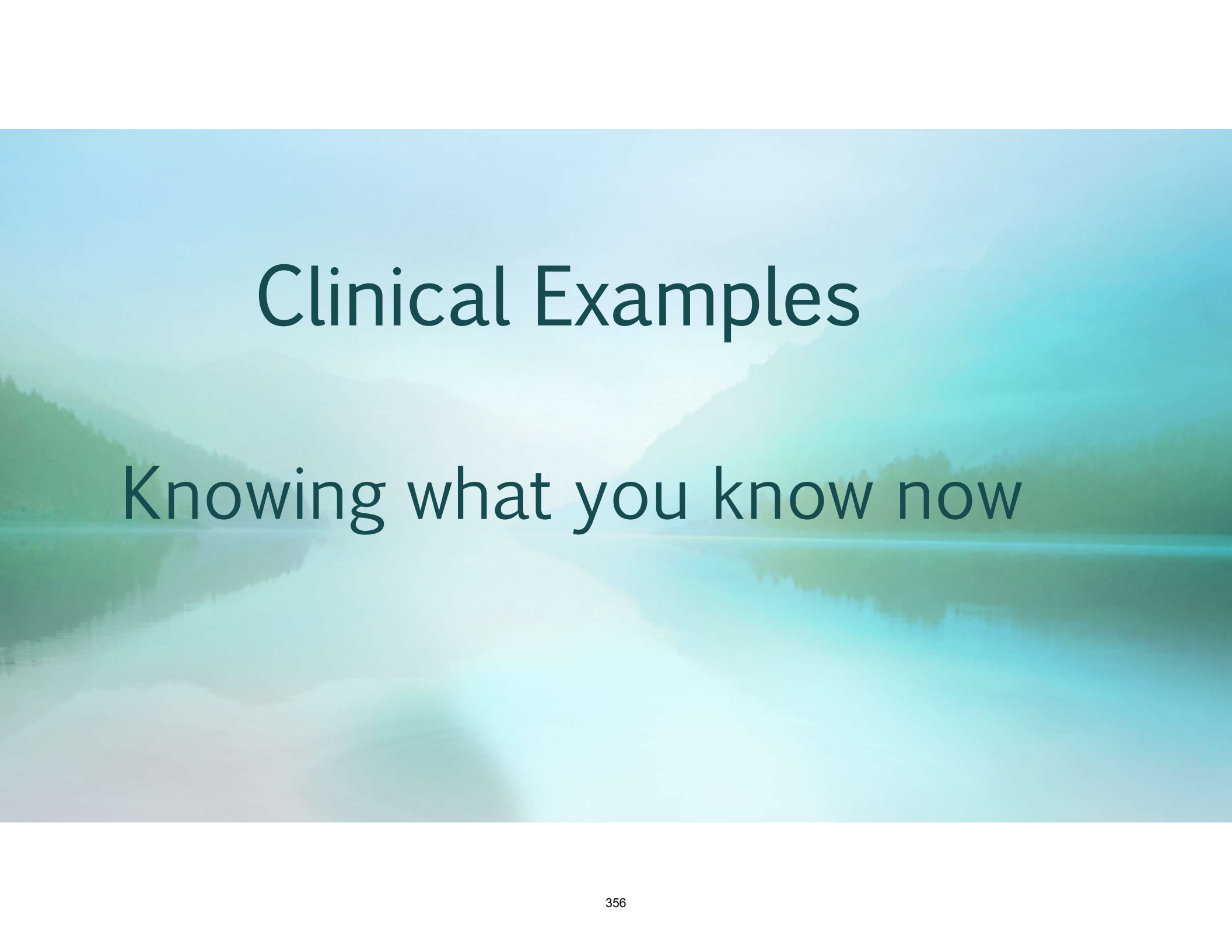
Bulbar

- Important to note that bulbar issues may only be one-third as a primary onset diagnoses. However, most will eventually develop bulbar issues (ex: swallowing, slurred speech, etc.).
- How to test via EMG?



Medications

- Riluzole: glutamate blocker
 - Helps to slow down the worsening of this disease and prolong survival
 - **What is glutamate?** An amino acid that acts as **the brain's main excitatory neurotransmitter**. It's also found in foods, often as Monosodium Glutamate (MSG) for flavor enhancement. It excites neurons to trigger responses, playing a key role in brain function
- Radicava (Edaravone): free-radical scavenger that may reduce oxidative stress
 - Approved by FDA in 2017
 - Found to slow functional deterioration in some patients with ALS
 - Evidence most clear for patients in early stages of ALS (less than two years, live independently)
 - Recommended as an adjunct to Riluzole for most patients with ALS

A serene landscape with misty mountains and a calm lake reflecting the scene. The text is overlaid on this background.

Clinical Examples

Knowing what you know now

Patient A Revisited

Patient A is a 43-year-old male with generally good health. He works as a supervisor at a large chicken egg farm where they collect about a million eggs per day.

In the course of about 2-3 months, he has been noticing progressive weakness in the left hand. There are no complaints in the right hand. The weakness mostly affects fine motor activities, such as writing with a pen, buttoning a shirt and tying shoes.

Nerve conduction studies show mild slowing of the ulnar nerve across the elbow. There is also mildly diminished CMAP amplitude above and below the elbow, normal at the wrist. All sensory findings are normal. All EMG findings are normal (sampled flexor carpi ulnaris and 1st dorsal interosseous for the ulnar nerve and multiple other muscles in bilateral upper extremities).

What does *YOUR* EMG/NCV summary look like for this patient?

Patient A Continued

- Ortho who sent for the study didn't buy that there were ulnar nerve issues at the elbow because the patient had no elbow complaints
- Ordered an MRI of the neck. All normal
- Patient was seen one week later for a lower extremity EMG/NCV study because of foot drop on the left side as well
- NCV showed mild slowing and diminished CMAP across the knee in the peroneal nerve
- **EMG showed fasciculations in several lower extremity muscles**
- EMG was repeated in the upper extremities with a few fasciculations noted, including in the tongue

Patient A Continued

What key findings lead to a suspicion of something more than an isolated ulnar mononeuropathy and isolated peroneal mononeuropathy?

Why is it significant that both an ulnar and peroneal nerve compromise are present and that both started insidiously at about the same time?

Patient B

- 56 yo male is wheeled into the room in a wheelchair. Too weak to walk. Too weak to even feed himself. Can barely talk with a soft raspy voice. Cannot swallow food.
- Had a prior EMG/NCV test performed by a highly recognized neurologist about 6 months prior. That study found reduced CMAPs in the radial motor nerve, along with reduced motor recruitment via EMG in radial innervated muscles. **Diagnosed with a Radial Nerve Mononeuropathy**
- Review of the previous study shows median and ulnar sensory and median and radial motor nerves comprised the entire NCV study, with about 5 muscles sampled via EMG, including triceps and extensor carpi radialis. No contralateral studies were performed with EMG or NCV.

Patient B Continued

- NCV shows reduced CMAPs throughout in the median, ulnar and radial motor nerves bilaterally
- EMG shows fibrillations, positive sharp waves, and fasciculations throughout most muscles sampled, including in the tongue
- Do you agree with the assessment from the previous NCV/EMG study of a radial nerve mononeuropathy?
- What might you have done differently in the prior EMG/NCV study? Why?

Patient C

- 71 yo male complains about not being able to tie fishing flies. Fishing is his favorite pastime as he finds himself declining invitations lately.
- NCV testing shows some mild slowing and amplitude loss across the carpal tunnel as well as at the cubital tunnel bilaterally.
- EMG shows 3+ fibrillations and positive sharp waves in the hypothenar and thenar muscles only bilaterally. None observed in the proximal muscles sampled. No fasciculations.
- Findings on report say bilateral median and ulnar mononeuropathies, with acute processes noted via EMG.
- What would your report say?

Patient C Continued

- Instead of going to orthopedics, PCP refers him to neurology
- Diagnosed with ALS and dies about 3 months later

Patient D

29 yo male. Moved to Utah in June 2025

“June: felt like I was walking around with a 10 lb backpack

July: felt like was walking around with a 50 lb backpack

August: difficult to get out of a chair

September: difficult to walk across the room”

October (when seen for EMG/NCV): can barely tie shoes

- Requires almost a minute to get out of a chair and stand
- Walks like “Flat Stanley” to the exam room

Patient D Continued

- EMG/NCV Findings
 - NCV normal bilaterally in sensory fibers. Reduced amplitude throughout in the motor fibers
 - EMG showed reduced recruitment, reduced amplitude, long duration and polyphasic in proximal muscles in UE and LE. Fasciculations seen only in the left deltoid and left medial gastrocnemius. But had several in both. **No fasciculations seen elsewhere, including in the tongue**
 - What do you suspect?

Key Takeaways

- 1) Don't overstate or "over-find" motor neuron findings (ex: benign fasciculation disorders). But also don't stop looking further if it might be a possibility.
- 2) If the findings do not match the patient presentation, go back and look a little more just to make sure you did things right (remember patients A-C)
- 3) Especially in the case of progressive situations (ex: progressive weakness), it does not hurt to recommend follow-up studies for comparison
- 4) ***Be aware that motor neuron disorders have variable presentations, OFTEN posing as mononeuropathies, especially in the early stages

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WRITING EMG REPORTS: OUT OF THE BOX STRATEGIES FOR SUCCESS

Romero L

ABSTRACT

BACKGROUND

In the field of electrodiagnostic testing (EMG Testing), the EMG report can be considered the most Valuable Final Product of any organization providing testing services. Once the needs of each end customer are identified, reporting can be tailored to meet those needs to achieve success.

DISCUSSION

Beyond the technical aspects of collecting and presenting accurate data in EMG reports, it is prudent for EDX providers to consider how to present the data in a format that meets the needs of different end-users.

CONCLUSION

When the design and delivery of EMG reports identifies and addresses the needs of its various end-customers, it can improve organizational efficiency, promote brand awareness, and streamline the workflow and decision-making of customer that is it delivered to.

Leon C. Romero, PT, DSc, OCS, ECS

Writing EMG Reports

Out of the box strategies for success

Dr. Leon C. Romero, PT, DSc, OCS, ECS

2026 ENMG Symposium

Rocky Mountain University of Health Professions, Provo UT

March 27th-28th, 2026





Dr. Leon C. Romero, PT, DSc, OCS, ECS

- BSc. Biology (1997)
 - Andrew's University (Chemistry Minor; Zool. Emphasis)
- MSc. Physical Therapy (2005)
 - North Georgia College & State Univ. (Dahlonega, GA)
- DSc Clinical Electrophysiology (2016)
 - Rocky Mountain Univ. of Health Professions (Provo, UT)
- ABPTS Orthopedic Clinical Specialist (OCS) - (2008 -present)
- ABPTS Clinical Electrophysiology Specialist (ECS) - (2013-present)

Objectives

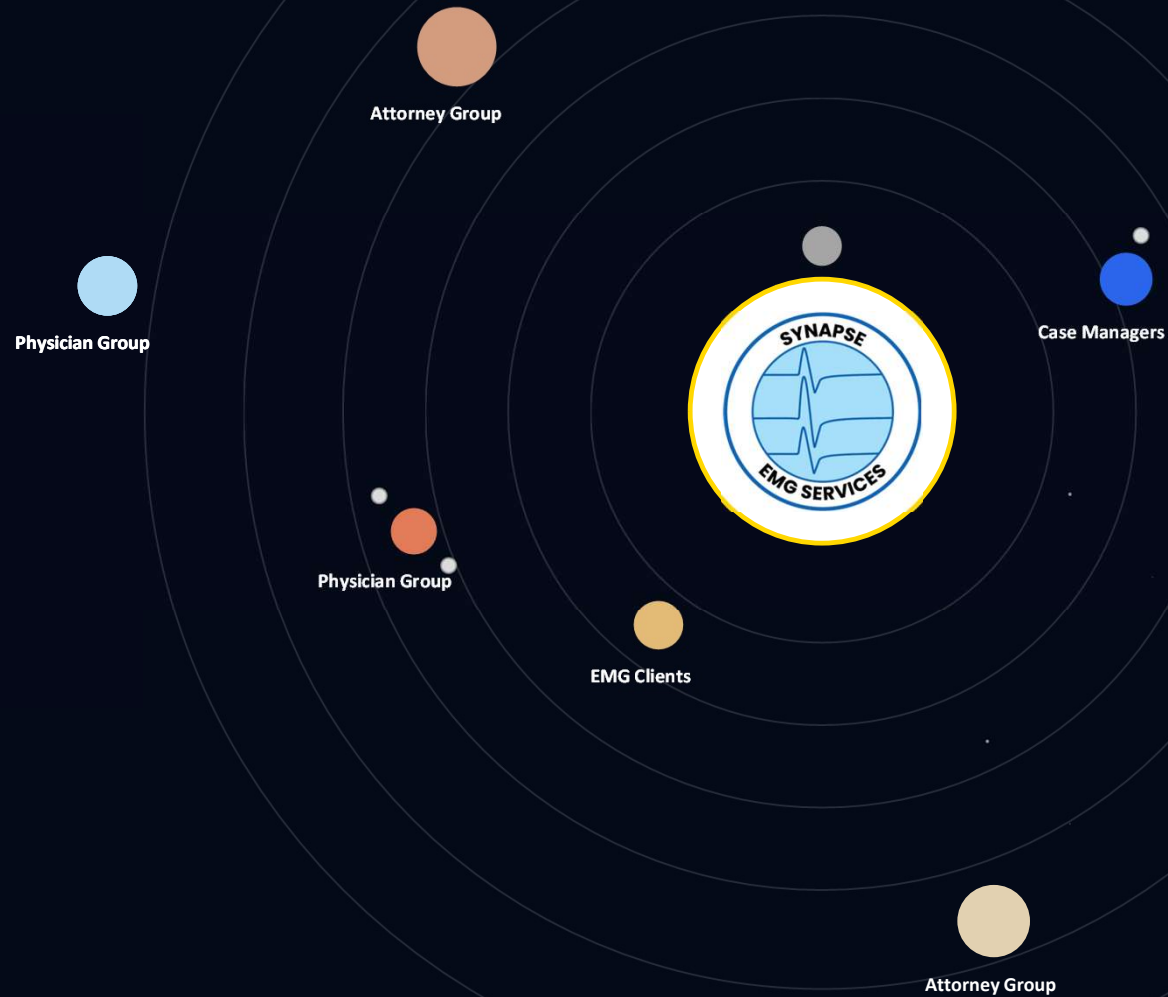
- To outline and discuss perspectives on report writing and the basic components of an EMG report.
- To place emphasis on a customer centric approach to writing EMG reports.
- To discuss with the audience strategies that have worked in practice and explore other potential strategies for success.

Disclaimer

- EMG Reporting is a high level skill where science and art meet.
- Paul Cezanne¹
 - Regarded as one of the most influential painters in history.
 - His story was one of ongoing improvement and dedication to his craft
 - Was rejected by the Salon for 40 years.
 - His later works were considered more valuable than his early works.
 - His work inspired future creatives like Picasso and Matisse.



SITTING AMONGST THE STARS



THE SYNAPSE UNIVERSE

Synapse EMG Services: The central Sun powering a diverse diagnostic ecosystem.

Perspectives on Report Writing

- If the report is our most valuable end-product, how do we refine the process to consistently create something more valuable than what exists in the market?



Perspectives on Report Writing – Dumitru²

- Once the exam is complete and all the data is analyzed, conveying the report information in a concise and understandable way is one of the most important aspects.
- The final report can take many forms. There is no one “correct” way to document the exam and findings.
- Consideration of how central lesions can present and manifest.
- History**, Physical Exam, NCS data, EMG data.
- Interesting recommendation of having an EDX impression, Clinical Impression, and Recommendations.
- If abbreviations are used, there should be a legend somewhere in the final report for benefit of physicians not familiar with terminology.
 - “Pt. c/o headaches, NT in the BUE as well as weakness in the APB, ECU and FCR > FPL.”
 - Dumitru, Chapter 14, p515-524.

Perspectives on Report Writing - AANEM

- American Association of Neuromuscular & Electrodiagnostic Medicine (AANEM) Position Statement.³
- Reporting the Results of Nerve Conduction Studies and Needle EMG.³
 - “This report serves as a communication tool to the referring physician and documents the results and conclusions of the electrodiagnostic (EDX) evaluation.”



Perspectives on report writing

- AANEM Model-Report.⁴
- Lists 11 items to include in an EMG report.
 - Patient demographics
 - Reason for referral
 - Description of History and Physical Exam
 - Temperature ($> 32^{\circ}\text{C}$ in the UE; $> 30^{\circ}\text{C}$ in the LE)
 - Tabular NCS Data
 - Reference Values
 - Tabular EMG Data
 - Description of Findings
 - Probable Diagnosis & Location of Pathology
 - Study Limitations & Previous NCS Data
 - Signature with Time Stamp

[illegible]

Interestingly the inclusion of a description on the type of needle electrode used is listed as optional and is not listed as a practice recommendation.

Perspectives on Report Writing

- How do we take all the things that we know should be in a report, and present them in a way that creates greater success?



Are we not telling people's stories?

- In every culture, the storytellers have a special place in society.
 - The Griots of West Africa,
 - Comedians,
 - Players of Instruments.
- How do create better stories that last and withstand the test of time?



Identify Purpose and Identify Our Target Audience

- Purpose
 - To help make the story clearer.
 - What do we see, what else do we see, what we don't see.
- Identify Target Audience.
 - Clients who need EMG.
 - Referring Physicians.
 - Case Managers
 - Attorneys
 - Medical Billers and Coders.



How do we Rank each?

Needs Assessment

- Clients who need EMG.
 - A positive customer experience.
 - Professionalism, Tone of Voice, Making the experience as comfortable as possible.
 - Listening, Documenting and Communicating their stories clearly and accurately.
 - Rapid scheduling and report turnaround.
- Referring Physicians.
 - Pertinent information and impression all on one page.
 - Clear, concise messaging to help guide their decision-making. 10 or 12th grade reading level.
 - Rapid scheduling and report turnaround.
- Case Managers
 - Clear, concise messaging to help guide case resolution.
 - Rapid scheduling and report turnaround.
- Attorneys
 - Most important and pertinent information all on one page. 10 or 12th grade reading level.
 - Clear, concise messaging to help guide case resolution, written in way that does not cloud the presentation or create legal doubt.
 - Stick to the facts supported by data.
 - Correct dates (Jun 2025 vs Jun 1st 2025).
 - Rapid scheduling and report turnaround.
- Medical Billers and Coders.
 - Clear, concise billing instructions.
 - ICD-10/CPT Codes that match test data recorded in the report.
 - Charges on last page.
 - Low denial rate (means less work for them).



How do we then also use the report as a marketing tool?



Marketing

Purpose

- Increase awareness of **diagnostic services**.
- Build strong relationships with **referring healthcare providers**.

Strategies

- Communicating **clear and timely reports**
- Providing **reliable diagnostic information**
- Maintaining **professional collaboration with healthcare clinics**

Impact

- Helping physicians to make **better clinical decisions**
- Improving **patient care and outcomes**
- Strengthening **trust and referral network relationships**



The Marketing Mix (4P's)

- E. Jerome McCarthy (1960's)
- The Marketing Mix (4Ps)
 - Product
 - Price
 - Place
 - Promotion

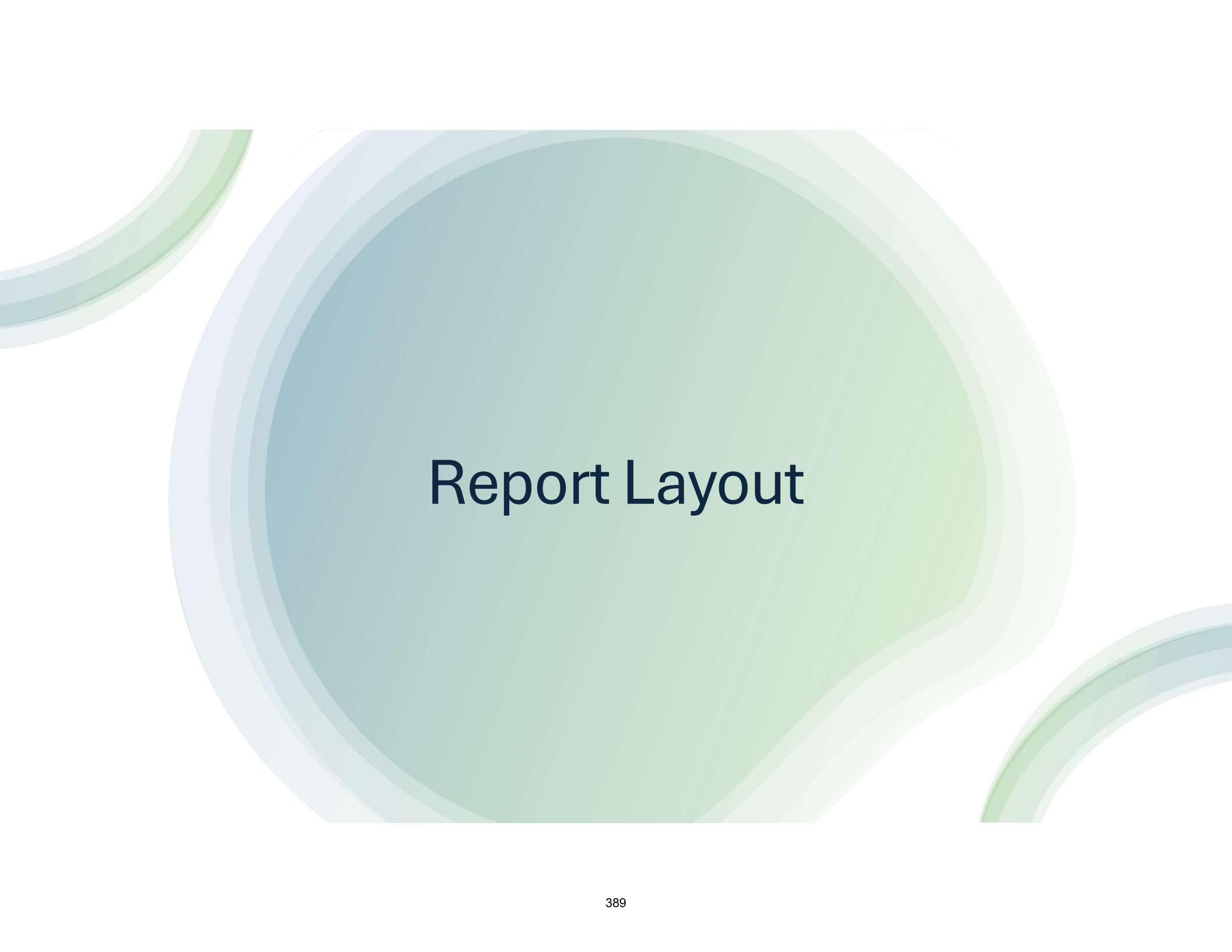


Lester Wunderman (Direct Marketing)

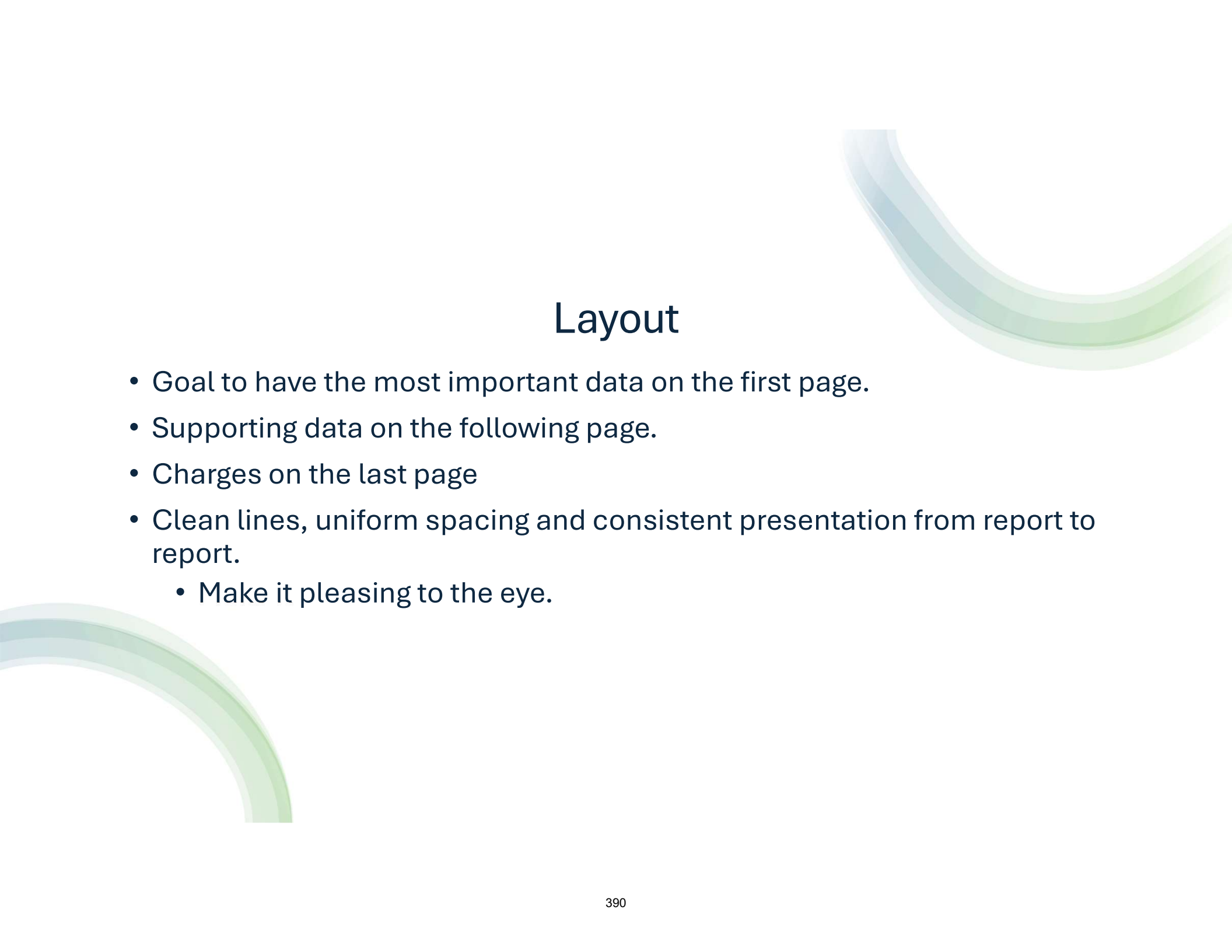
- Key Concepts
 - “The Consumer, Not the Product, Must be the Hero.”
 - “The product must create value for each of its consumers. It must satisfy consumers’ unique differences, not their commonalities.
 - “Customers have to know and feel the brand as an experience that serves their individual needs.”

Philip Kotler (Modern Marketing)

- Key Concepts:
 - Also promoted the Four P's of Marketing.
 - Marketing has never been static
 - It changes with time and technological advancement.
 - Data, Internet of things (IoT), and AI.
 - Treating customers as whole humans with minds, hearts, spirits.



Report Layout



Layout

- Goal to have the most important data on the first page.
- Supporting data on the following page.
- Charges on the last page
- Clean lines, uniform spacing and consistent presentation from report to report.
 - Make it pleasing to the eye.

Organizing Thoughts and Information

- Branding
- Demographics
- Sensory NCS summary
- Motor NCS summary
- Late Responses summary
- EMG Summary with description of type of needle electrode used.
- Impression
- Signature Line
- Supporting data (Tables, Waveforms, Normative Data) on the following pages.
- ICD 10 / CPT Codes on the last page.

Branding, Demographics, Reason for Referral



Synapse EMG Services, LLC
Tel: (678) – 753 – 4364 • Fax: (678) – 736 – 4329
Referrals@emgtesting.com

Test Date: 01/01/2026

Patient:	Aldwyn Roberts	DOB:	01/01/1955	Physician:	Dr. Leroy Calliste, MD
Sex:	Male				

Diagnosis: Carpal Tunnel Syndrome G56.02

Demographics, Reason for Referral, Hx, Physical Exam, Temperature

- Names are important
 - Names hold personal and cultural significance.
 - Correct spelling of client names and referring provider names is important.
- Credentials
 - Credentials are important as they are a marker of the journey that the person / provider has travelled.
 - They also represent that person's dedication to mastering their craft.
- Getting names right communicates attention to detail.
 - Fosters a level of trust in the report data that is to be presented.
 - What if the referring person prefers to be referred to by their middle name?
 - Dr. J. Edgar Hoover MD; Dr. O. Shane Smith, MD

Patient:	Aldwyn Roberts	DOB:	01/01/1955	Physician:	Dr. Leroy Calliste, MD
Sex:	Male				

History and Physical Exam

Complaint:

The patient complains of nocturnal paresthesia in the left hand primarily in digits I to III. There are no specific complaints of cervical pain, right upper extremity or lower extremity paresthesia. Patient denies a history of DM, thyroid, kidney or autoimmune disorders. A brief clarifying exam was performed prior to testing and sensibility to light touch was impaired at the palmar aspect of the tip of digit I. There was a (+) Carpal compression test on the (L), strength testing was grossly 5/5 in the (C5-T1) myotomes bilaterally, and Spurling's test was also (-). Temperature was maintained above 31C in the left upper extremity.

NCS Summary

ELECTRONEUROMYOGRAPHIC SUMMARY

Nerve Conduction:

Sensory NCS:

The left median nerve to digits I and III was delayed across the wrist with a low amplitude response to digit I. The left ulnar and left radial nerves were WNL.

Motor NCS:

The left median nerve was delayed across the wrist with amplitudes WNL. The left radial and left ulnar nerves were WNL.

Late Responses:

The left ulnar F-wave was WNL.

EMG Summary

Electromyography:

Using a monopolar needle electrode, sampling consisted of ventral primary rami innervated muscles of the C5-T1 myotomes in the left upper extremity. All muscles tested showed normal activity at insertion, rest and during voluntary recruitment.

Impression

- Here is what the data tells us.
- Here is what else the data tells us.
- Here is what the data does not show.
 -and BTW doc, here are some other pertinent clinical things that you may want to know about.
- 10 or 12th grade reading level.

Impression

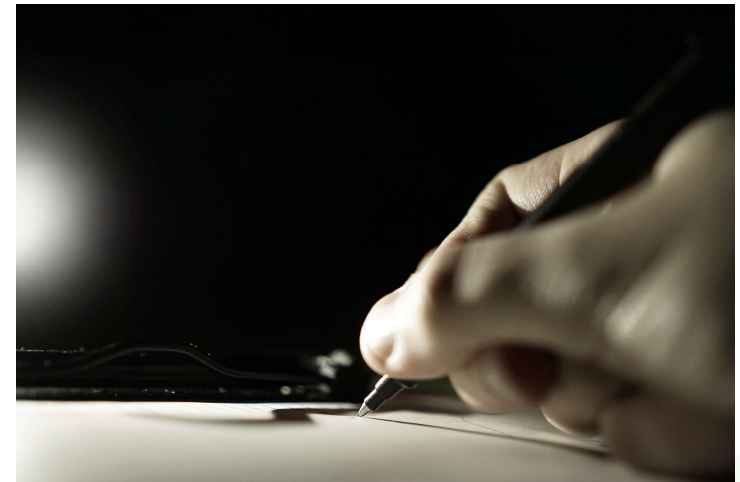
Impression: (Abnormal Study)

- 1. Evidence suggests a moderate focal mononeuropathy of the left median nerve across the wrist.**
- 2. There was no evidence to suggest a polyneuropathy, plexopathy or radiculopathy.**

Signature Line

Thank you,

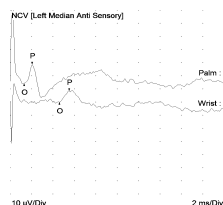
Leon C. Romero, PT, Sc.D., OCS, ECS
Sc.D. Clinical Electrophysiology.
ABPTS Board Certified Clinical Orthopedic Specialist.
ABPTS Board Certified Clinical Electrophysiology Specialist.
GA License # 8390



Tables, Waveforms, Norms

- Abnormal results clearly identifiable to the reader.
- Limitations of highlighted data

Site	NR	Onset (ms)	Norm Onset (ms)	O-P* Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Motor Summary Table												
Left Median Motor (Abd Poll Brev)												
Wrist 8cm		6.6	<4.2	5.6	>2.8	7.34	Elbow	Wrist 8cm	3.9	20.5	53	>50
Palm		1.7		6.2		7.66	Wrist 8cm	Palm	4.9	7.0	14	>29
Elbow		10.5		5.3		4.06						
Left Radial Motor (Ext Ind Prop)												
Forearm 8cm		2.3	<3.0	5.6		8.44	Brachium	Forearm 8cm	2.9	20.0	69	>50
Brachium		5.2		5.4		8.13						
Left Ulnar Motor (Abd Dig Minimi)												
Wrist 8cm		3.0	<3.6	7.8	>4.2	6.41	B Elbow	Wrist 8cm	2.3	16.0	70	>49
B Elbow		5.3		7.8		6.56	A Elbow	B Elbow	1.6	10.0	62	>47
A Elbow		6.9		7.5		6.56						



EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psy	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Left	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Abd Poll Brev	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	FlexCarRad	Median	C6-7	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	BrachioRad	Radial	C5-6	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	

ICD-10 and CPT Codes on Last Page

- Billers can get data they need electronically or can snip or print as needed.
 - No need to call to you for billing sheets or to know what to bill.

CPT	Units	Extremity	Modifier	Description
		L	R	
95886	x1	●	○	EMG Complete (5 or more muscles) each extremity; with or w/out paraspinals performed with NCS
95886		○	○	
95885		○	○	EMG Limited (<5 muscles) each extremity; with or w/out paraspinals when performed with NCS
95885		○	○	
95887		○	○	EMG non-extremity muscles (cranial nerve supplied or axial muscles), with NCS
95907				Nerve conduction studies 1-2 tests
95908				Nerve conduction studies 3-4 tests
95909	x1			Nerve conduction studies 5-6 tests

The Spirit of Reporting

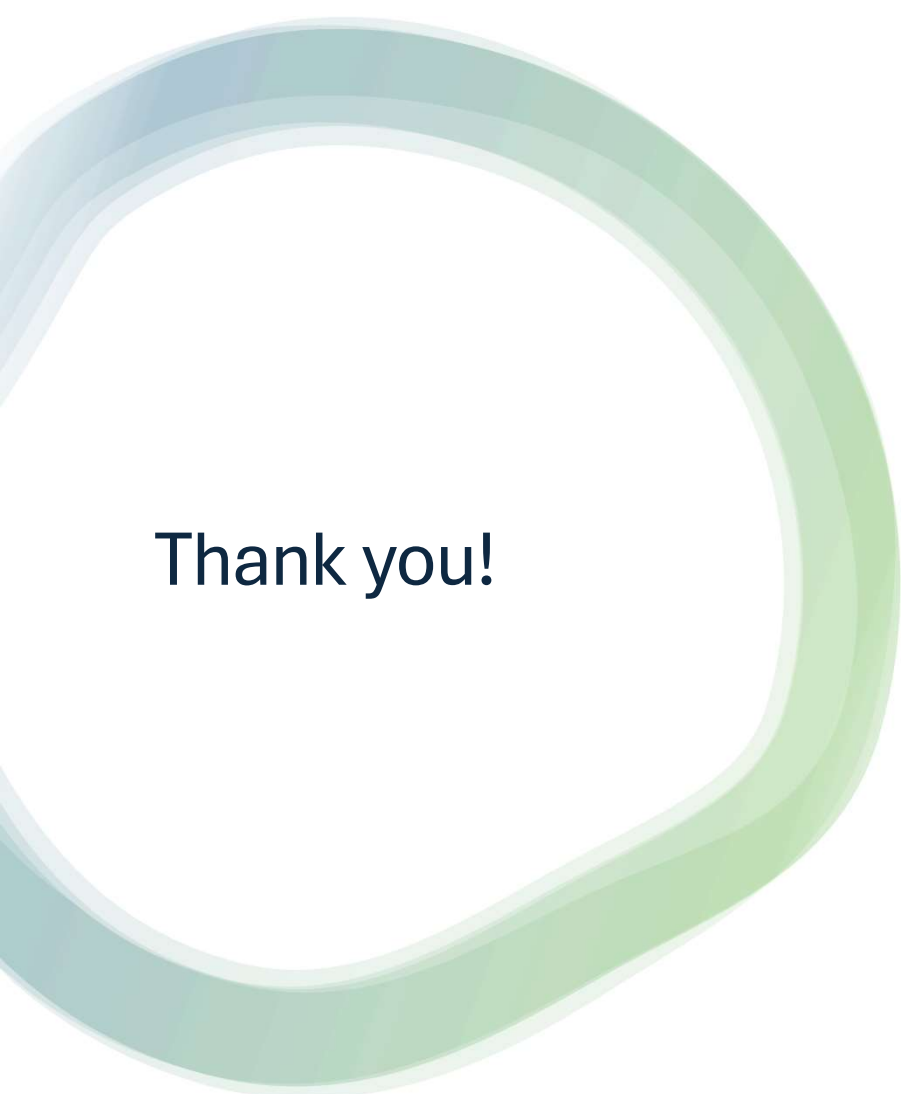
- If done well, the physical report becomes an embodiment of your contribution to the profession on a daily basis.
 - All the Anatomy, Physiology, Pathophysiology, EMG/NCS and pattern recognition knowledge.
 - All the time spent honestly mastering your craft and dedicating yourself to excellence.
 - Who you are as a specialist will manifest itself to your audience on a daily basis.
- Caveat

Indicators of Success

- Time Efficiency
 - How long does it take to complete a final report?
 - Ability to add more cases to your schedule if desired?
 - Rapid delivery of reports?
 - All reports done end of day so you can go home to your family.
- Enhanced Brand awareness
 - Organic Referrals Created.
 - Marketing to internal and external customers.
- No report of denials (or very infrequent reports of denials) from the billers.
- Finance \$\$\$
- Customer Feedback
 - “My doctor sent me for this test and said that you are a rockstar.”
 - “My doctor said to me that you are the best in the business.”
 - Physicians calling saying “I don’t know what exactly is going on, but tell me what you think.”
 - “We really appreciate your reports, they are way better than such-and-such up the street and really help us figure out what is going on?”



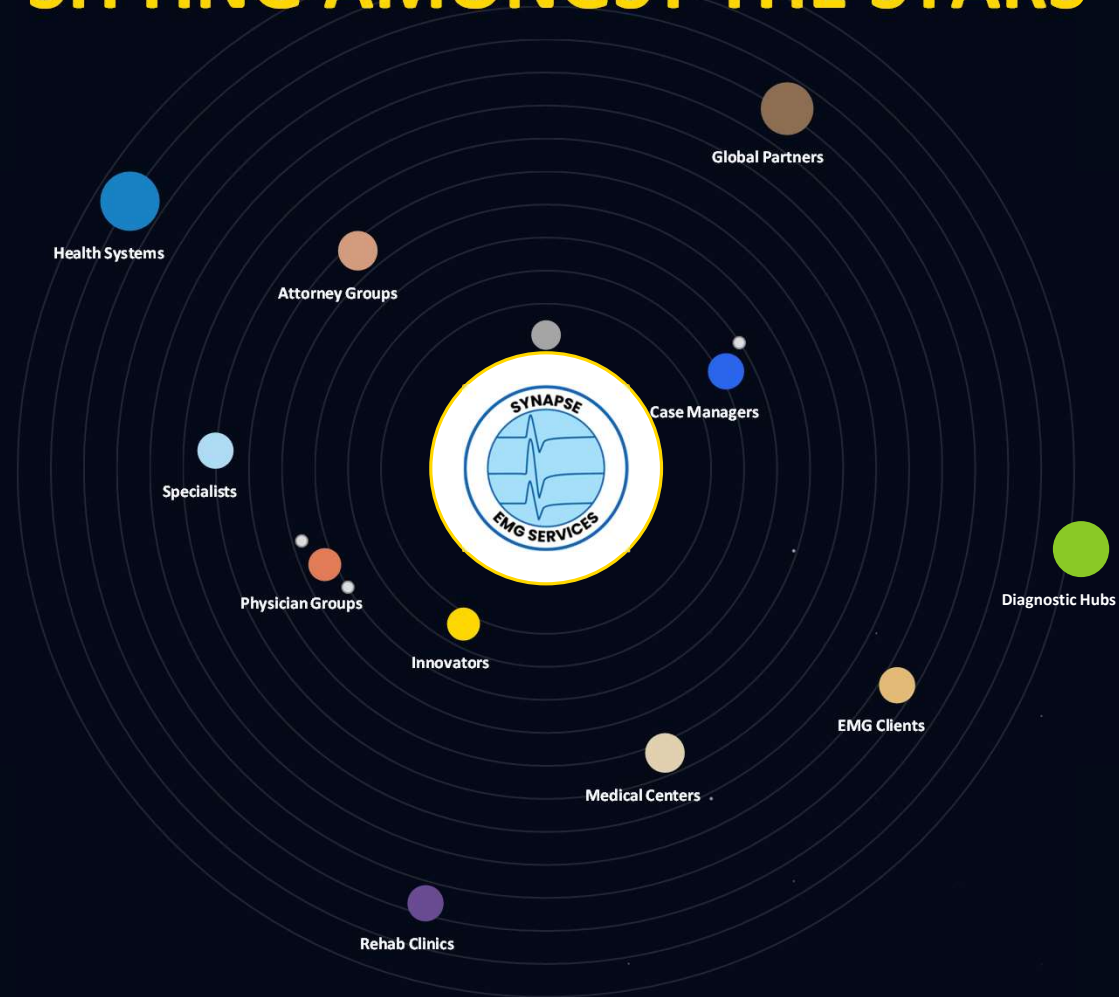
Questions / Open Discussion



Thank you!

- Best wishes and keep creating great reports!

SITTING AMONGST THE STARS



THE SYNAPSE UNIVERSE

Synapse EMG Services: The central Sun powering a diverse diagnostic ecosystem.

References

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SEVERE BRACHIAL PLEXOPATHY POST FALL AND FRACTURE DISLOCATION OF THE SHOULDER

Oh T

ABSTRACT

BACKGROUND: Falling is a major problem for seniors > 65 with 1 in 4 falling every year. Of those, 37% suffers an injury that requires medical attention¹. Though not one of the more common injuries suffered, a fracture/dislocation of the shoulder can be a devastating injury with nerve injuries occurring in 21%–50% of cases². This case presents on a 75 year old referred for EMG/NCS testing with suspected radial and ulnar nerve palsy status post fall and fracture dislocation of the right shoulder.

DISCUSSION: The patient presented 4 weeks post fall resulting in a fracture/dislocation of the right shoulder. No medical records were provided so the specifics of the injury were not known, though the patient's son stated the fracture was minor. The patient had a history of CVA but had full use of the right UE prior with no other significant past medical history. The clinical exam showed diminished sensation to light touch throughout the whole right UE with minimal ability to use the right UE actively, though he had active elbow and wrist/finger flexion which were graded at 3+/5. UE muscle stretch reflexes were 0 but symmetrical bilaterally and Special testing (Tinel's at the wrist and elbow and Spurling's) were negative. The clinical picture was complicated due to the fracture dislocation of the shoulder affecting the myotome screen. Differential diagnosis was peripheral nerve pathology vs brachial plexopathy especially the lower trunk. NCS/EMG testing was extensive and showed a severe injury to the brachial plexus with an impression of "A severe, acute axonal pathology affecting mostly the entire right brachial plexus (Pan plexus)."

CONCLUSION: Nerve injuries post shoulder dislocation may occur more commonly than we think with an incidence of 39.8% to 65.5%⁴ when EMG testing was performed. Though brachial plexus injuries are commonly thought to be most common amongst young adults post high energy injuries such motorbike accidents, a simple fall may be one of the more common causes (43–67%³). Of these, Pan Plexus injuries are more common when accompanied with fracture of the greater tuberosity and has been found to occur as high as 41.7% of these nerve injuries³. Clinicians should be aware of the high level of shoulder dislocations amongst seniors with the possibility of complicated nerve injury especially when associated with a fracture.

Ting Oh, PT, ECS

References:

1. Kakara R, Bergen G, Burns E, Stevens M. Nonfatal and Fatal Falls Among Adults Aged ≥ 65 Years—United States, 2020–2021. *MMWR Morb Mortal Wkly Rep* 2023;72:938–943.
2. Praselia R, Handoko HK, Rosa WY, Ismiarto AF, Petrasama, Utoyo GA. Primary traumatic shoulder dislocation associated with rotator cuff tear in the elderly. *Int J Surg Case Rep*. 2022 Jun;95:107200
3. Gutkowska O, Martynkiewicz J, Urban M, Gosk J. Brachial plexus injury after shoulder dislocation: a literature review. *Neurosurg Rev*. 2020 Apr;43(2):407–423
4. Lorente A, Mariscal G, Barrios C, Lorente R. Nerve Injuries after Glenohumeral Dislocation, a Systematic Review of Incidence and Risk Factors. *J Clin Med*. 2023 Jul 7;12(13):4546

Questions:

1. Which of the following is most likely to be normal in an infraclavicular brachial plexus lesion:
 - a. Lateral antebrachial cutaneous nerve
 - b. Suprascapular nerve (Correct)**
 - c. Axillary nerve
 - d. Radial nerve
2. Which injury when accompanying a shoulder dislocation has been found to be associated with a Pan Plexus injury?
 - a. Rotator cuff tear
 - b. Greater tuberosity fracture (Correct)**
 - c. Cervical strain/sprain
 - d. Subacromial bursitis

Severe Brachial Plexopathy Post Fall and Fracture Dislocation of the Shoulder

Ting Oh, PT, ECS



Rocky Mountain
University
of Health Professions

Case Presentation

Friday, September 19, 2025

79°C and sunny Fall day

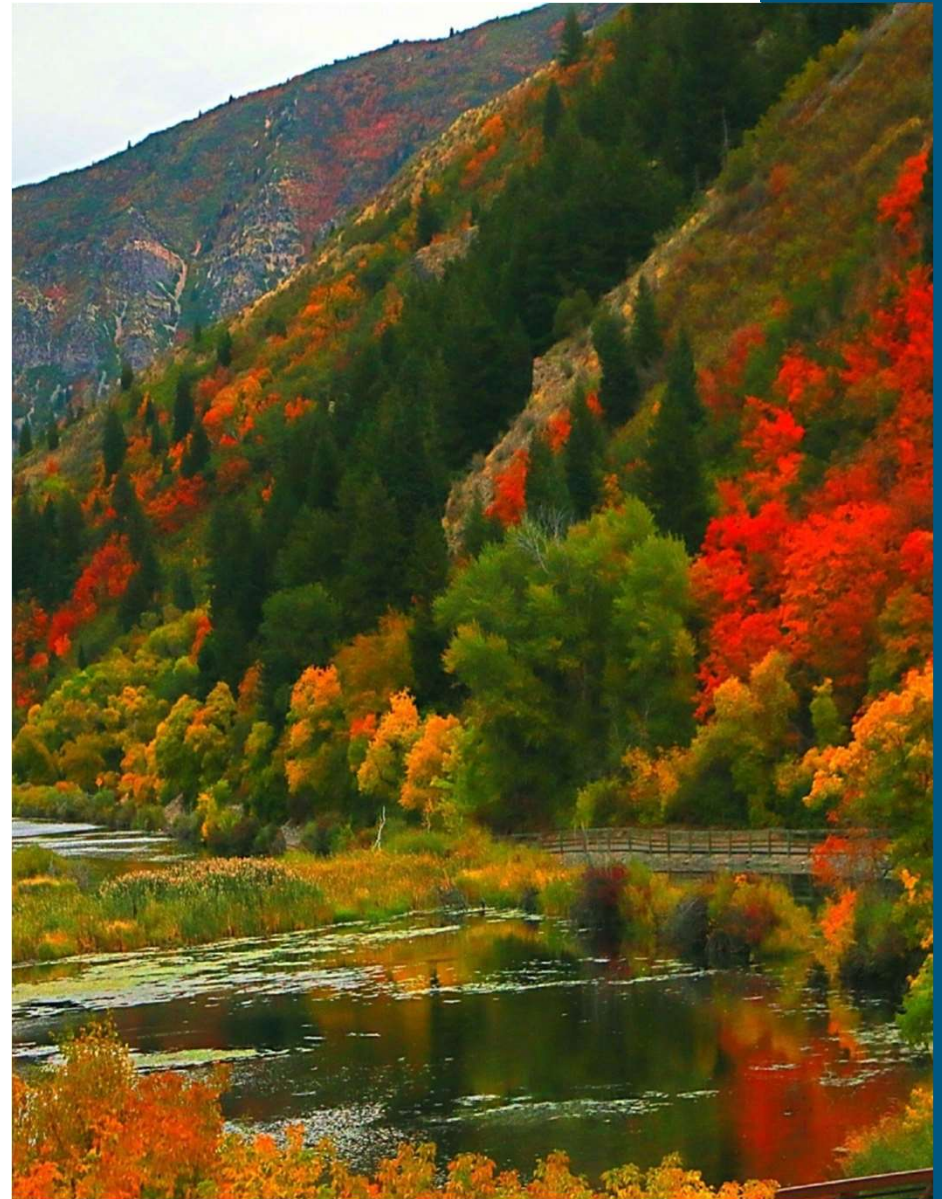
Morning went smooth

Afternoon schedule

1:20 EMG (1hr)

2 IE

Beautiful Day in NEPA



Order Date: Sep 3, 2025

EMG/NCS/US (NEU5) (Order ID: 1600796970)

Expiration Date: Sep 3, 2026 **Count:** 1/1

Diagnosis: Ulnar nerve palsy of right upper extremity (G56.21)

Radial nerve palsy, right (G56.31)

Priority: Routine

Note that neuromuscular ultrasound will be performed at the discretion of the performing neurologist unless otherwise specified.
Ultrasound is available at the Cedar Crest site only.

Reason for Exam: Other (specify in comments) (Evaluate for ulnar and radial nerve palsy)

Is the patient on an anticoagulant? Yes

Extremity (Maximum 2): Right upper extremity

Signature:



Electronically signed by: Marcos M. Martinez, MD

Order entered by: Jaclynn Matusiewicz

Authorized by: Marcos M. Martinez, MD (NPI: 1396052130)

To assist in the coordination and continued quality of your medical care, we encourage you to use a Lehigh Valley Health Network facility for the provision of the health services recommended above. Please call 888-402-LVHN to schedule your appointment.



Radial and Ulnar Palsy

Isolated mononeuropathies?

- Saturday Night variation
- Crutch Palsy
- Trauma – humerus fracture

Brachial Plexus?

- Lower trunk?
 - Radial Palsy likely upper and middle trunk
 - Median not mentioned
- Medial and Posterior Cords

Cervical?

- C7-T1
- Not convinced



Clinical Presentation

- Late arrival
- Unhealthy looking 70 y.o. male in a wheelchair
- 5'9", 145lbs
- Spanish speaker
 - Son is able to interpret
- Right hand swollen, in a cock up splint



Subjective exam

Chief complaint:

He complains of being unable to move his right hand which is also swollen. He can bend his arm a little at the elbow. The right arm also is very painful. The problem started around 1 month ago after a fall resulting in a fracture and dislocation of the right shoulder. He has a history of a stroke but his son reports he had normal use of his right hand prior to the accident. He denies left sided symptoms.

Past Medical History:

He denies history of diabetes, thyroid and kidney disease. There is no family history of neuromuscular disease and no previous exposure to toxins.



Physical exam

Observation:

Observation shows edema in the right wrist/hand with general muscle atrophy in the right UE.

Sensation Testing:

Sensation testing is diminished to light touch throughout the right UE.

Muscle Stretch Reflexes:

Muscle stretch reflexes were 0 in bilateral biceps, triceps and brachioradialis.

Manual Muscle Testing:

Manual muscle testing revealed minimal voluntary use of the right UE. He has some active motion in elbow flexion and wrist/finger flexion.

Provocative Testing:

Tinel's test in the carpal and cubital tunnel: Negative

Spurling's test: Negative

Pain:

Patient reports pain complaints up to 7/10.



Differential Dx

- 1 - Ulnar/Radial neuropathy likely at the shoulder/upper arm
- 2 - Brachial plexopathy trunk or cord levels. (Unsure of proximal weakness)

NCS Plan:

Sensories:

Radial – posterior cord

Ulnar D5 – lower trunk, medial cord

Median D2 – upper trunk, lateral cord

LABC? – upper trunk, lateral cord

MABC? - lower trunk, medial cord

Motor:

Radial - lower trunk, posterior cord

Ulnar – lower trunk, medial cord

Median - lower trunk ,medial cord

Axillary, Musculo, suprascap – didn't really want to do it

EMG Plan:

Will be doing an extensive screen including paraspinals

Key will be APB and potentially Upper trunk, Lateral cord



Sensory NCS

Stim Site	NR	Peak (ms)	Norm Peak (ms)	O-P Amp (μV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median D2 Antidromic Transcarpal Sensory (2nd Digit)											
Wrist		3.7	<4.0	17.4	>11.0	Wrist	2nd Digit	3.7	14.0	38	>39
Right Median D2 Antidromic Transcarpal Sensory (2nd Digit)											
Wrist		3.4	<4.0	7.9	>8.0	Wrist	2nd Digit	3.4	14.0	41	>39
Palm		1.8	<2.3	6.6	>6.0	Palm	2nd Digit	1.8	7.0	39	>32
						Wrist	Palm	1.6	7.0	44	>39.0
Left Radial Sensory (1st Digit)											
Wrist		2.5	<2.8	20.5	>7.0	Wrist	1st Digit	2.5	10.0	40	>36
Right Radial Sensory (1st Digit)											
Wrist		2.3	<2.8	4.9	>7.0	Wrist	1st Digit	2.3	10.0	43	>36
Left Ulnar D5 Sensory (5th Digit)											
Wrist		3.5	<4.0	6.8	>5.0	Wrist	5th Digit	3.5	14.0	40	>35
Right Ulnar D5 Sensory (5th Digit)											
Wrist	NR		<4.0		>5.0	Wrist	5th Digit		14.0		>35



Sensory NCS - Brachial plexus

Trunks:

Upper trunk – LABC 96% – Median – thumb 96% – Superficial radial 58% – Median – (index) 20% – Median (middle) 10%

Middle Trunk – Median to middle 100%

Lower trunk – Ulnar – small 96% – DUC 96% – MABC 65%

Cords:

Lateral cord – LABC 100% – Median – thumb 100% – Median – index 100% – Median – middle (80%)

Posterior cord – Superficial radial 100%

Medial cord – Ulnar – small 100% – DUC 100% – MABC 100% – (Median – middle 20%)

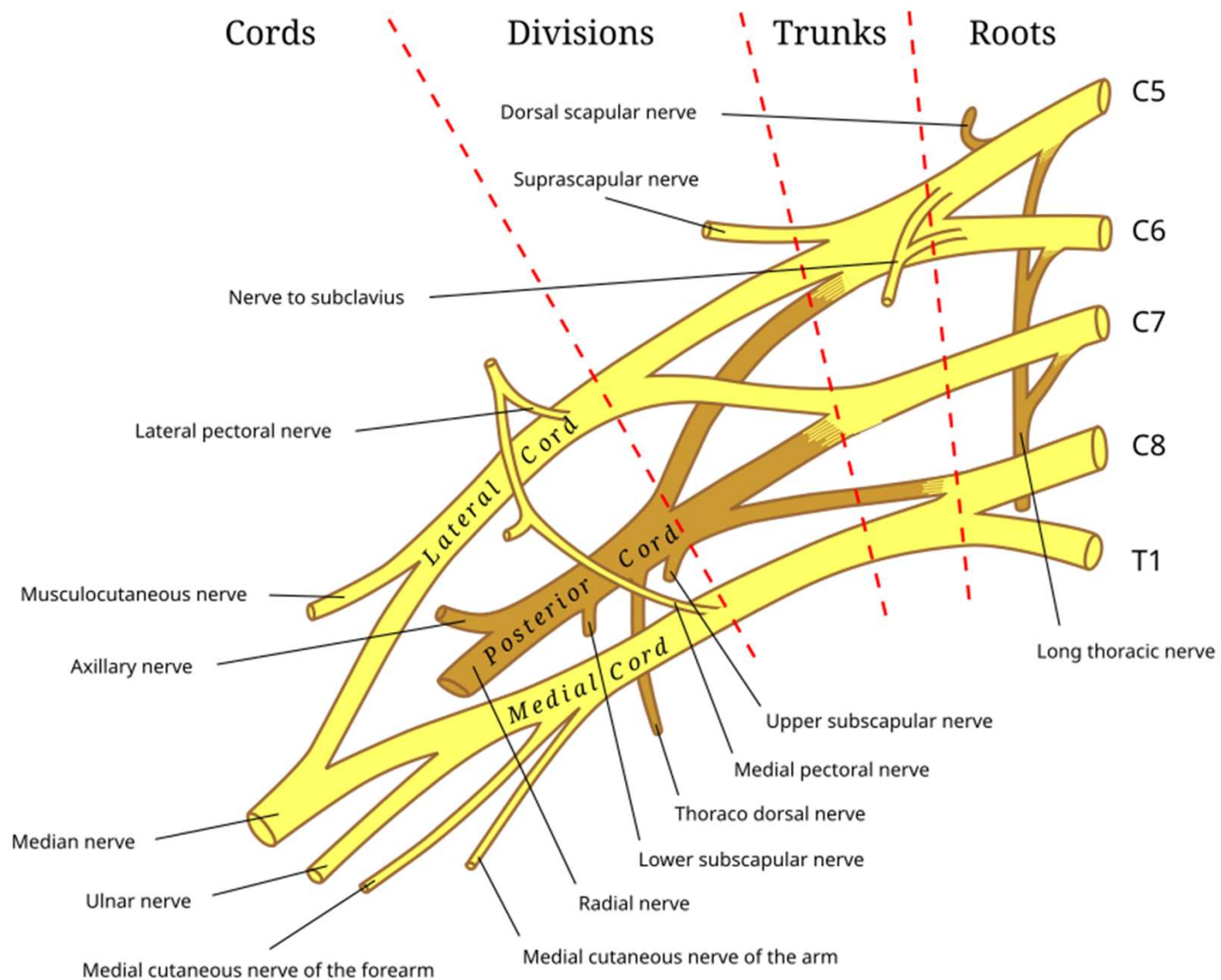


Motor NCS

Stim Site	NR	On-set (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Full Dur (ms)	Neg Area (mVms)	Site1	Site2	Delt a-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Motor (Abd Poll Brev)														
Wrist		3.3	<4.5	8.4	>4.1	6.09	19.22	28.06	Wrist	Abd Poll Brev	3.3	8.0		
Elbow		7.7		7.6		6.72	17.34	25.78	Elbow	Wrist	4.4	24.0	55	>49.0
Right Median Motor (Abd Poll Brev)														
Wrist		3.6	<4.5	1.7	>4.1	6.72	14.69	6.66	Wrist	Abd Poll Brev	3.6	8.0		
Elbow		8.3		1.3		7.19	15.31	4.90	Elbow	Wrist	4.7	24.0	51	>49.0
Left Radial Motor (Ext Ind Prop)														
Forearm		2.2	<3.4	3.9	>1.0	10.00	18.44	24.40	Forearm	Ext Ind Prop	2.2	8.0		
A Elbow		3.9		3.7		10.47	19.22	23.34	A Elbow	Forearm	1.7	11.0	65	>50
A Sp Groove		5.6		3.6		10.78	19.53	22.14	A Sp Groove	A Elbow	1.7	10.0	59	>50
Right Radial Motor (Ext Ind Prop)														
Forearm	NR		<3.4		>1.0				Forearm	Ext Ind Prop		8.0		
A Elbow	NR								A Elbow	Forearm		11.0		>50
A Sp Groove	NR								A Sp Groove	A Elbow		10.0		>50
Left Ulnar ADM Motor (Abd Dig Minimi)														
Wrist		3.0	<3.7	10.5	>4.9	5.78	19.38	29.34	Wrist	Abd Dig Minimi	3.0	8.0		
B Elbow		7.0		9.3		5.94	22.50	27.60	B Elbow	Wrist	4.0	22.0	55	>52.0
A Elbow		8.6		9.1		6.41	22.97	27.56	A Elbow	B Elbow	1.6	8.0	50	>43.0
Right Ulnar ADM Motor (Abd Dig Minimi) 21.4 °C														
Wrist	NR		<3.7		>4.9	3.59	9.84	0.82	Wrist	Abd Dig Minimi		8.0		
B Elbow	NR								B Elbow	Wrist		22.0		>52.0
A Elbow	NR								A Elbow	B Elbow		8.0		>43.0



Motor NCS



(Wikipedia - https://en.wikipedia.org/wiki/Brachial_plexus)



Motor NCS Cont.

Stim Site	NR	On-set (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Full Dur (ms)	Neg Area (mVms)	Site1	Site2	Delt a-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Axillary and Musc. Motor (Deltoid and Biceps)														
Erb's - Axillary	NR		<5.4		>4.6									
Erb's - Musc.		5.2	<5.6	3.8	>4	15.16	36.09	38.37						
Left Suprascap - Infra Motor (Infraspinatus)														
Erb's Pt		3.6	<4.8	2.9	>1.5	10.47	26.88	17.64						
Right Suprascap - Infra Motor (Infraspinatus)														
Erb's Pt		3.0	<4.8	0.6	>1.5	7.19	15.31	3.19						



EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Fasc	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	Deltoid	Axillary	C5-6	Inc	2+	2+	0	Unable to assess					
Right	Biceps	Musculocut	C5-6	Nml	0	0	0	Nml	Nml	Nml	Nml	Complete	
Right	Triceps	Radial	C6-7-8	Inc	1+	1+	0	Unable to assess					
Right	FlexCarRad	Median	C6-7	Inc	1+	1+	0						
Right	FlexDigProf	Ulnar	C8,T1	Inc	2+	2+	0						
Right	1stDorInt	Ulnar	C8-T1	Inc	2+	2+	0						
Right	Abd Poll Brev	Median	C8-T1	Inc	1+	1+	0						
Right	CervParasp Middle	Rami		Nml	0	0	0						
Right	CervParasp Lower	Rami		Nml	0	0	0						
Right	ExtIndicis	Radial (Post Int)	C7-8	Inc	2+	2+	0	Unable to assess					
Right	Infraspinatus	SupraScap	C5-6	Inc	2+	2+	0						
Left	1stDorInt	Ulnar	C8-T1	Nml	0	0	0	Nml	Nml	Nml	Nml	Complete	
Left	Deltoid	Axillary	C5-6	Nml	0	0	0	Nml	Nml	Nml	Nml	Complete	



Summary of Findings

NCS Findings:

- The right ulnar D5, bilateral lateral antebrachial cutaneous and bilateral medial antebrachial cutaneous nerves showed no response.
- The right median D2 and right radial nerves showed reduced amplitude.
- The right Axillary motor, right radial motor and right Ulnar motor nerves showed no response.
- The right median motor and suprascapula motor nerves showed reduced amplitude.
- All remaining nerves tested exhibited normal latencies, amplitudes and conduction velocities.

EMG Findings:

- Needle evaluation of the right deltoid, right triceps, right flexor digitorum profundus, right flexor carpi ulnaris, right first dorsal interosseous, right extensor indicis, right abductor pollicis brevis and right infraspinatus muscles showed increased insertional activity and increased spontaneous activity. Motor unit morphology was unable to be assessed due to inability to voluntarily contract the muscles.
- All remaining muscles examined exhibited normal insertional activity, electrical silence at rest and normal motor unit configuration and recruitment.



What is going on?

Signs of severe widespread axonal pathology affecting the motor > sensory nerves of the majority of the right brachial plexus.

- 1 – Why the extensive damage from a simple fall? Could something else be going on? PTS
- 2 – Why such extensive motor involvement and sensories were not as bad?
- 3 – What did the musculocutaneous nerve do to deserve to be spared?
- 4 – Who the heck would scheduled this test on a Friday afternoon?

Phone a Friend

The PA Coalition

Similar experience level with similar questions

“You nerds are going to crash my phone with all this EMG talk.”



Could this be something else?

Parsonage Turner Syndrome:

- Immune response post immunization, viral illness mostly – but can be triggered by Trauma.
- “Mechanical stretching and compression of nerves may disrupt the blood-nerve barrier and allow the immune system access to peripheral nerve antigens”
- Involves mostly motor nerves but sensory is often involved (78% has sensory involvement).

What is the most likely explanation?



Falls in Seniors

- 1 in 4 seniors fall every year
- 37% suffers an injury that requires medical attention
 - Most often hip fractures, concussion, wrist fractures etc
- Fracture/dislocation of the shoulder can be a devastating injury with nerve injuries occurring in 21%–50% of cases



Sh. Dislocation and BPI

- Highest cause of shoulder disloc. and BPI is from a simple fall (43-67%)
 - Higher than high energy trauma (36-43%)
- Arm positioning affects the area involved.
 - Elbow and wrist extended – Medial cord
 - Elbow flexed – med and posterior cord
 - All the cords likely to be involved if the fall is on an abducted and fully extended shoulder.
- Total plexus is involved as high as 41% though a recent systematic review found 9%.
 - More likely to have complex nerve damage with older age, female sex and low-energy fall
 - This was more likely when accompanied by a GTF “Terrible Triad”



Mechanism of Injury

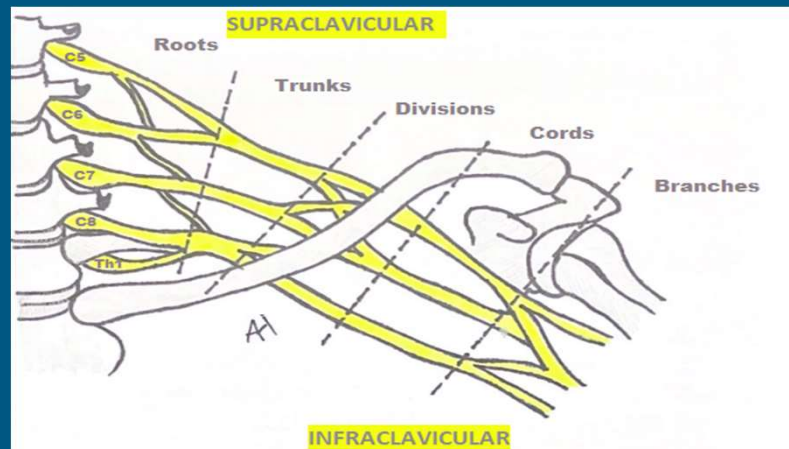
- Most likely mechanism of injury is due to traction of the BP
 - Compression by humeral head
 - Vascular
 - Hematoma
- Traction injures are more likely to affect the motor nerves
 - Larger myelinated fibers are less tolerant to stretch

Fiber Type	Likely Functional Threshold	Mechanism
Motor-dominant (large myelinated)	~5–8% strain	Conduction block, myelin distortion
Mixed nerve overall	~6–12% strain	Progressive conduction loss
Small sensory fibers	Probably >8–12%	Greater mechanical tolerance (inferred)



Common area affected

- Most common nerves affected:
 - Axillary, Ulnar, Median, Radial and Musculocutaneous nerve
- Conflicting evidence of the frequency of the musculocutaneous nerve being involved.
 - The above nerve order is from the 2 largest studies
 - Other list it to be 2nd most likely
- Mostly commonly infraclavicular affecting the posterior cord.
 - This case appears to be supraclavicular as the suprascapula nerve was involved – supports traction injury



Impression

This is an abnormal study.

There is electrophysiological evidence suggestive of:

- 1. A severe, acute axonal pathology affecting mostly the entire right brachial plexus (Pan plexus). Advanced imaging to further assess the injury is recommended.**
- 2. There is no electrophysiological evidence suggestive of a radiculopathic, polyneuropathic or myopathic process**



Clinical Insights

- Seniors are more susceptible to nerve injuries
 - Less muscular protection
- A simple fall can cause a major insult to the nervous system including the brachial plexus post shoulder dislocation
 - More likely than high velocity injury
- Don't assume weakness post shoulder dislocation especially when accompanied by RTC tear or GTF is due to orthopedic reasons only
 - Inability to lift the arm from the side may be more than the rotator cuff



CHARCOT-MARIE TOOTH: CAN IT BE TREATED? CASE STUDY AND EVIDENCE BASED SUMMARY OF CURRENT TREATMENT

Mansell N

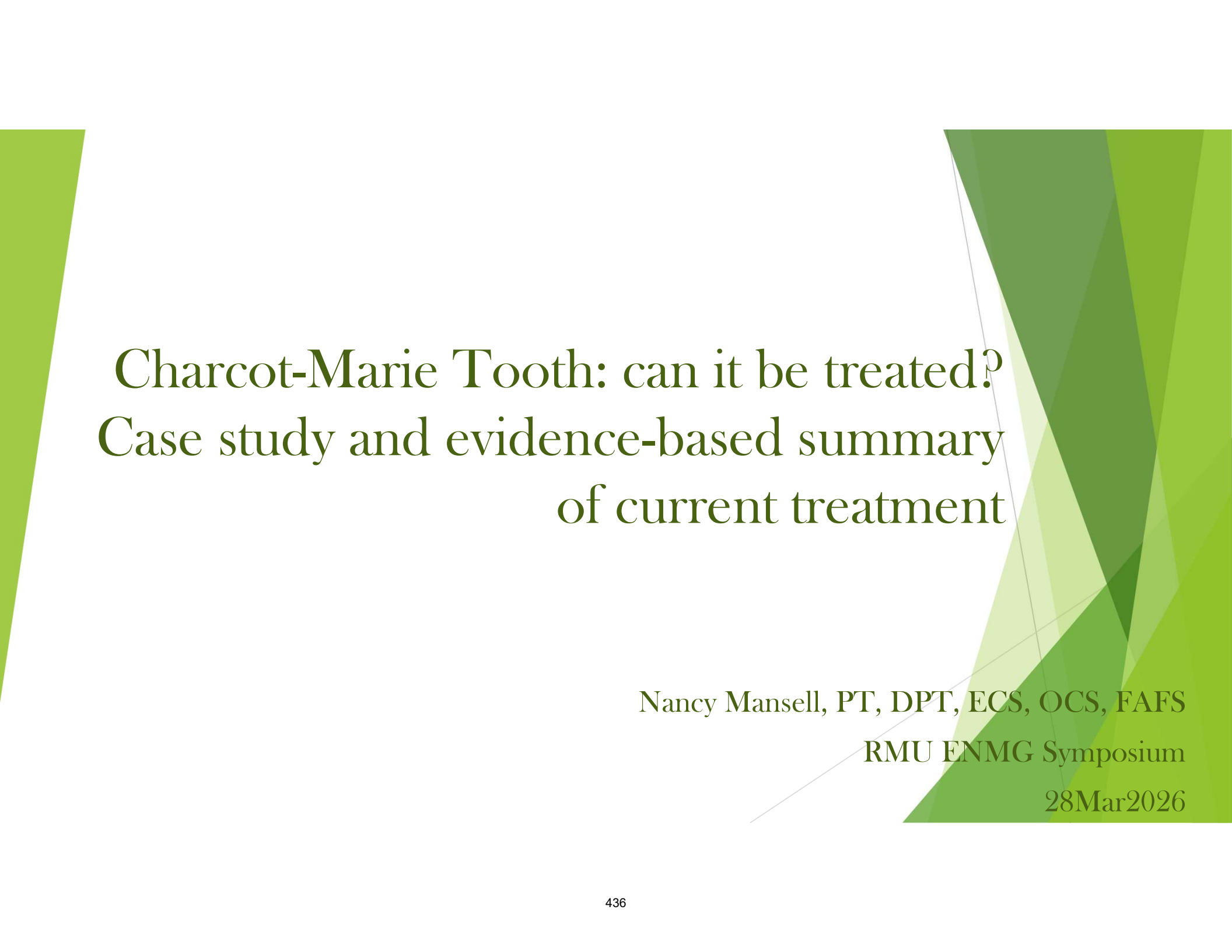
ABSTRACT

BACKGROUND: Charcot-Marie Tooth (CMT), also known as hereditary motor and sensory neuropathy, is a genetically heterogeneous group of hereditary peripheral nerve diseases, mainly affecting peripheral axons and Schwann cells. Due to the genetic diversity of CMT and its many subtypes, the current treatment is limited.

DISCUSSION: CMT1A is the most prevalent form of Charcot–Marie–Tooth disease, accounting for approximately 50% of all diagnosed cases and is attributed to mutations in the PMP22 gene. An EDX case of CMT1 will be presented and neurologist diagnostic findings will be discussed. While treatment options are limited for CMT, current management and future research will be explored.

CONCLUSIONS: CMT, a disease that is identified with EDX and genetic testing, has limited treatment options due to lack of biomarkers and the genetic variability of the disease. Despite these difficulties, researchers are optimistic about the future advancement of pharmacological treatment to improve quality of life for CMT patients.

Nancy S. Mansell, PT, DPT, ECS, OCS

The background of the slide features abstract, overlapping green geometric shapes, primarily triangles and polygons, in various shades of green, creating a modern and professional aesthetic.

Charcot-Marie Tooth: can it be treated?

Case study and evidence-based summary of current treatment

Nancy Mansell, PT, DPT, ECS, OCS, FAFS

RMU ENMG Symposium

28Mar2026

Outline

- ▶ Background Charcot-Marie Tooth Disease (CMT)
- ▶ Current Treatment
 - ▶ Drugs
 - ▶ Surgery
 - ▶ Rehabilitation
- ▶ Case study
 - ▶ EDX
 - ▶ Neurology consultation

Background CMT

► Charcot-Marie Tooth (CMT)

- Estimated prevalence of 1:2500 individuals.
- Also known as hereditary motor and sensory neuropathy
- Mainly affecting peripheral axons and Schwann cells
- Length-dependent slowly progressive muscular atrophy, starting in the feet (presenting as pes cavus or hammertoes) and legs and moving to the UE
- Affected individuals usually become symptomatic between ages 5 and 25 years, while fewer than 5% become wheelchair dependent and lifespan is not significantly shortened

Background CMT

- ▶ Genetically heterogeneous group of hereditary peripheral nerve diseases
 - ▶ CMT1A is the most prevalent form CMT (50% of all diagnosed cases)
 - ▶ Attributed to mutations in the PMP22 gene
 - ▶ The overexpression of PMP22 forms protein aggregates and correlates with reduced proteasome activity and the accumulation of detergent-insoluble ubiquitinated substrates, which induce apoptosis
 - ▶ CMT1B is the second most common type of CMT1
 - ▶ Resulting from mutations in MPZ gene, leading to ER retention, activation of the unfolded protein response (UPR), and disruption of myelin compaction
 - ▶ CMTX is an X-linked inherited CMT and the third most common form

Drug treatment

- ▶ 1. Upstream pathophysiological pathways
 - ▶ Given the role of PMP22 dosing in the pathogenesis of CMT1A, treatment efforts have focused on reducing PMP22 expression
 - ▶ Pmp22 transcription is a cAMP-induced action, as two binding sites for cAMP response element binding proteins (CREBs) reside in the Pmp22 promoter
 - ▶ By modulating the activity of signaling proteins acting upstream of the Pmp22 gene, ascorbic acid, a specific antioxidant, had an inhibitory effect on adenylate cyclase activity and reduced the production of intracellular cAMP, improving motor function in a CMT1A rat model, but its clinical efficacy is not obvious

Drug treatment

► 2. Downstream signaling pathways

- **PI3K-AKT/MEK-ERK**, two opposing pathways regulating Schwann cell differentiation, have been reported as promising therapeutic sites, for example, **PXT-3003**, in the pathomechanism of the disease
- In CMT, **PMP22** overexpression directly interferes with the **PI3K-AKT-mTOR** signaling pathway
 - Subsequently, **PI3K-AKT** leads to decreased activity of **MEK-ERK**
- **PXT-3003** is a combination of baclofen (activated on **GABA** receptors), naltrexone (an agonist of opioid receptors), and sorbitol (a chaperone-binding muscarinic acetylcholine receptor)
 - Activation of the above-mentioned receptors inhibits adenylyl cyclase conversion of **ATP** into **cAMP**, which decreases **PKA** phosphorylating activity and thus ameliorates the **PMP22** expression mediated through **CREBs**
 - **cAMP** maintains a balance between other signaling pathways such as **PI3K-AKT** and **MEK-ERK**

Drug treatment

▶ 3. Neuregulins (NRGs)

- ▶ Are a family of growth factors encoded by four genes (NRG1–4), all of which act through ErbB tyrosine kinase receptors and contain EGF-like domains that are essential for activity
- ▶ NRG1 is by far the most extensively characterized.
 - ▶ Schwann cell-derived NRG-1 type I (NRG1-I) acts via an ErbB2-receptor–MEK/ERK signaling axis and causes onion bulb formations in CMT1A
- ▶ Blockage of the ErbB2 receptor in adult CMT1A patients may prevent aberrant NRG1 signaling and constitutes a promising avenue for future therapies

Drug treatment

► 4. Changes in the gut microbiome

- Play a role in the development of numerous neurodegenerative disorders through bacterial-related metabolites or inflammatory signals via the gut-brain axis
- Interventions targeting the gut microbiome have demonstrated promising results in mitigating disease progression, highlighting their therapeutic potential
- Mitochondrial dysfunction is the most common mechanism of many types of CMT
 - Microbiome-derived metabolites such as nicotinamide, colonic acid, and methyl metabolites may regulate the processes of CMT by modulating mitochondrial activity and thereby influencing myelin sheath integrity and axonal transport
- For the CNS phenotype of CMT-X, the ongoing inflammatory response and abundant reactive oxygen species (ROS) synthesis may be the underlying mechanism
 - Microbial metabolites such as short-chain fatty acids and polyamines found within the gut can influence the immune response and have an impact on the CNS inflammatory state

Drug treatment

► 5. Gene therapies

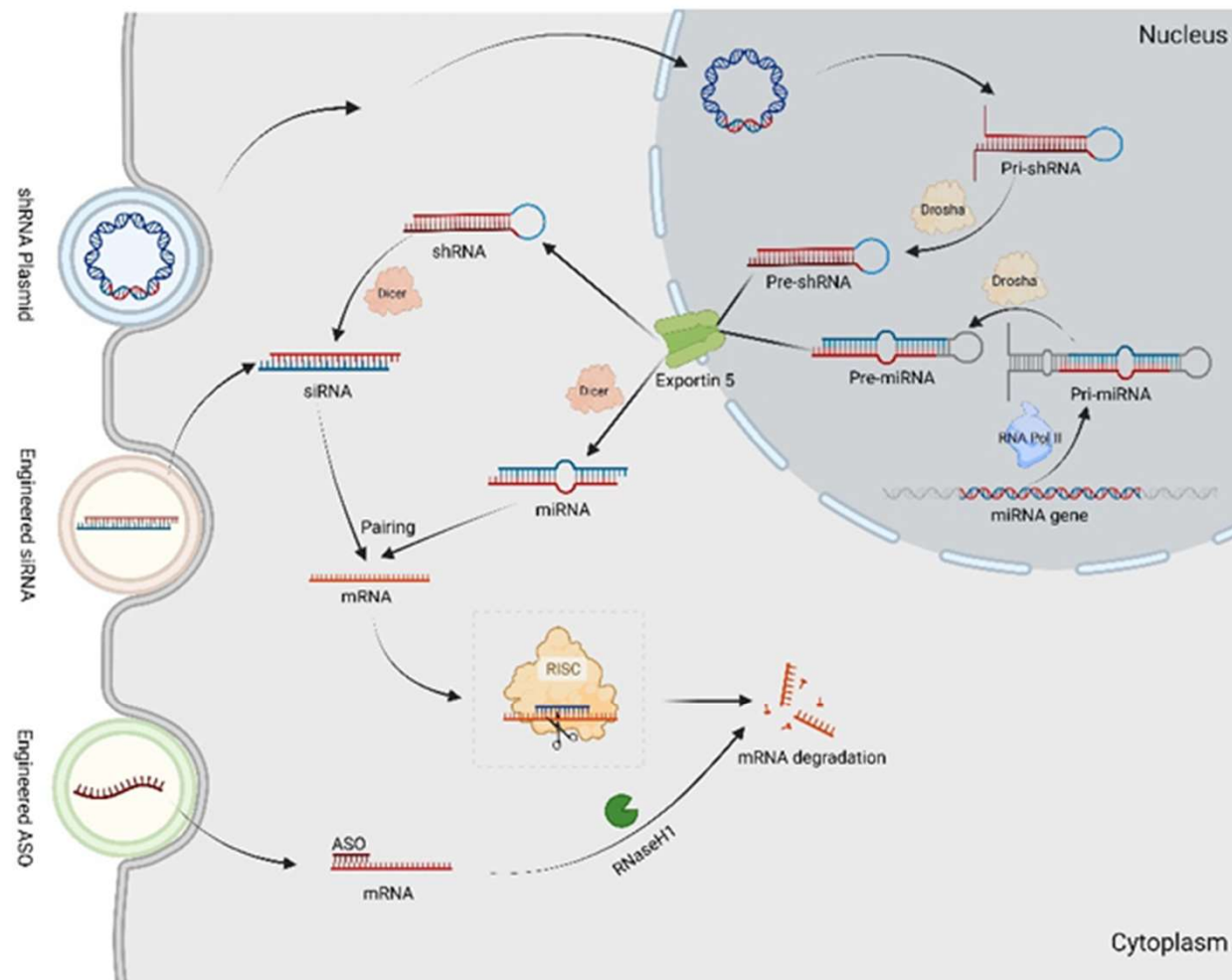
- Treatment of a disease through transferring genetic material into cells of the patients
- Gene addition and gene replacement are important methods for gene therapies
- Current gene editing technology is laying the foundation for the formulation of future disease therapies
- Gene expression of **PMP22** can be downregulated to preserve myelin and axons using intraneural injections

Drug treatment

► 6. RNA interference (figure following)

- The Pri-miRNAs are transcribed by DNA dependent RNA polymerase II (Pol II) transcriptional complex.
- After expression in the nucleus, Pri shRNAs and Pri-miRNAs are processed by Drosha to Pre-shRNAs and Pre-miRNAs.
- Afterwards, both Pre-shRNAs and Pre-miRNAs are exported by Exportin-5 to the cytoplasm.
- The shRNAs and Pre-miRNAs are associated with Dicer, resulting in the removal of the loop sequence and synthesis siRNAs as well as miRNA.
- By targeting complementary sequence mRNAs, they construct into RISC, resulting in mRNAs degradation.
- The synthetic ASO combined with target mRNAs generates a DNA-RNA heteroduplex in cytoplasm, which is recognized by RNase H1

Targets of Treatment



Dong, 2024

Drug treatment

▶ 7. Stem cell research

- ▶ Schwann cells derived from dental pulp stem cells (DPSCs), which are typically obtained from third molar extractions
- ▶ Present minimal ethical concerns due to their origin as medical waste, can also be utilized for studying demyelinating neurodegenerative diseases

Drug treatment

► 8. Genetic counseling

- Genetic counseling is crucial for CMT patients to comprehend and adjust to the medical, psychological, and familial implications of genetic factors contributing to the disease
- Whole-genome sequencing can identify the type and genetic spectrum of CMT
- Genetic counseling empowers patients with a comprehensive understanding of the disease's pathogenesis and inheritance risk to future generations, offering essential guidance for treatment timing and programs
- At the same time, genetic counseling has significant societal implications in terms of eugenics

Drug treatment

► 8. Genetic counseling (continued)

- Prenatal diagnosis is possible in several countries as well as preimplantation genetic diagnosis during in vitro fertilization procedures
- CMT women have only a little increase in risk of pregnancy complications, such as preterm delivery, abnormal presentation, and placenta previa, with no significant increased risks for the newborns
 - However, subjective worsening of CMT has been reported, in dependently from CMT type, in a percentage of cases ranging from 16% of the patients to 38% of the pregnancies with full recovery occurring only in a minority of cases

Drug treatment

- ▶ It should be borne in mind that the use of drugs that are toxic for the peripheral nerve should be avoided if possible
 - ▶ The decision depends on the duration of treatment (a short course is not a problem), the degree of toxicity (many drugs carry very low risks), and the risk-benefit ratio
- ▶ The most difficult choices are related to patients with cancer, lymphoma, or leukemia for whom vinka alkaloid, platin, or taxol compounds are prescribed
 - ▶ For example, vinka alkaloids have been reported to precipitate severe acute neuropathy, Guillain-Barrè-like, in previously undiagnosed CMT patients, and their use should be avoided if possible unless there is no real alternative

Targets of treatment

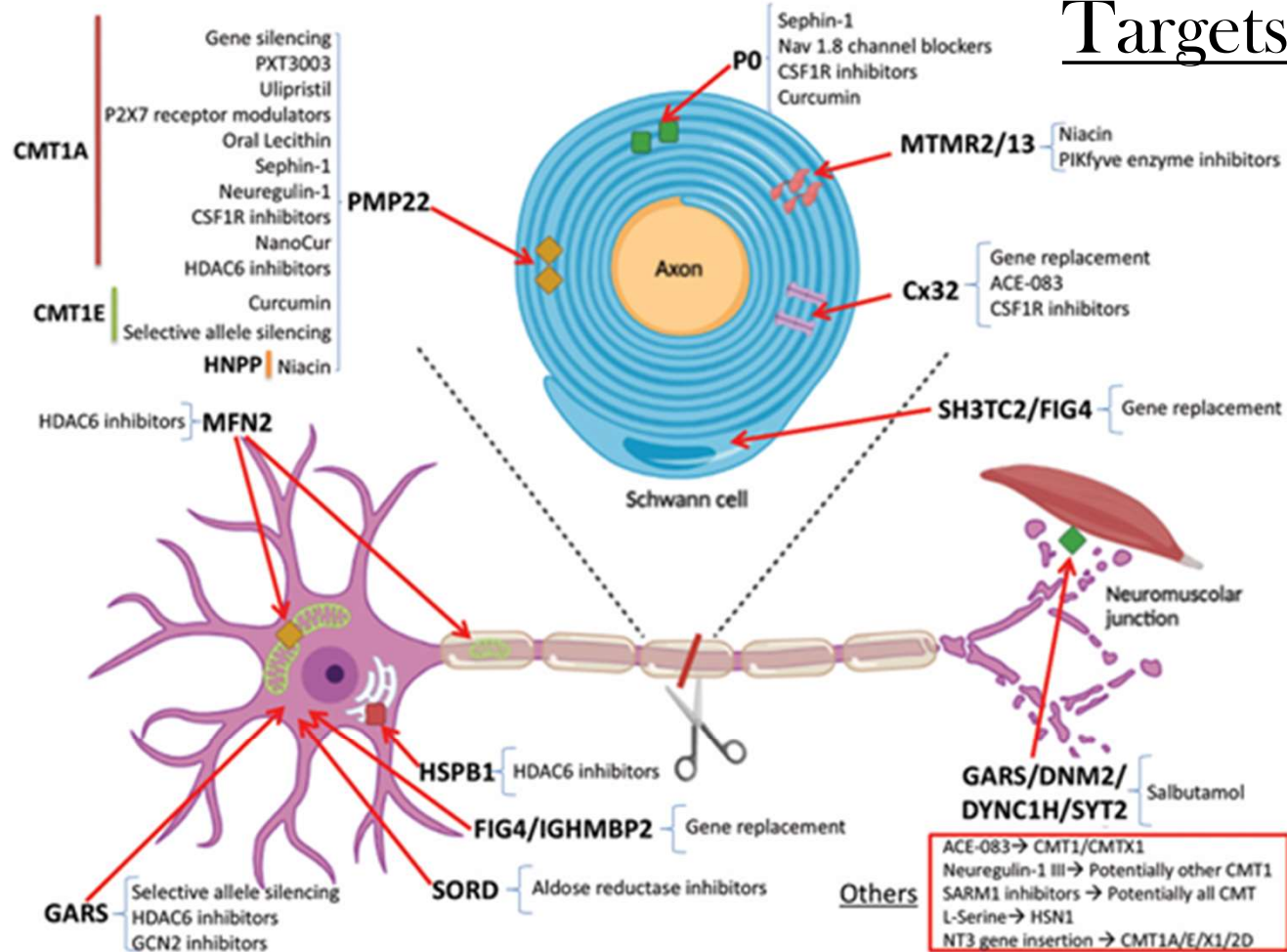


Figure 1. Schematic representation of a motor neuron with its myelinated axon, neuromuscular junction and innervated muscle fiber. A transversal section of an internode with the myelinating Schwann cell is also shown. A list of potential treatments already tested, under evaluation, or under development is provided for each CMT type or related protein. Reproduced from [4] with permission of Elsevier.

Degrado,
2025

Drug treatment

► Conclusions (Dong et al, 2024)

- Scientific knowledge of CMT mechanisms continues to evolve, and it is now clear that the dysregulation of myelin and axons is key in the pathogenesis of CMT
- In the past two decades, considerable progress has been made in our understanding of the importance of next generation sequencing for identifying targets within pathways that lead to CMT

Operative treatment

- ▶ In the early stages of the condition, flexible deformities including equinus, cavus, or forefoot valgus and hindfoot varus can be corrected by joint-sparing osteotomies, soft-tissue release, and tendon transfers, with elaborate preoperative evaluation and individualized plans
- ▶ Operative interventions at this stage may prevent the progression of deformities and joint fusions in the future
 - ▶ However, for patients in later stages, who have severely rigid deformities, a complex plan combining ligament and soft-tissue release, osteotomies, and arthrodesis may be an inevitable choice

Operative treatment

- ▶ For adults, referral for surgical evaluation is recommended when conservative treatments fail, deformities become rigid and severe, or there is a risk of musculoskeletal injury due to the foot deformities
- ▶ Data on optimal surgical timing for children remains insufficient to make specific recommendations
 - ▶ Experts also agreed on procedures to avoid, such as isolated peroneus longus or tibialis posterior tendon lengthening, isolated plantar fascia release, ankle arthroplasty, and arthrodesis in children

Operative treatment

- ▶ About 20% of CMT patients undergo surgery for foot deformities
- ▶ Scoliosis occurs in 20–30% of patients and may require bracing, physical therapy, and rarely surgery, according to unpublished data from the Italian CMT Registry

Orthoses

- ▶ 266 participants from the Italian CMT Registry showed that only 55% of prescribed orthoses were used, with tolerability issues reported by 59% of patients
- ▶ Blouin et al. reported that in a cohort of 940 CMT patients in France, who participated in a survey examining the relationship between the care pathways followed and the use of orthotic devices, only 49% of the prescribed AFOs were actually used
- ▶ Early and customized orthotic prescriptions have been shown to improve tolerability and effectiveness. In a cohort of 314 CMT patients, Zuccarino et al. found that over one-third experienced dissatisfaction with AFOs due to both physical and psychological discomfort

Orthoses

- ▶ The recent adoption of 3D printing for orthotic customization has the potential to enhance tolerability and patient satisfaction
 - ▶ One study demonstrated that 3D-printed replica AFOs not only matched the performance of traditional AFOs but also improved foot positioning at initial contact and during the loading response and significantly reduced pressure under the entire foot, rear foot, and forefoot compared to wearing shoes alone

Orthoses

- ▶ 3-D printing allows for the replication of traditional handmade AFOs while facilitating the creation of lighter, redesigned versions with innovative features to enhance bio mechanics
 - ▶ Multidisciplinary management, including regular reassessment to monitor the progression of foot deformities and weakness, is recommended to ensure appropriate orthotic adaptation over time

Rehabilitative treatment

- ▶ Strengthening exercises focus on compensating for the weaker distal muscles, while stretching is employed for joint motion maintenance and contracture prevention
 - ▶ Improvements in tibialis anterior strength remained evident after 2 years from the strengthening trial in children, suggesting that early intervention may be particularly beneficial
- ▶ Importantly, recent expert consensus has led to the development of clinical management guidelines for children and adolescents with CMT, based on Delphi and GRADE methodologies

GRADE approach

- ▶ Strengthening Paediatric CMT Best Practice Guidelines Consortium to develop evidence and consensus- based recommendations for the clinical management of children and adolescents with CMT
 - ▶ Primary objective of promoting optimal, standardised care globally
- ▶ Development of this clinical practice guideline involved a series of systematic reviews covering 10 clinical questions, modified Delphi methodology involving an international panel of clinicians to generate consensus where evidence did not exist
- ▶ Application of the Grading of Recommendations, Assessment, Development and Evaluation (GRADE) approach to evaluate the body of literature and formulate recommendations

GRADE approach

- ▶ The final guideline includes three evidence- based and 31 consensus- based recommendations to encompass the management of muscle weakness, balance and mobility impairment, sensory symptoms, muscle cramps, impaired upper limb function, respiratory impairment, maintenance of joint range of motion and non- surgical management of joint deformity
- ▶ Consensus was not achieved in some management areas, reflecting differences in practice between clinicians and healthcare settings, and highlighting the need for further research
- ▶ Clinical practice guideline provides practical and implementable guidance on the management of common clinical problems experienced by children with CMT and advocates for improved access to multidisciplinary care

Box 1 Final clinical management questions

- ▶ In children with Charcot-Marie-Tooth disease (CMT), what treatments should be recommended for weakness?
- ▶ In children with CMT, what treatments should be recommended for maintaining and improving joint range of motion?
- ▶ In children with CMT, what treatments should be recommended for impaired mobility?
- ▶ In children with CMT, what treatments should be recommended for balance impairment?
- ▶ In children with CMT, what treatments should be recommended for muscle cramps?
- ▶ In children with CMT, what non-surgical treatments should be recommended for joint (foot, hand, hip, knee) or spine deformity?
- ▶ In children with CMT, what treatments should be recommended for sensory symptoms/impairment?
- ▶ In children with CMT, what treatments should be recommended for impaired upper limb function?
- ▶ In children with CMT, what treatments should be recommended for fatigue?
- ▶ In children with CMT, what treatments should be recommended for respiratory deficits?

Box 2 Clinical practice guideline for the management of childhood Charcot-Marie-Tooth disease (CMT)

Evidence-based recommendations

Strong evidence-based recommendations:

- We recommend the use of progressive resistance exercise of the ankle dorsiflexors to improve muscle strength and slow progression of muscle weakness (⊕⊕⊕○).

Conditional evidence-based recommendations:

- In children with CMT1A, we suggest not treating with high-dose ascorbic acid for weakness, impaired mobility, balance impairment or impaired upper limb function (⊕⊕○○).
- In children with CMT1A, we suggest not using botulinum toxin in the lower limbs for the management of flexible cavus foot deformity (⊕⊕○○).

Consensus-based recommendations

1. In children with CMT, what treatments should be recommended for weakness?

- ▶ A tailored strength training program should be encouraged, under the supervision of the child's treating physician and/or allied health clinician. The clinician should provide guidance in order to ensure that the exercise program is carried out safely, monitor progress and modify the program as appropriate.
- ▶ Strength training should start at low resistance and gradually build up (as per generic paediatric recommendations from the American Academy of Sports Medicine).²⁸
- ▶ Strength training of proximal/core muscles (trunk, hip and shoulder girdle) should be encouraged.
- ▶ Exercise programs should include rest days to encourage recovery. That is, strength training should occur on non-consecutive days for the same muscle group (as per generic recommendations from the National Strength and Conditioning Association).²⁹
- ▶ Exercise should cease temporarily and the exercise regimen should be modified, if there are any signs of exercise-induced muscle damage (eg, excessive muscle cramping, dark brown urine).

2. In children with CMT, what treatments should be recommended for maintaining and improving joint range of motion?

- ▶ Stretching of joints prone to contracture should form part of rehabilitation management.
- ▶ Serial casting should be used to improve ankle dorsiflexion range of motion in children who have restricted joint range of motion.
- ▶ Serial night casting may be used as an alternative to serial casting for improving ankle dorsiflexion range of motion in children for whom serial casting is contraindicated, or for those children unable to tolerate serial casting.

3. In children with CMT, what treatments should be recommended for impaired mobility?

- ▶ Ankle–foot orthoses should be used in carefully selected children with impaired functional ambulation due to:
 - foot drop (ankle dorsiflexion weakness)
 - trips and falls
 - ankle instability.
 - ▶ Foot orthoses may be used in children with foot pain.
 - ▶ Children with impaired mobility should wear well-fitting/ supportive footwear.
-
- ▶ Gait and mobility aids should be used for children with impaired community ambulation and prescribed by a qualified health professional with experience in their provision.

4. In children with CMT, what treatments should be recommended for balance impairment?

- ▶ Activities such as balance retraining or core and postural strengthening tailored to the individual child should be used to improve balance.
- ▶ Child-appropriate or adolescent-appropriate recreational/ community-based activities (eg, dance, Pilates, non-contact martial arts and play-based therapy) may be used to improve balance.

5. In children with CMT, what treatments should be recommended for muscle cramps?

- ▶ Stretching of involved muscle groups may be used for the treatment of cramps.

6. In children with CMT, what non-surgical treatments should be recommended for joint (foot, hand, hip, knee) or spine deformity?

- ▶ Children should be referred to an orthopaedic surgeon for assessment and management of:
 - Painful, disabling or progressive pes cavus not responsive to conservative measures (eg, foot orthoses).
 - Ankle contracture not responsive to stretching interventions.
 - Features of hip dysplasia.
 - Features of moderate scoliosis (eg, Cobb angle $>20-30^{\circ}$).
- ▶ A hip X-ray should be considered in all children to screen for hip dysplasia.

7. In children with CMT, what treatments should be recommended for sensory symptoms/impairment?

- ▶ Anticonvulsants such as gabapentin and pregabalin may be used for the treatment of positive sensory symptoms.
- ▶ Musculoskeletal pain may be secondary to structural, orthopaedic or mechanical causes. The underlying cause should be assessed and managed as appropriate.
- ▶ Physical therapy such as stretching and strengthening should be used for musculoskeletal pain.
- ▶ Non-pharmaceutical approaches should be used in conjunction with prescribed medications in chronic pain (pain lasting >6 months) and, if available, within a specialised multidisciplinary health care team.
- ▶ Potentially addictive medications such as muscle relaxants, narcotics and opioids should be avoided when possible for the treatment of pain.

8. In children with CMT, what treatments should be recommended for impaired upper limb function?

- ▶ Adapted equipment should be used to improve performance of activities of daily living in children who have impairment in the hands.
- ▶ Adapting keyboard settings to reduce frequency of errant key strokes, word prediction software and/or voice to text software should be used to improve typewritten communication in children who have impairment in the hands.
- ▶ In carefully selected children with upper limb impairment impacting performance of activities of daily living, functional splints (eg, neoprene thumb spica) may be used to improve function in targeted activities.
- ▶ In carefully selected children with upper limb impairment impacting performance of activities of daily living, resting splints (eg, wrist cock up splint for children with wrist drop) may be used to improve function in targeted activities.

9. In children with CMT, what treatments should be recommended for fatigue?

► *No consensus-based recommendations.*

10. In children with CMT, what treatments should be recommended for respiratory deficits?

- Baseline pulmonary function testing should be undertaken in the following children when they are able to complete this reliably (usually from the age of 5–6 years):
 - Children with symptoms of sleep-disordered breathing (eg, unexplained headaches, daytime somnolence or symptoms of obstructive sleep apnoea).
 - Children with recurrent lower respiratory tract infections (defined as: >2 courses of antibiotics over 4 months or >2 hospital admissions for a respiratory tract infection in 12 months).
 - Children with scoliosis, with a Cobb angle of >40°.
 - All non-ambulant children.

- ▶ The following children are considered high risk for having respiratory deficits and should be referred to a respiratory physician/sleep physician for ongoing management (including for consideration of a sleep study):
 - Children with symptoms of sleep-disordered breathing.
 - Forced vital capacity <60% predicted for age.
 - Scoliosis, with a Cobb angle of >40°
 - Recurrent lower respiratory tract infections (defined as: >2 courses of antibiotics over 4 months or >2 hospital admissions for a respiratory tract infection in 12 months).
 - Non-ambulant children.

- 
- ▶ In children who have an acute lower respiratory tract infection and are considered high risk for respiratory deficits (as outlined above):
 - Acute respiratory illness management should include a low threshold to start antibiotics.
 - Chest physiotherapy should be used when acutely unwell with a lower respiratory tract infection.
 - A respiratory physician should be involved for consideration of additional measures (eg, mechanical cough assistance or increased respiratory support) when acutely unwell with a lower respiratory tract infection.

- ▶ Formal evaluation of swallowing should be undertaken in children with recurrent respiratory tract illnesses or history of aspiration.
- ▶ All children at high risk of respiratory deficits (as outlined above) should have the influenza vaccination annually.

Grade ratings:

⊕⊕⊕⊕ High.

⊕⊕⊕○ Moderate.

⊕⊕○○ Low.

⊕○○○ Very low.

CMT Outcome measures

- ▶ MRI imaging of lower limbs has been mainly studied in CMT1A patients, demonstrating that the free intramuscular fat accumulation (IMFA) increases by 1-2% per year within calf muscles independently of a subject's overall level of activity or fitness
 - ▶ Makes MRI the most sensitive measure of progression that has been identified to date.
 - ▶ Evaluation of both thigh and calf muscles will enhance the MRI sensitivity across different stages of the disease, as proximal thigh muscle involvement may continue to progress in a linear mode even after the distal calf muscle have reached an end-stage condition reducing their IMFA sensitivity to detect further changes

CMT Outcome measures

- ▶ Blood biomarkers provide another emerging tool that may complement clinical and MRI-based outcome measures
 - ▶ Molecular markers reflecting abnormalities in SCs, degenerating axons, or denervated muscle could serve as biomarkers for severity and progression in CMT neuropathies
 - ▶ Recent studies have shown increased plasma NFL levels in patients with different types of CMT

CMT Future Research

- ▶ Due to the genetic diversity of CMT and its many subtypes, the current treatment is limited
- ▶ One of the major hurdles is the unavailability of sensitive outcome measures that would allow the detection of treatment response in a slowly progressive chronic disease such as CMT1A, which also shows marked phenotypic variability
- ▶ While treatment options are limited for CMT, current management and future research will be explored
- ▶ CMT has limited treatment options due to lack of biomarkers and the genetic variability of the disease. Despite these difficulties, researchers are optimistic about the future advancement of pharmacological treatment to improve quality of life for CMT patients

Case study (EDX July 2025)

Test Date: Jul2025

Reason for Referral: BLE pain

Patient Name: DOB: 1977 **Gender:** Female **HT:** 5'7" **WT:** 186 lbs.

Referring Provider:

Brief History: Patient with complaints of joint pain that occurs in bilateral hips/ankle > knees (right = left). Patient notes that symptoms occurred about a month following diagnosis of pericarditis. Patient is referred from the rheumatologist to examine LE for myositis. Patient denies BLE weakness although notes fatigue with walking that has improved with hydroxychloroquine a few months ago. Patient notes intermittent BLE numbness primarily when legs are elevated. Patient denies LBP. Patient notes that she also has pain in bilateral hands (right = left) and numbness primarily while sleeping.

PMH: Pericarditis

PSH: C-section x 2, Hysterectomy

Case study (EDX July 2025)

Nerve Conduction Studies

Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	P-T Amp (µV)	Norm P-T Amp	Full Dur (ms)	Site1	Site2	Delta-P (ms)	Norm Delta (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Superficial Peroneal Anti Sensory (Ant Lat Mall)													
Ant Lat leg	NR		<4.5		>4.0		Ant Lat leg	Ant Lat Mall			14.0		
Left Sural Anti Sensory (Lat Mall)													
Calf	NR		<4.5		>4.0		Calf	Lat Mall			14.0		
Right Sural Anti Sensory (Lat Mall)													
Calf	NR		<4.5		>4.0		Calf	Lat Mall			14.0		

Case study (EDX July 2025)

Motor Summary Table

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Full Dur (ms)	Site1	Site2	Delta-0 (ms)	Norm Delta (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Peroneal Motor (Ext Dig Brev)													
Ankle	NR		<6.5		>1.3		Ankle	Ext Dig Brev			8.0		
B Fib	NR				>1.3		B Fib	Ankle			34.0		>38
Poplt	NR				>1.3		Poplt	B Fib			11.0		>42
Right Peroneal Motor (Ext Dig Brev)													
Ankle	NR		<6.5		>1.3		Ankle	Ext Dig Brev			8.0		
B Fib	NR				>1.3		B Fib	Ankle			35.0		>38
Poplt	NR				>1.3		Poplt	B Fib			11.0		>42
Left Peroneal TA Motor (Tib Ant)													
Fib Head		3.0		0.8	>1.3	24.45	Fib Head	Tib Ant	3.0		8.0		
Poplit		12.9		0.3	>1.3	21.17	Poplit	Fib Head	9.9		11.0	11	>42
Right Peroneal TA Motor (Tib Ant)													
Fib Head		4.7		1.2	>1.3	21.25	Fib Head	Tib Ant	4.7		8.0		
Poplit		11.6		0.4	>1.3	20.08	Poplit	Fib Head	6.9		11.0	16	>42
Right Tibial Motor (Abd Hall Brev)													
Ankle		16.7	<6.1	0.3	>4.4	40.31	Ankle	Abd Hall Brev	16.7		8.0		
Knee		46.9		0.1	>2.0	18.28	Knee	Ankle	30.2		43.5	14	>39

Techniques and reference values used above were published in Muscle & Nerve; 54:371-377, Sept 2016.

Case study (EDX July 2025)

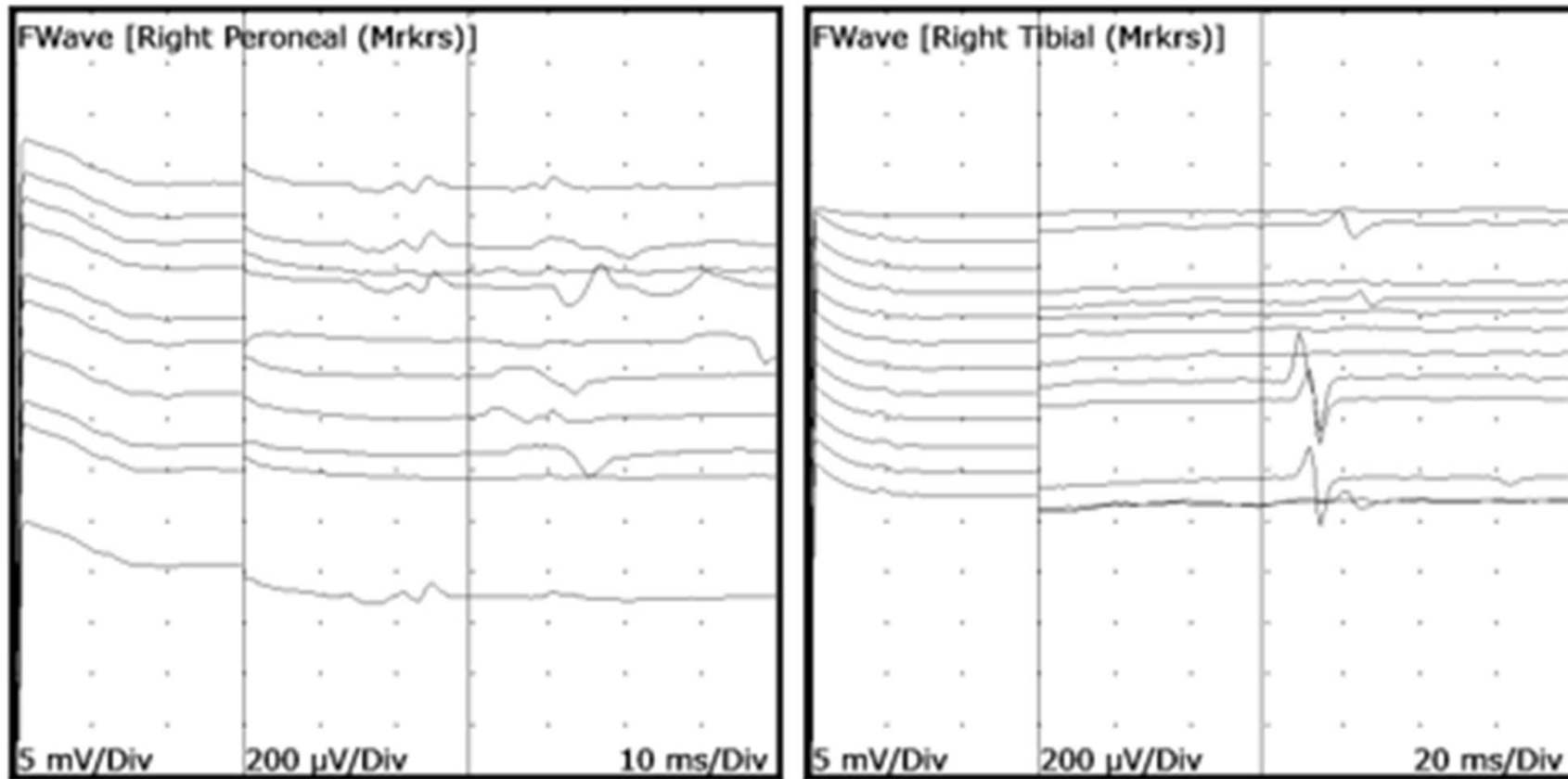
F Wave Studies

NR	F-Lat (ms)	Lat Norm (ms)	L-R F-Lat (ms)	L-R Lat Norm
Right Peroneal (Mrkrs) (TA)				
	59.51	<39		<2.0
Right Tibial (Mrkrs) (Abd Hallucis)				
	118.79	<59		<2.0

H Reflex Studies

NR	H-Lat (ms)	Lat Norm (ms)	L-R H-Lat (ms)	L-R Lat Norm	Leg Length (cm)
Left Tibial (Soleus)					
	55.77	<33.2	2.87	<1.5	42.00
Right Tibial (Soleus)					
	52.90	<33.2	2.87	<1.5	42.00

Case study (EDX July 2025)



Case study (EDX July 2025)

EMG*

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Dischg	Fasc	Amp	Dur	Poly	Recrt	Int Pat
Right	MedGastroc	Tibial	S1-2	Incr	1+	1+	Nml	Nml	>5mV	Nml	>50%	Nml	Nml
Right	GluteusMax	InfGluteal	L5-S2	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml
Right	AntTibialis	Deep Peroneal	L4-5	Incr	1+	1+	Nml	Nml	>5mV	Nml	>50%	Nml	Nml
Right	VastusMed	Femoral	L2-4	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml
Right	TensorFascLat	SupGluteal	L4-5-S1	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml
Right	UpperLumbParaspinals	PPRami	L1-2	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml
Right	MidLumbParaspinals	PPRami	L3-4	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml
Right	LowerLumbParaspinals	PPRami	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml
Left	MedGastroc	Tibial	S1-2	Incr	1+	1+	Nml	Nml	>5mV	Nml	>50%	Nml	Nml
Left	GluteusMax	InfGluteal	L5-S2	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml
Left	AntTibialis	Deep Peroneal	L4-5	Nml	Nml	Nml	CRD	Nml	>5mV	Nml	>50%	Nml	Nml
Left	VastusMed	Femoral	L2-4	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml
Left	TensorFascLat	SupGluteal	L4-5-S1	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml
Left	UpperLumbParaspinals	PPRami	L1-2	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml
Left	MidLumbParaspinals	PPRami	L3-4	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml
Left	LowerLumbParaspinals	PPRami	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml	Nml

*Monopolar needle

Case study (EDX July 2025)

Interpretation: Abnormal study. There is electrophysiologic evidence suggestive of:

1. Severe length-dependent sensorimotor polyneuropathic process, demyelinating > axonal in nature, affecting the bilateral lower extremities symmetrically with markedly slowed motor nerve conduction velocities, absent sensory responses and evidence of ongoing denervation in the bilateral MG and right TA muscles.

Referral to neuromuscular specialist is highly recommended for further workup.

There is no definitive electrophysiological evidence of a bilateral L3-S1 spinal nerve lesion of the motor fibers. The L3-S1 sensory fibers cannot be assessed secondary to the nature of dorsal root ganglion sparing with radicular lesions. Patient recommended to follow up with referring provider.

Neurology consult (September 2025)

- ▶ 47 y.o. woman with a history that includes undifferentiated connective tissue disease (inflammatory arthropathy, possible ITP and idiopathic pericarditis) presenting for evaluation of neuropathy.
- ▶ In 2023 she developed chest pain and was diagnosed with pericarditis. She also developed joint pain, which she has had since then. She was found to have elevated cardiolipin abs and mildly elevated CK (200s-300s). She was treated with indomethacin and colchicine
- ▶ She was started on plaquenil by Dr. XXX in early 2025, with possible mild benefit, but ongoing significant joint and muscle pain. CK was persistently mildly elevated in early 2024, but she has not had any muscle weakness, though she does report general exercise intolerance.
- ▶ She reports transient paresthesias as well as temperature sensitivity in the hands/feet, without fixed numbness. She reports some decline in her balance, and often trips going up stairs. With balance worse in the dark or on uneven ground.

Neurology consult (September 2025)

- ▶ She is not aware of any family members with neuropathy.
 - ▶ She has never been exposed to chemotherapy.
 - ▶ She does not drink alcohol currently, and never drank heavily.
- ▶ EDX July 2025 EMG/NCS (“On my review of data”)
 - ▶ LE sensory responses absent (UE sensories not checked)
 - ▶ LE motor response was severely reduced amplitudes and CVs in the 10s.
 - ▶ EMG legs showed acute-on-chronic denervation in distal leg muscles.

Neurology consult (September 2025)

► PHYSICAL EXAMINATION:

- Musculoskeletal: She had high arches, mild hammertoes and slightly tight heel cords bilaterally.

► NEUROLOGICAL EXAM:

- Mental status: Patient was alert, attentive, and oriented x 3.
- Speech was coherent and fluent without dysarthria or aphasia.
- Memory, comprehension and ability to follow commands were intact.
- Cranial nerves: Visual fields were full. Pupils were round and reacted to light and accommodation. Extraocular movements were full. There was no ptosis. There was no face, jaw, palate or tongue weakness or atrophy. Facial sensation was normal. Hearing was grossly intact. Shoulder shrug was normal.

Neurology consult (September 2025)

► NEUROLOGICAL EXAM:

- **Motor:** Mild atrophy in the distal shins and feet, without normal muscle bulk in the hands/arms. Manual muscle testing revealed the following MRC grades (R/L where applicable): SA 5/5, EF 5/5, EE 5/5, WE 5/5, FF 5/5, FE 5/5, FDI 5/5, APB 5/5. HF 5/5, HAbd 5/5, KE 5/5, KF 5/5, ADF 4/4, APF 5/5, AInv 4+/4+, AEv 4+/4+, EHL 4-/3.
- **Complex motor skills:** Normal coordination.
- **Sensory:** Vibratory sensation using Rydel fork was absent at the toes, 3-4 at the ankles, 4-5 at the knees and 7-8 at the fingers. Sensation of pinprick was reduced below the mid-thighs bilaterally, intact throughout the hands/arms.
- **Gait:** Patient had a normal casual gait. She could stand on her toes, but had difficulty standing on her heels. She had mild difficulty with tandem. There was mild sway with Romberg.
- **Reflexes:** Deep tendon reflexes were absent at the biceps, triceps and brachioradialis, absent at the knees and ankles. Plantar responses were mute bilaterally. Hoffman's sign was absent bilaterally.

Neurology consult (September 2025)

► ASSESSMENT:

- 47 y.o. woman with a history that includes undifferentiated connective tissue disease (inflammatory arthropathy, possible ITP and idiopathic pericarditis) presenting for evaluation of neuropathy.
- In 2023 she developed chest pain and was diagnosed with pericarditis. She also developed joint pain, which she has had since then. She was found to have elevated cardiolipin abs and mildly elevated CK (200s-300s). She was treated with indomethacin and colchicine without benefit, then more recently started on plaquenil with mild benefit in arthralgia/myalgia.
- CK has remained only mildly elevated and she has not had weakness. She also has not noticed numbness, but did report transient paresthesias and decline in balance.
- EMG/NCS was done at OMC and showed a demyelinating polyneuropathy.
- Examination today showed distal leg/foot muscle atrophy, high arches/mild hammertoes/tight heel cords, mild distal leg/foot weakness and distal sensory loss.

Neurology consult (September 2025)

- ▶ Polyneuropathy, likely demyelinating
 - ▶ Her presentation, particularly the degree of sensory loss, without her awareness of it, as well as distal weakness and foot morphology changes, raise concern for a hereditary neuropathy, such as CMT.
 - ▶ Given possible demyelination on outside NCS, CMT1A seems most likely at this time.
 - ▶ I recommend repeat EMG/NCS of 1 arm and leg for better characterization of her neuropathy.
 - ▶ I also recommend genetic testing with Prevention Genetics Hereditary neuropathy sponsored panel, which includes PMP22.
 - ▶ I counseled the patient regarding the risks and benefits of genetic testing, including implications for family members, indeterminate results (I.e. variants of uncertain significance) and off-target information that could be obtained as a result of genetic testing. She consented to gene panel testing.
 - ▶ For completeness, I will also check SPEP/IFE and B12/MMA.

Neurology consult (September 2025)

► Mild chronic CK elevation

- At this juncture, significance is uncertain.
- She does not have proximal weakness on exam to suggest an inflammatory myopathy, nor myopathic changes on recent EMG/NCS.
- She could have mild idiopathic hyperCKemia.
- Mild CK elevation could also be related to her undifferentiated connective tissue/rheumatologic disease, though as noted above, her presentation does not suggest a significant inflammatory myopathy/myositis.
- I recommend EMG/NCS of an arm and leg, as above, and will needle proximal arm and leg muscles.

Neurology consult (September 2025)

► RECOMMENDATIONS:

- Check SPEP/IFE, B12/MMA
- Prevention Genetics Sponsored Hereditary Neuropathies Panel ordered.
- EMG/NCS L arm and leg.
- Follow up with me for EMG/NCS and in ~4 months.

Neurology EDX (November 2025)

Sensory Sites

	Neg Peak Lat	Amplitude (O-P)	Segment	Distance	Start CV	Comment
Site	(ms)	(μ V)		(mm)	(m/s)	
Left Median Sensory						
Wrist-Dig II	NR	NR	Wrist-Dig II	130	NR	
Left Radial Sensory						
Forearm-Wrist	NR	NR	Forearm-Wrist	100	NR	
Left Superficial Fibular Sensory						
14 cm-Ankle	NR	NR	14 cm-Ankle	140	NR	
Left Sural Sensory						
Calf-Lat mall	NR	NR	Calf-Lat mall	140	NR	
Left Ulnar Sensory						
Wrist-Dig V	NR	NR	Wrist-Dig V	110	NR	

Neurology EDX (November 2025)

Motor Nerve Results

	Latency	Amplitude	Segment	Distance	CV	Total Duration	Temperature	Comment
Site	(ms)	(mV)		(cm)	(m/s)	(ms)	(°C)	
Left Fibular (EDB) Motor								
Ankle	NR	NR				NR	30.3	
Bel fib head	NR	NR	Bel fib head-Ankle	360	NR	NR	30.3	
Pop fossa	NR	NR	Pop fossa-Bel fib head	80	NR	NR	30.2	
Left Fibular (TA) Motor								
Fib head	6.1	2.7				14.0	30	
Pop fossa	11.3	2.8	Pop fossa-Fib head	80	15	13.3	30	
Left Median (APB) Motor								
Wrist	10.3	7.8				9.1	31.8	
Elbow	19.8	6.0	Elbow-Wrist	210	22	9.3	31.9	
	Latency	Amplitude	Segment	Distance	CV	Total Duration	Temperature	Comment
Site	(ms)	(mV)		(cm)	(m/s)	(ms)	(°C)	
Left Tibial (AH) Motor								
Ankle	12.6	0.35				14.5	23.6	
Knee	42.8	0.06	Knee-Ankle	400	13	14.1	23.8	
Left Ulnar (ADM) Motor								
Wrist	7.7	4.8				9.9	30.9	
Bel elbow	14.9	5.0	Bel elbow-Wrist	190	26	10.8	30.7	
Ab elbow	19.0	4.9	Ab elbow-Bel elbow	100	24	9.5	30.8	

Neurology EDX (November 2025)

Electromyography

Side	Muscle	Ins.Act	Fibs.PSW	Fasc	Other	Amp	Dur	Poly	Recrt	Comment
Left	FDI	Nml	Nml	Nml	Nml	Incr	Incr	0	ModRed	
Left	Vastus med	Nml	Nml	Nml	Nml	Nml-Incr	Nml-Incr	0	MildRed	
Left	Tib ant	Nml	Nml	Nml	Nml	Incr	Incr	0	ModRed	
Left	Gastroc MH	Nml	Nml	1+	Nml	Incr	Incr	0	ModRed	
Left	EHL	Incr	1+	Nml	Nml	Incr	Incr	0	Discrete	
Left	Tens fasc lat	Nml	Nml	Nml	Nml	Nml-Incr	Nml-Incr	0	Nml	

Neurology EDX (November 2025)

Impression:

This is an abnormal study of the left arm and leg.

1. There is electrodiagnostic evidence of severe, length-dependent, sensorimotor, demyelinating polyneuropathy, with features suggestive of a hereditary process (uniformly slowed conduction velocities, absence of conduction block or abnormal temporal dispersion).

Clinical Addendum:

I discussed the results of this study with the patient. The findings on today's study are consistent with CMT1A, due to her known PMP22 duplication. We discussed the diagnosis of CMT1A. I counseled her that it may be beneficial for her siblings and adult children to be tested, and I can help connect them to providers who can facilitate this if needed. She will follow up with me in February 2026 as scheduled.

Conclusions

- ▶ CMT1A is the most prevalent form of Charcot-Marie-Tooth disease, accounting for approximately 50% of all diagnosed cases and is attributed to mutations in the PMP22 gene
- ▶ Due to the lack of biomarkers and the genetic variability of the disease, the current treatment is limited.
 - ▶ While treatment options are limited for CMT, current management and future research will be explored
 - ▶ Despite these difficulties, researchers are optimistic about the future advancement of pharmacological treatment to improve quality of life for CMT patients
- ▶ EDX can help identify CMT patients and expedite referral to neurology for workup and genetic counseling

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THE PRIMARY CARE PHYSICAL THERAPIST AS A REFERRING PROVIDER FOR ELECTRODIAGNOSTIC TESTING

Andrus A

ABSTRACT

BACKGROUND: In the United States Military, physical therapists have a long history of successful primary care practice, including expanded practice privileges such as imaging and medication referrals. With the state of Utah now granting primary care provider status to physical therapists, we seek to discover how effectively civilian therapists are using their referral privileges in the field of electrodiagnostics.

MATERIALS AND METHODS: Electrophysiologic testing referrals placed by outpatient physical therapists in the States of Utah and Idaho were retrospectively analyzed to identify trends in referring diagnoses, their relationships to electrophysiologic findings, and patient care before and after testing. These results were compared with a similar sample of electrophysiologic cases drawn from physician referrals.

DISCUSSION: Physical Therapists demonstrate their excellent qualifications as referring providers through a level of diagnostic accuracy comparable to physician colleagues, however they may be held back by lack of education regarding diagnostic techniques and some misconceptions regarding nerve pathologies. Similar to physicians, physical therapists most commonly use electrodiagnostic (EDX) testing to evaluate for radiculopathies, polyneuropathies, and entrapment neuropathies, however referring diagnoses from physical therapists do not align with electrodiagnostic findings as well as with physician referrals. Most therapists refer for testing shortly after initial examination. EDX results often influence the course of physical therapy care or help to determine whether further physical therapy intervention will be pursued or the patient will be referred to another professional.

CONCLUSIONS: As physical therapists enter a new era of practice as primary care providers, diagnostic testing provides a rich opportunity to enhance patient care, but to make the most of this new and practicing therapists must be better educated in their expanding role and in the gamut of diagnostic resources available to them and their patients.

Austin Andrus, PT, DPT, ECS



The Primary Care Physical Therapist as a Referring Provider for Electrodiagnostic Testing

Austin S. Andrus, March 28, 2026

APTA Vision 2020

Released 2000

“Physical therapy, by 2020, will be provided by physical therapists who are **doctors of physical therapy** and who may be **board-certified specialists**. Consumers will have **direct access to physical therapists** in all environments for patient/client management, prevention, and wellness services. Physical therapists will be practitioners of choice in patients’/clients’ health networks and will hold all privileges of autonomous practice.”

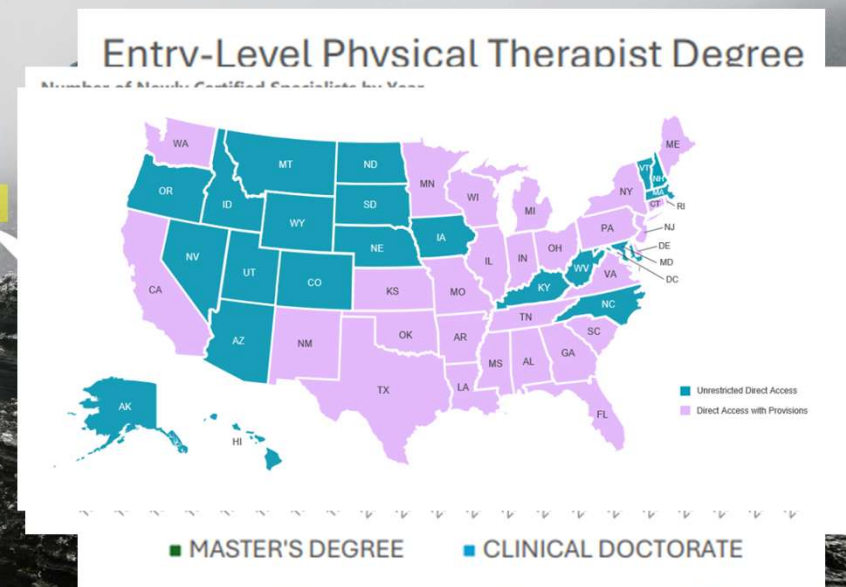


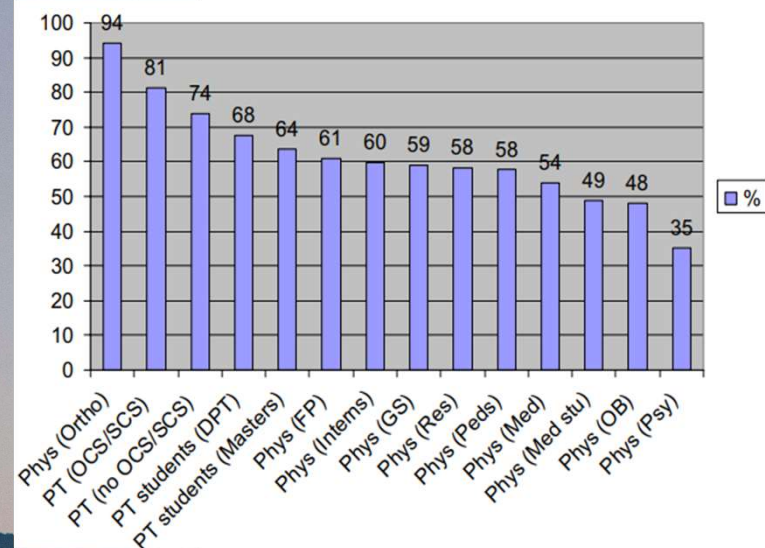
Photo by iStockphoto: <https://www.pexels.com/photo/a-person-standing-on-rock-formations-during-foggy-weather-4339677/>

Rising to the Occasion

Childs et al, 2005, 2007

- PTs outperform all medical specialties except orthopedic surgeons on musculoskeletal assessment

Standardized MSK Exam Scores



Denninger et al, 2018

- Patients who access PT directly have less time in PT, lower healthcare costs, and similar outcomes to those referred to PT

Variable	Mean Referral From Physician (n = 276) [†]	Mean Direct Access (PT First) (n = 171) [†]
Pain at discharge (NPRS, 0-10)	2.0 (1.7, 2.3)	2.0 (1.6, 2.4)
Disability at discharge (ODI/NDI, 0-50)	6.1 (5.2, 6.9)	5.6 (4.5, 6.7)
Total visits, n	7.6 (6.8, 8.3)	6.6 (5.7, 7.5)
Total time in care, d	46.9 (39.7, 54.1)	36.4 (27.4, 45.5)
Physical therapy costs	915 (790, 1040)	655 (499, 812)
Radiology costs	375 (277, 473)	206 (83, 330)
Surgical and injection costs	1634 (508, 2760)	600 (-813, 2013)
Other costs	96 (74, 118)	43 (15, 71)
Emergency room costs	61 (20, 102)	37 (-15, 89)
Total costs	3085 (1939, 4224)	1542 (108, 2976)

APTA Vision 2020

Released 2000

“Physical therapy, by 2020, will be provided by physical therapists who are doctors of physical therapy and who may be board-certified specialists. Consumers will have direct access to physical therapists in all environments for patient/client management, prevention, and wellness services. Physical therapists will be practitioners of choice in patients’/clients’ health networks and will **hold all privileges of autonomous practice.**”



Photo by iStockphoto: <https://www.pexels.com/photo/a-person-standing-on-rock-formations-during-foggy-weather-4339677/>

PT in the US Military

Since 1972, military regulations have allowed PTs as non-physician primary care providers for neuromusculoskeletal disorders

- Supervising physician required, as with PAs
- 6-month training program for diagnostic imaging, medical screening, and pharmacological management
- Certified PTs can refer patients for appropriate imaging, refer to other providers, and prescribe analgesic medication



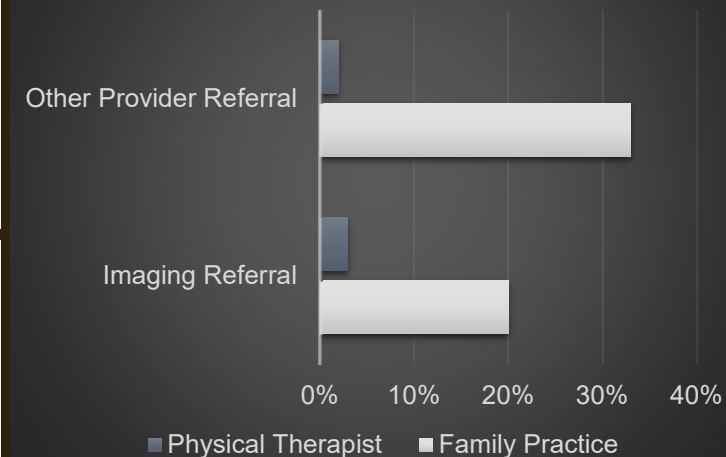
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PT in the US Military

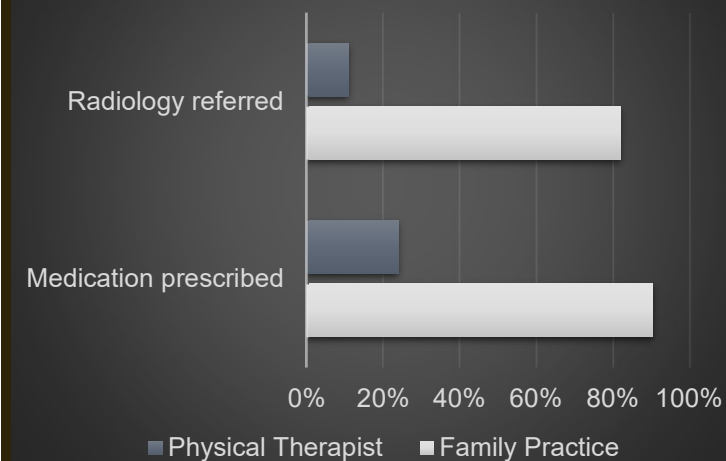
PTs as primary care providers save the military millions of dollars every year

- 17-50% higher return to duty rate
 - Less medical evacuations required
- Dramatically less imaging and medication ordered than family practice

% Visits Resulting in Referral
Mabry, 2020



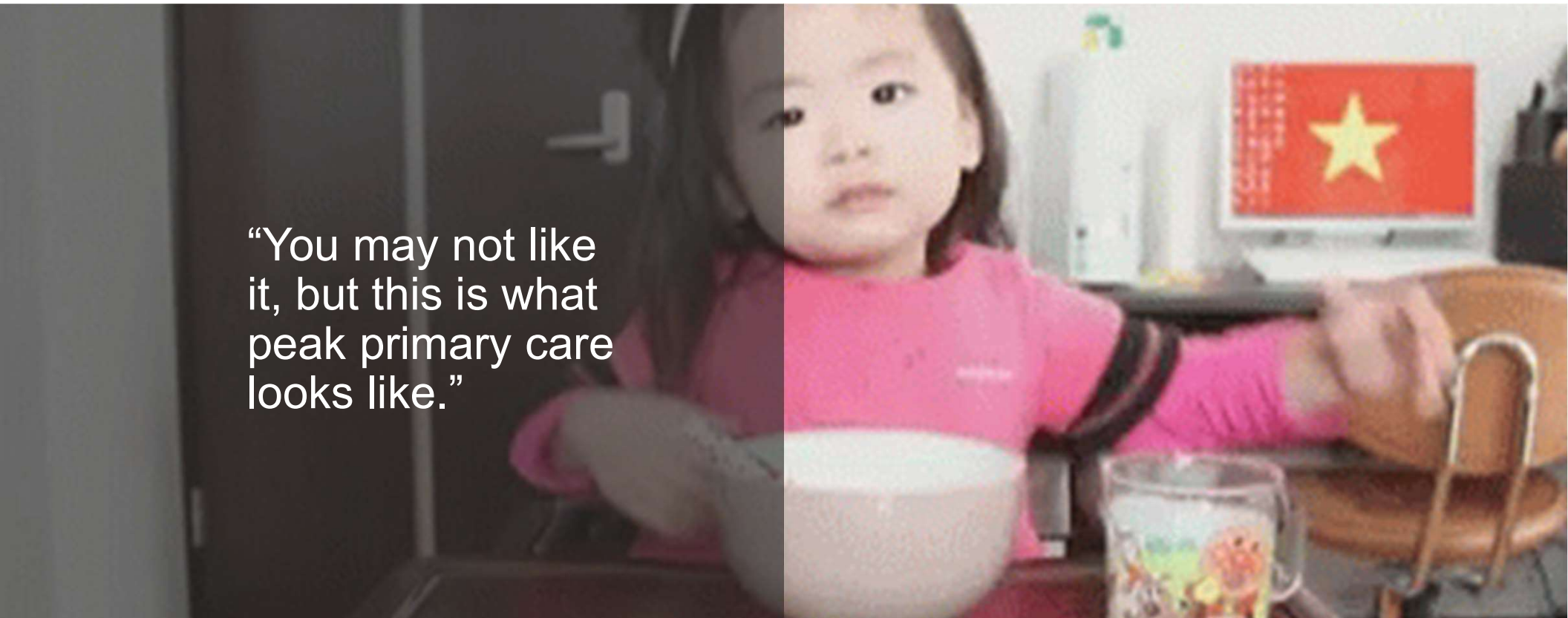
Prescription Rate
McGill, 2013



Challenging the Narrative

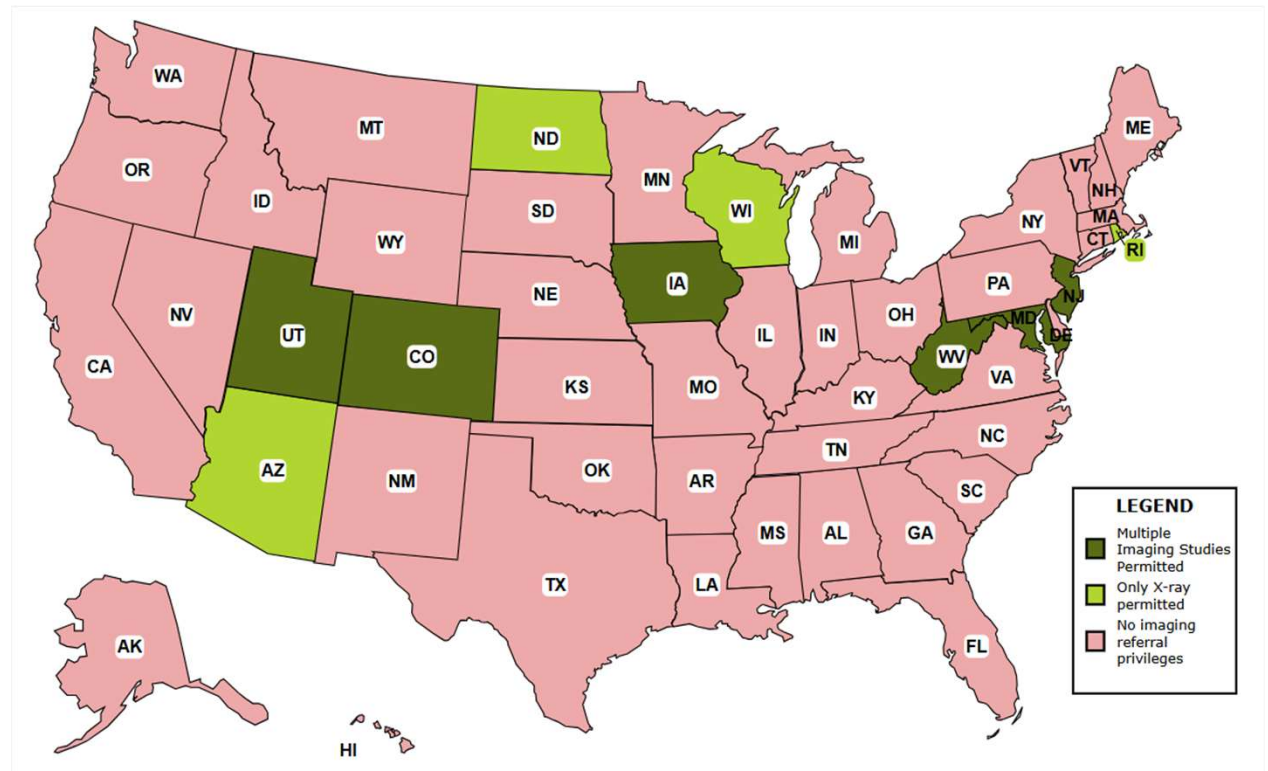
Primary care is the first step, not the last resort

“You may not like it, but this is what peak primary care looks like.”



Beyond the Military

9 states and the District of Columbia allow PTs to refer for diagnostic imaging



Living Below Our Privileges

Rundell et al, 2021

- >90% of DPTs believe they have the knowledge and training to recommend imaging modalities
 - Greater confidence among newer PTs and those with specialist training
 - More confidence in ordering X-ray and MRI than CT or ultrasound
- 79% believe they have the knowledge and skills to directly order plain radiographs
 - 64% confidence for ordering MRI
- 74% feel ordering radiographs would be useful
 - 66% for MRI
 - 54% for US
- Those with Bachelor's or certificate in PT rate ordering CT and US as more useful than DPTs
- Many PTs do not know whether they live in an area with imaging referral privileges

Keil et al, 2019

- Over a 5-year period, 10 PTs in Washington DC saw 503 direct access evaluations
- 63 imaging orders were placed
 - 43 radiographs, 20 advanced images
 - 4% referral rate



Photo by David Solce: <https://www.pexels.com/photo/springtime-at-the-utah-state-capitol-31661816/>

Primary Care in Utah

Effective May 7th, 2025, Senate Bill 196
Amended the insurance code

- 26B-4-310(1).(11)
- (a)"Primary health care" means:
 - (i) basic and general health care services given when a person seeks assistance to screen for or to prevent illness and disease, or for simple and common illnesses and injuries; and
 - (ii) care given for the management of chronic diseases.
- (b)"Primary health care" includes:
 - (i) services of physicians, nurses, physician's assistants, **physical therapists**, and dentists licensed to practice in this state



Photo by David Solce: <https://www.pexels.com/photo/springtime-at-the-utah-state-capitol-31661816/>

Primary Care in Utah

Effective May 7th, 2025, Senate Bill 196
Amended the insurance code

- “an accident and health insurance policy that requires an insured to select a primary care provider or physician to receive optimum coverage **may permit** an insured to select a participating provider who is a **physical therapist** to provide physical therapy services.”

SB-196 is intended to reduce the financial burden of PT services for patients by reclassifying therapists as primary care providers instead of specialists



Rising to the Occasion

- PCP Status can be more than “one weird trick” to reduce cost for patients
- Filling our new role includes providing and referring for diagnostic testing

There's a new referring provider on the block!

Let's get to know them!



Chart review of 125 recent
EMG/NCS patients referred
internally by physical therapists

Photo by Cottonbro studio:
<https://www.pexels.com/photo/photo-of-two-girls-playing-with-stuffed-animals-7462449/>



Comparison with 125 recent
EMG/NCS patients from traditional
medical referrals

Placing the Referral

About 7 EMG/NCS referrals placed companywide per 1000 PT evaluations ($\approx 1\%$)

Number of Therapy Visits Before EDX Referral

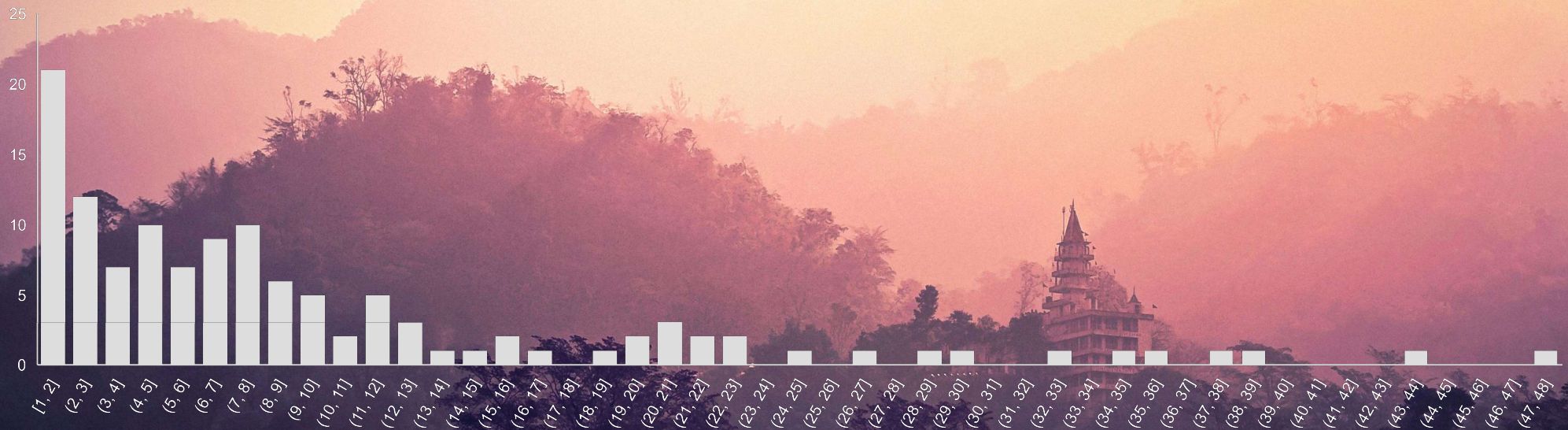
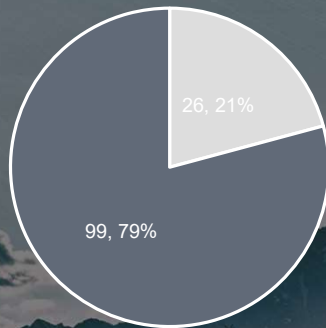


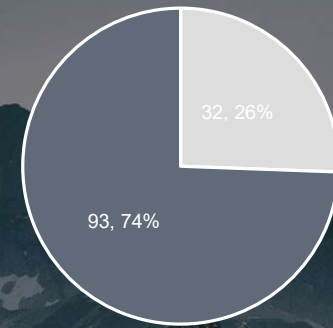
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Medical Referrals



■ Normal Results ■ Abnormal Results

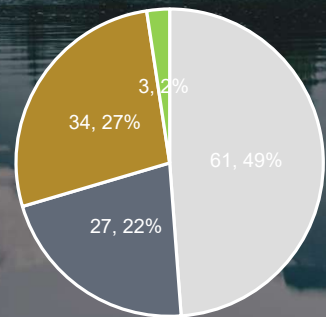
PT Referrals



■ Normal Results ■ Abnormal Results

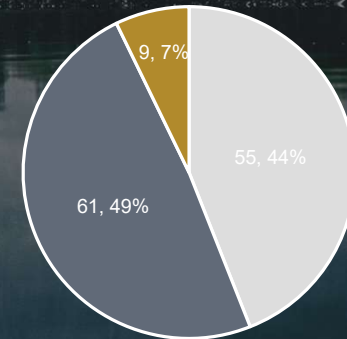
Outcomes

Medical Referrals



■ Referring Diagnosis Confirmed ■ Referring Diagnosis Not Confirmed
 ■ No diagnosis (symptoms only) ■ No diagnosis (extremities only)

PT Referrals

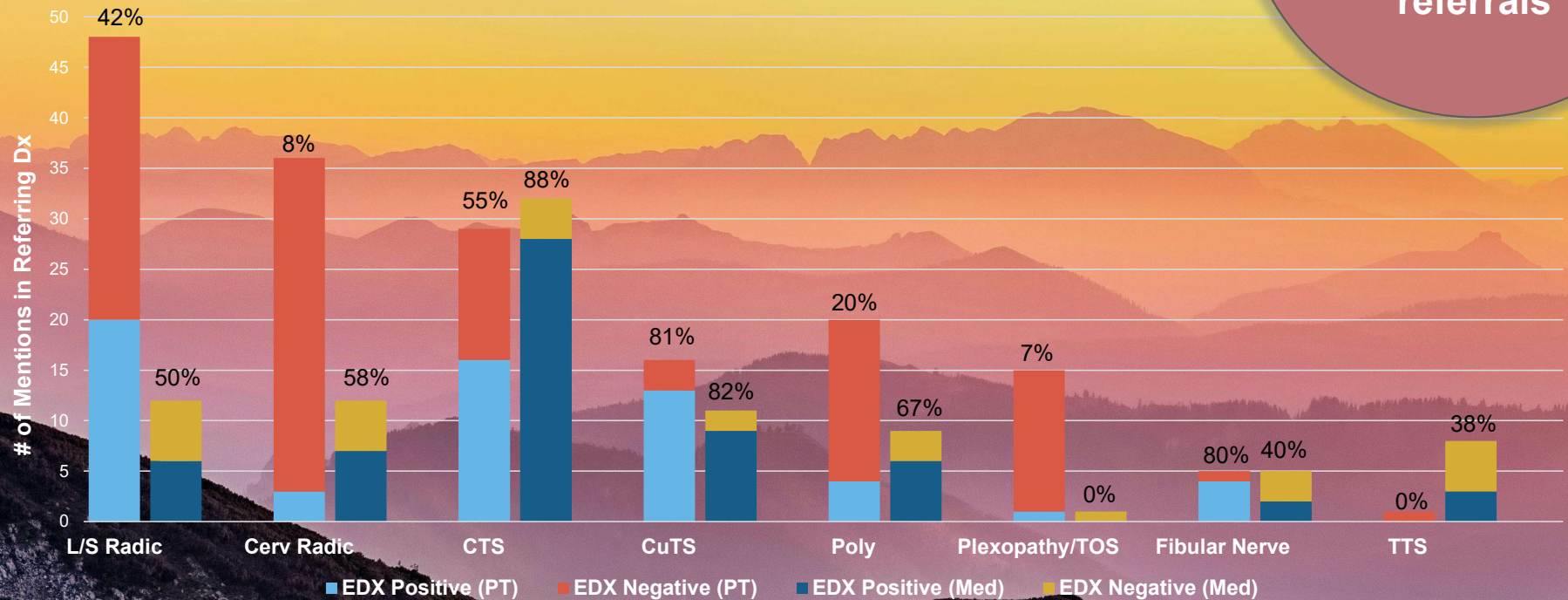


■ Referring Diagnosis Confirmed ■ Referring Diagnosis Not Confirmed
 ■ No diagnosis (symptoms only)

Referring Diagnoses vs. EDX Results

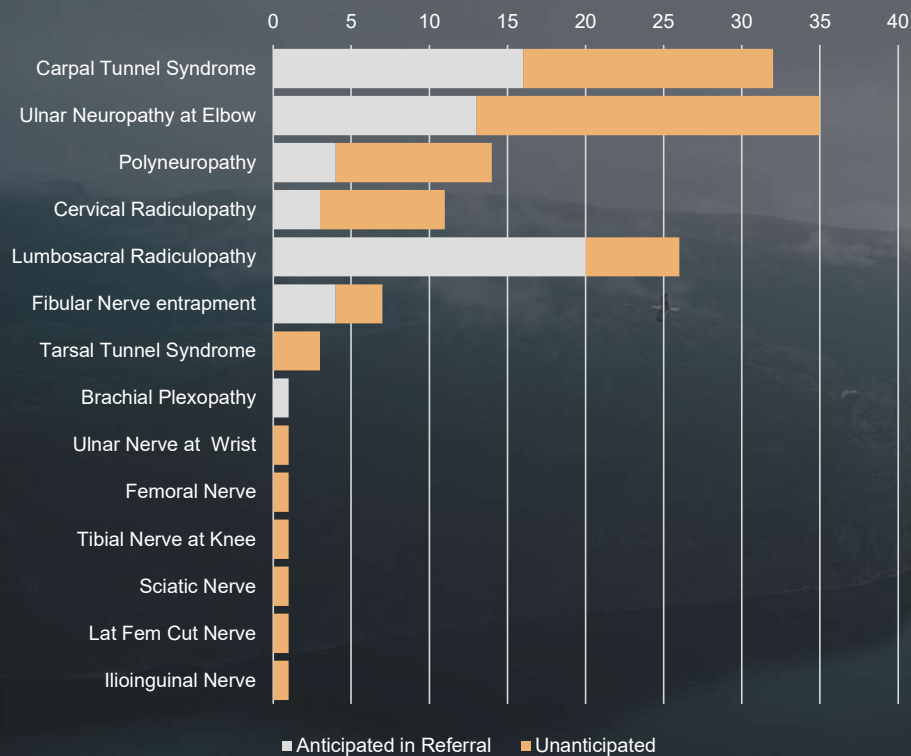
PTs averaged
1.57 diagnoses
per referral,
versus 1.21
from medical
referrals

Referring Diagnosis Volume and Accuracy



Beyond the Referral

Pathologies Observed (PT Referral)



Pathologies Observed (Medical Referral)

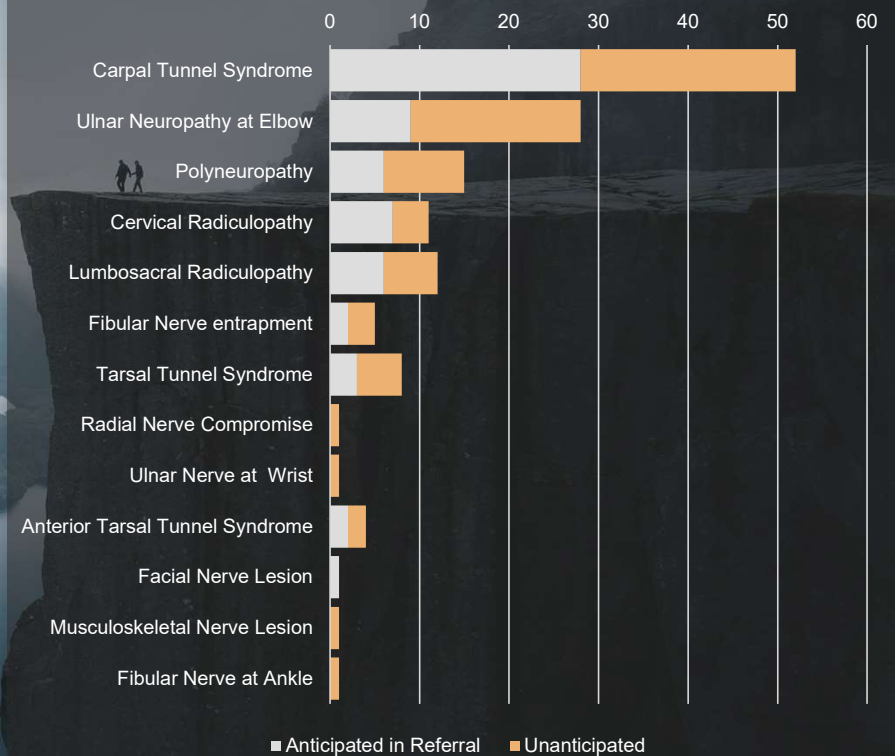


Photo by Valdemaras D.: <https://www.pexels.com/photo/two-people-on-mountain-cliff-1647962/>

“Pt reports she is very sore from EMG yesterday.”

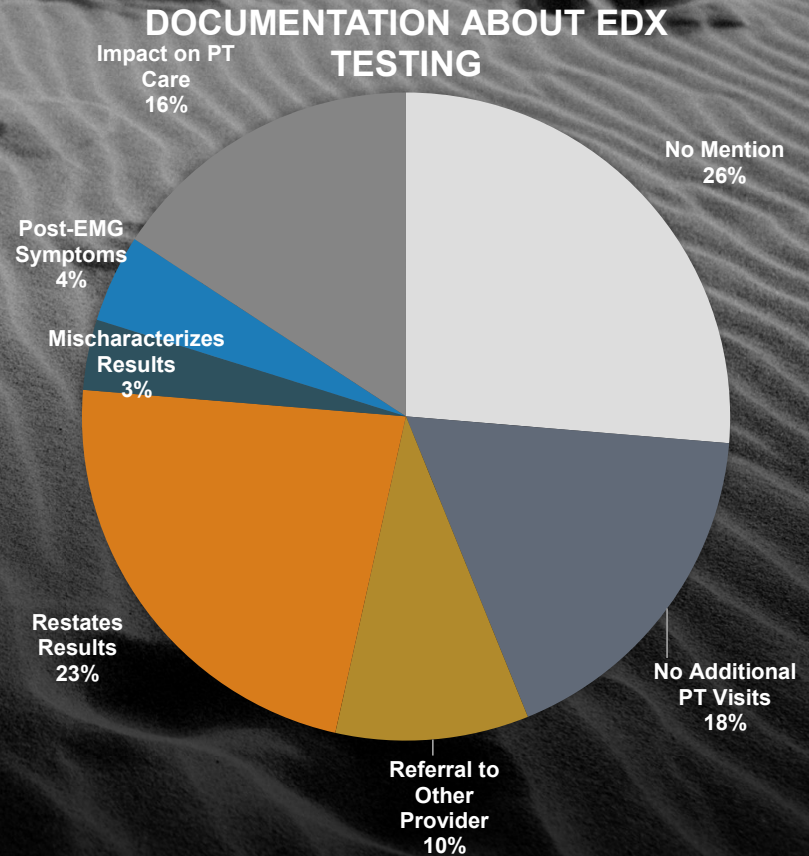
“Pt educated on EMG/NCV results of active on chronic bilateral L5/S1 radiculopathic process. Will continue to monitor sxs.”

“Discussed EMG results and likelihood that current limitations in ROM and function are not isolated to carpal tunnel syndrome but this does need to be resolved to prevent future damage to the nerves. Referred patient to Dr. XXX and recommended continued PT in the interim until surgical date is set.”

“Pt returns with recent EMG results of healthy median nerve and mild ulnar nerve compromise. Possible that pt's continued pain is related to CMC arthritis. Recommended CMC-specific brace worn daily for at least 1 week, then wean down to just aggravating activities.”

Next Steps

Review of chart notes following EDX testing



<https://www.pexels.com/photo/footprints-on-ribbed->



Brynne Gerrard, PT, DPT



Ryan Pope, PT, DPT



Spencer Vickery, PT, DPT

The Names Behind the Numbers

Interviews with top referring therapists

“What value does EMG/NCS testing bring to your practice?”



“I think it’s huge to have that extra test for specificity ...a lot of the screening tests that I typically use will only catch part of it. I send a referral and it’s actually *that* AND *this*. Getting that extra clarity has been really helpful. I think that a lot of patients are also responsive to it. They like to know what’s going on.”



“The more info the better...as much objective information information as we can get is best practice...Physicians do it all the time...they get results from X-rays, MRIs, lab values to see what’s going on with the patient. ...Why don’t we do everything objective possible? That’s going to give the strongest evidence and the strongest care to direct how we should be treating our patients.”



“I find that EMG is very beneficial in helping patients understand what’s going on. I also think it helps validate physical therapy and our skill sets. ...Because of that, you get way more buy in, and you get good conversations with other providers.”

“What makes a good candidate for EMG/NCS testing?”



“If they’re not responding well to treatment...If I had a pretty good plan but they’re still having a lot of numbness and tingling, then I’m instantly referring over.”



“Numbness, tingling, or a burning sensation is a red light of “let’s do a deeper dive and see what’s going on.’ ...I do it during the eval.”



“If it’s something very acute, like after a motor vehicle accident, a fall, or some type of change in symptoms. ...On the other hand, if it’s something more chronic...in that case I want to know how bad of a lesion it is and what damage has been done. ...How can [I] help manage it?”

“What impact do EMG/NCS results have on your care?”



“It’s been helpful to guide treatments, to know how cautious we have to be. ...I really like it to help me hone in and pinpoint where I have to focus. Symptoms can vary widely regardless of the EMG findings. ...If you have a really irritable patient and their EMG findings are also severe, you know you’re going to want to take it slow. On the flip side, maybe they are super irritable symptom-wise, but their EMG is relatively normal, maybe you’re going to lean into pain and neuroscience education.”

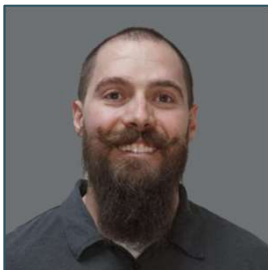


“Usually the results are what I figured, but it’s a comfort and a reassurance to the patient. We live in a day where [patients] need to have the answers right away and need to have concrete answers.”



“The testing results give me more direction in how much I need to emphasize things like nerve glides, avoiding repeated movements, etc. to address the current issue.”

“How comfortable are you with understanding EMG/NCS reports and explaining them to your patients?”



“Not very. I have a really good working relationship with ...the one I’m sending my EMGs to, so if I have any questions I ask him.”



“I’d say pretty comfortable. The terminology is pretty straightforward...I let them know what was impacted and how it may affect their care. ...Is this something that we need a spine surgeon for, do we need further imaging, or is this something totally in our control?”



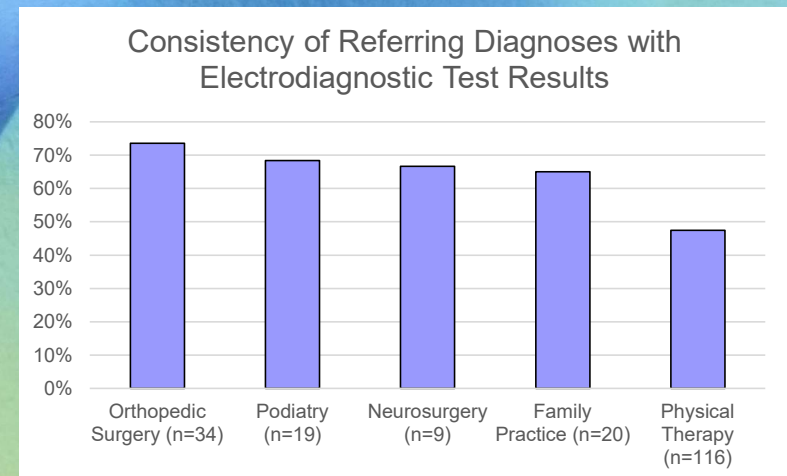
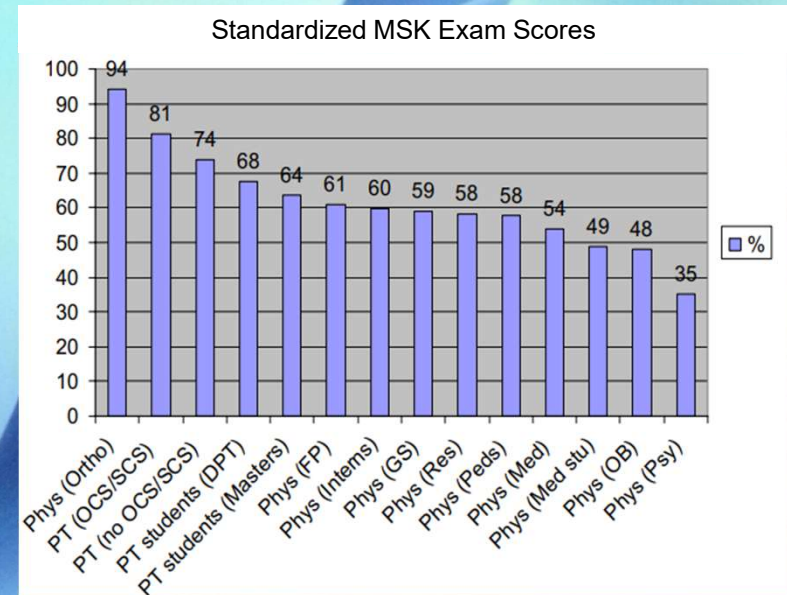
“I probably need a little more guidance. ...I’d like to know how to interpret an abnormal finding, and also what looks normal [on the graphs]. ...I value your interpretations as to what’s going on.”

Takeaways

The Outpatient PT clinic is home to a wide variety of nerve compromises

EMG/NCS testing is a meaningful adjunct to outpatient physical therapy practice

Physical therapists are musculoskeletal experts, but can struggle with diagnosing nerve disorders





The Road Forward

Educating Referral Sources

- Review results together
- Inservices
- Clinical exam training

Breaking the Taboo

- Educate patients directly
- Respect your scope of practice

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ELECTRODIAGNOSTIC EVALUATION OF TRAUMATIC BRACHIAL PLEXOPATHY

Abshire D

ABSTRACT

BACKGROUND: Traumatic brachial plexopathy occurs when high-energy injuries—such as motorcycle crashes, falls, or traction forces to the shoulder—damage the brachial plexus. Because the plexus spans multiple anatomical levels, the pattern of weakness and sensory loss can be complex, making accurate localization difficult. Prompt and thorough evaluation is essential, as outcomes and treatment strategies differ depending on location and severity of injury.

DISCUSSION: Traumatic brachial plexus injuries are challenging to assess because of the plexus's intricate structure, overlapping nerve distributions, and the presence of other traumatic factors that can mask early neurological deficits. Clinical findings often change as the injury evolves and there is limited imaging that clearly defines a brachial plexus lesion. EMG and NCS provide critical clarity by mapping patterns of denervation, helping differentiate plexopathy from radiculopathy or isolated nerve injuries, and using sensory responses—particularly preserved or absent SNAPs—to distinguish preganglionic from postganglionic involvement. These studies offer essential insight into injury severity, expected recovery, and the most appropriate surgical or conservative treatment approach.

CONCLUSIONS: EMG/NCS remains an essential component in the diagnostic workup of traumatic brachial plexopathies, offering precise physiologic information that complements clinical examination and imaging. By accurately localizing lesions, identifying the degree of axonal injury, and distinguishing plexus involvement from other neurologic conditions, electrodiagnostic testing provides critical guidance for treatment planning and prognosis. A systematic, well-executed EMG/NCS assessment ensures timely decision making and supports optimal outcomes for patients with complex brachial plexus injuries.

Dylan Abshire, DPT, ECS

ELECTRODIAGNOSTIC EVALUATION OF TRAUMATIC BRACHIAL PLEXOPATHY

Dr. Abshire, DPT, ECS

Slide 1

DA13

Hi im Dr. Abshire I will be giving this presentation on traumatic brachial plexopathy.

Dylan Abshire, 3/8/2026



- Nerve Clinic 4 orthopedic surgeons, 1-2 orthopedic surgery fellow and 1 neurophysiologist.
- Half day with all doctors rounding on patients who are referred for complex nerve injuries or cases.
- BPI, Lacerations, Limb Loss, Neuromas



Slide 2

DA11 My practice is with the Indiana hand to shoulder center I work with 10 orthopedic surgeons and I also get outside referrals for EMG so I am roughly 70/30 upper to lower extremities. I only practice electrophysiology no PT.

At our practice we have yearly orthopedic hand fellows and I teach EMG didactic for them yearly. Additionally our practice has a nerve program which is once a month me and 4 surgeons and a fellow do case rounds on complex nerve injuries that get sent to from the surrounding areas.

Dylan Abshire, 3/8/2026

Traumatic Brachial Plexus Learning Objectives

By the end of this lecture, participants should be able to:

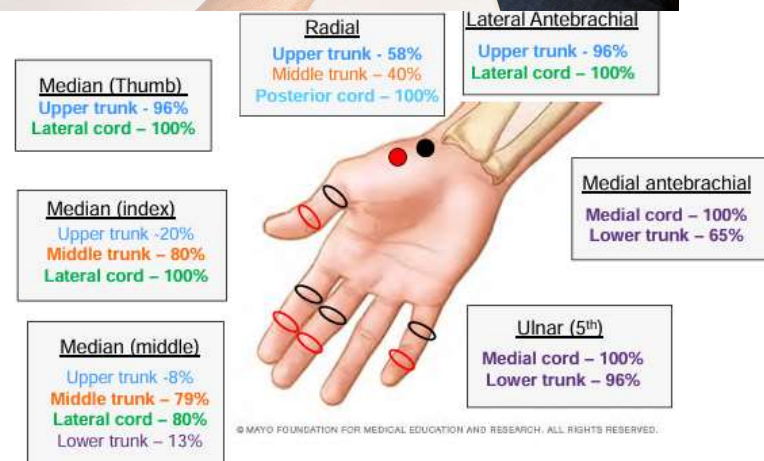
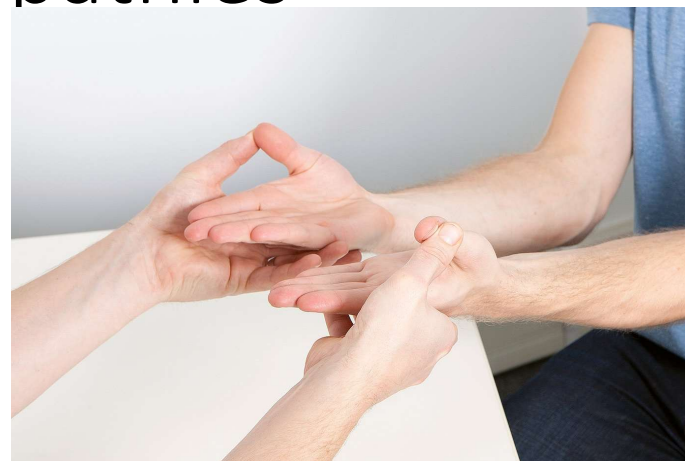
- Evaluating brachial plexus anatomy for traumatic localization and mechanism of injury
- Apply electrodiagnostic strategies for traumatic plexopathy
- Localize lesions using a structured root → trunk → cord → terminal nerve approach
- Recognize prognostic indicators and surgical timing considerations



- DA6** Traumatic brachial plexus injuries represent some of the most complex and challenging conditions we encounter in electrodiagnostics. This presentation will be more focused on injuries that occur in high-energy trauma and can result in devastating functional loss of the upper extremity. For clinicians involved in the evaluation of these patients, accurate localization of the lesion and early identification of prognostic indicators are critical, because they directly influence surgical decision-making and long-term outcomes. In this talk, I'll review the clinically relevant anatomy of the brachial plexus, discuss how we differentiate pre-ganglionic versus post-ganglionic lesions, and walk through the electrodiagnostic strategies that I use when evaluating traumatic plexopathies.
Dylan Abshire, 3/8/2026
- DA10** MVA/GSWs/Falls
Dylan Abshire, 3/8/2026

Evaluating Traumatic Plexopathies

- **Electrodiagnostic studies should always be considered an extension of your physical exam**
- **Hand Muscle testing- Mandatory**
- Thumb Opposition- Thenar APB/OP- Median- medial cord/lower trunk/ C8/T1 Roots.
- Thumb Retropulsion- EPL/EPB- PIN- posterior cord- C7-C8 nerve root
- Thumb IP Flexion- AIN- C7-C8
- IF Abduction- FDI- Deep ulnar motor- C8/T1
- Hypothenar- SF Adduction C8/T1
- **Sensory examination paired with motor**
 - **2 point discrimination**
 - **Light touch**
 - **Tuning fork**



Ferrante & Wilbourn. Muscle and Nerve 1995.

Slide 4

DA14 Every EMG study should begin with a very thorough physical exam. Every textbook highlights the fact that electrodiagnostics are an extension of the physical exam. So thorough and complete sensory motor examination is always warranted.

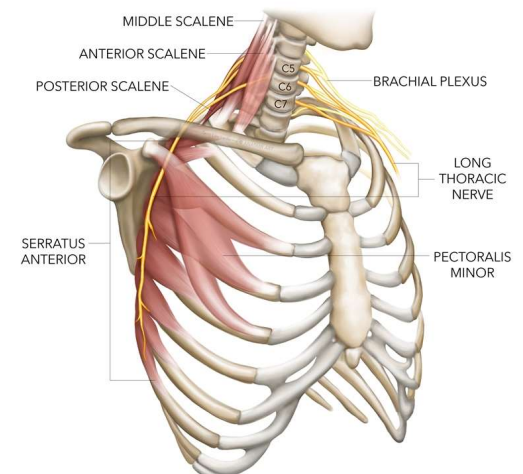
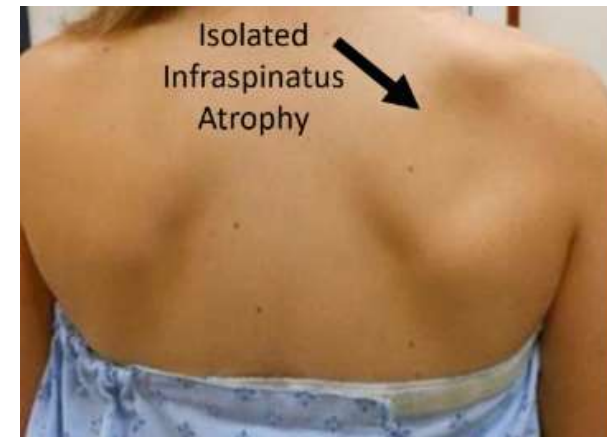
Dylan Abshire, 3/8/2026

DA15 The hand is incredibly important in helping distinguish pathology as they hand intrinsic and intrinsics will weaken much easier than the strong upper extremity muscles. Additionally sensory distributions give us key information to pair with motor loss.

Dylan Abshire, 3/8/2026

Evaluating Traumatic Plexopathies

- MMT continued..
- Upper arm larger muscles to gauge with MMT.
- Wrist flexion/Extension-
- Elbow Flexion/Extension
- Shoulder abduction/Flexion-
- Shoulder ER/IR
- Scapular winging
- CNXI



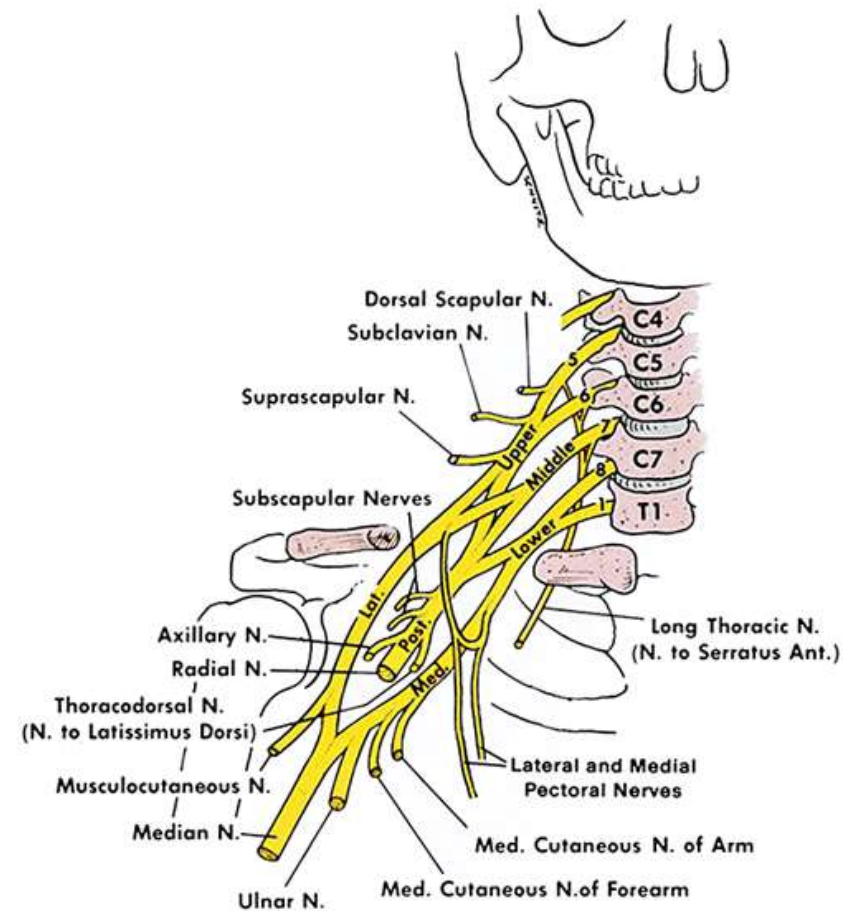
Slide 5

DA16 Continued evaluation I always assess patient with shirt off so I can evaluate scapular region compared to asymptomatic limb. Can see SS atrophy, Lateral/Medial winging, Trapezius atrophy. All of those nerves are motor innervated so can be easily missed.

Dylan Abshire, 3/8/2026

Plexus Anatomy- Pre-Trunk->Roots

- Plexus formed from C5-T1 anterior rami, posterior rami innervated paraspinals. DA7
DA8
- C4/C5- Dorsal Scapular N.
- C3-C5- Phrenic N.
- C5-C7- Long Thoracic Nerve
- Individual Roots/Paraspinals- Posterior Rami
- Consider CNXI in pre-trunk assessment



Slide 6

DA7 Several key nerves arise directly from the roots before trunk formation. The dorsal scapular nerve, typically from C5, innervates the rhomboids. The long thoracic nerve, from C5 to C7, supplies the serratus anterior and is important when evaluating scapular winging.

We should also remember the phrenic nerve, arising from C3 to C5, which may be involved in severe traction injuries. And clinically, it's important to assess the spinal accessory nerve, CN XI, in the pre-trunk region because trapezius dysfunction can sometimes mimic or coexist with upper plexus pathology."

Dylan Abshire, 3/8/2026

DA8 Root Avulsion/

Dylan Abshire, 3/8/2026

Long Thoracic N DA9

- Long thoracic nerve, C5–C6–C7- Arises directly from the roots.
- Innervating Serratus Anterior.
- Chest wall or supraclavicular

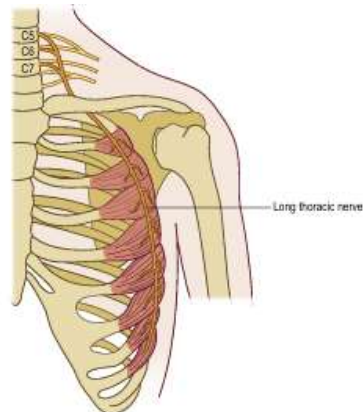


Fig. 34.9 Anatomy of the long thoracic nerve. The long thoracic nerve originates directly from the C5, C6, and C7 roots, proximal to the brachial plexus proper. It runs inferiorly to supply the serratus anterior muscle. There is no cutaneous sensory innervation. (Reprinted with permission from Fisher M. Other mononeuropathies of the upper extremity. In: Brown WF, Bolton CF, eds. *Clinical Electromyography*. 2nd ed. Boston, MA: Butterworth; 1993: 271.)



Slide 7

DA9

Motor nerve directly from the roots. Can be easily missed if not evaluating with shirt off. Can become damaged supraclavicularly or at the chest wall

Dylan Abshire, 3/8/2026

Spinal Accessory N

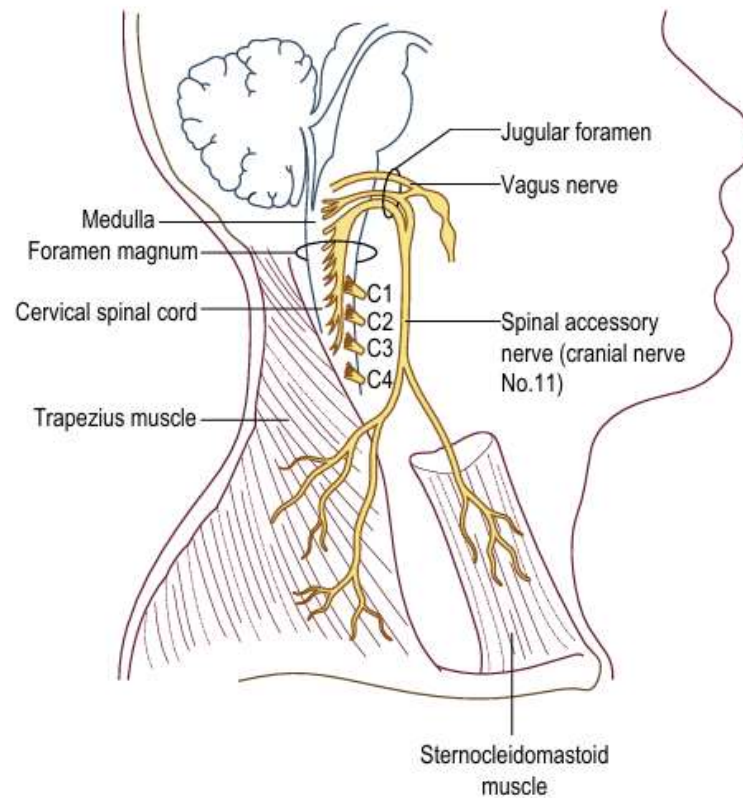
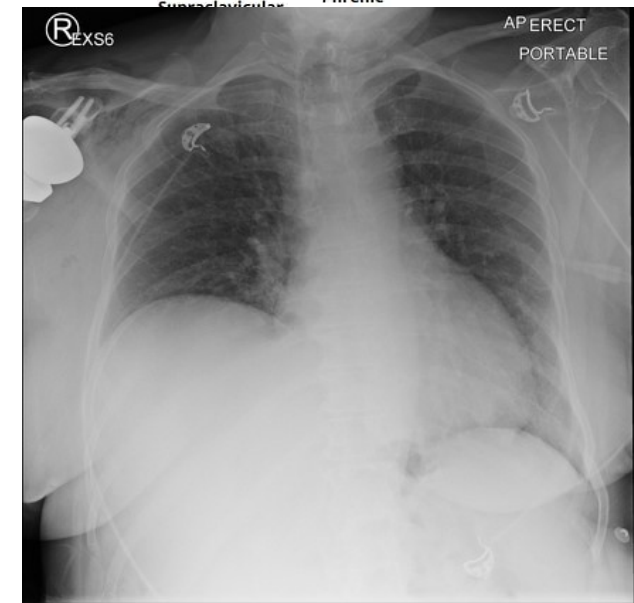
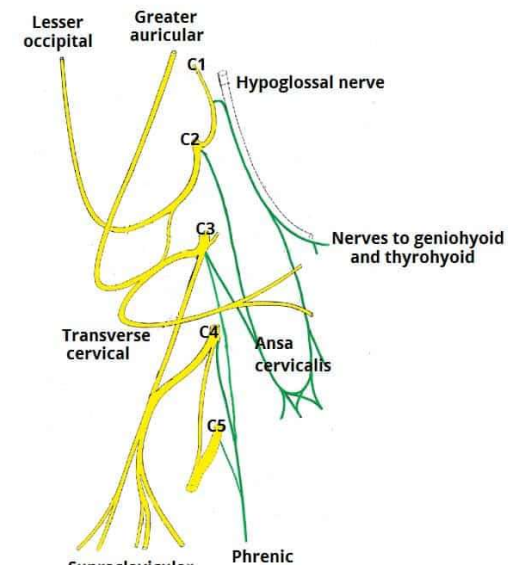


Fig. 34.10 Long thoracic neuropathy. Isolated weakness of the serratus anterior occurs in long thoracic neuropathies. Serratus anterior weakness results in "winging" of the scapula. Winging becomes most pronounced with the arm extended in front of the body. As the serratus anterior normally pulls the scapula forward, weakness of the serratus anterior results in the scapula being displaced closer to the midline. In this figure, a red circle marks the tip of each scapula. Note the left scapula is displaced closer to the midline than the right scapula.

Phrenic N

- **Nerve roots** – anterior rami of C3, C4 and C5.
- **Motor functions** – innervates the [diaphragm](#).
- **Sensory functions** – innervates the central part of the diaphragm, the pericardium and the mediastinal part of the parietal pleura.



Phrenic Motor Study

Nerve	Record	Amplitude (μ V)	Distal Latency (ms)
Phrenic	Diaphragm	597 ± 139 >320	6.3 ± 0.8 <8.0

From Markand ON, Kincaid JC, Pourmand RA, et al. Electrophysiologic evaluation of diaphragm by transcutaneous phrenic nerve stimulation. *Neurology*. 1984;34:606-614.

Slide 9

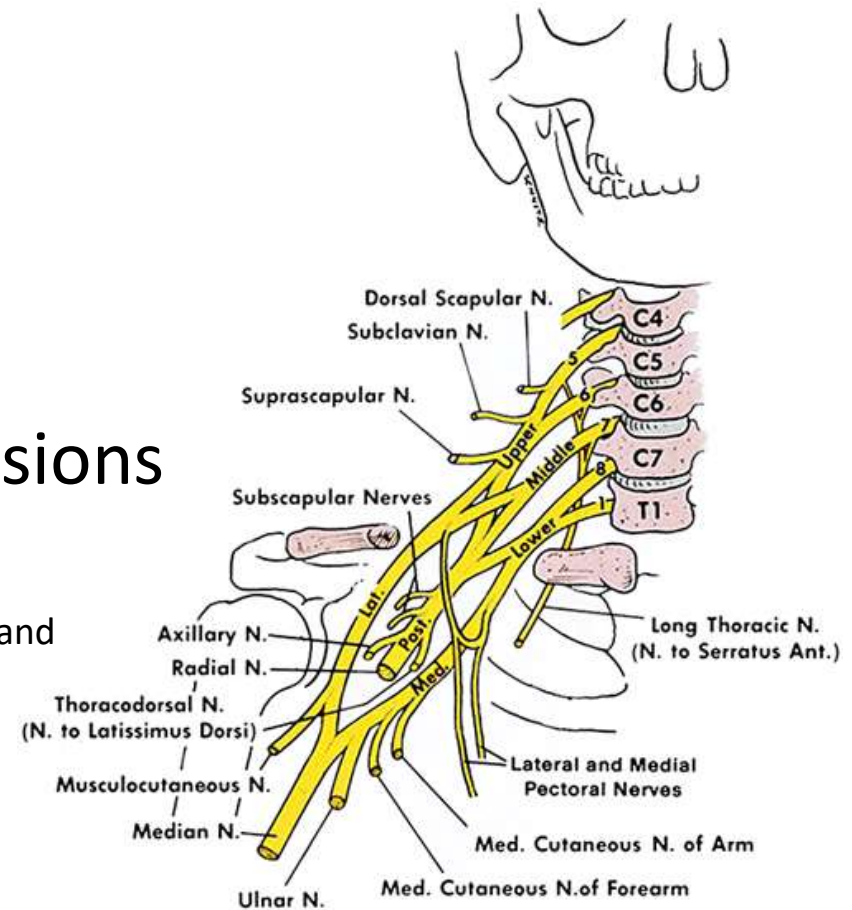
DA12 right phrenic nerve. Flaccid diaphragm.
Dylan Abshire, 3/8/2026

Plexus Anatomy- Roots -> Trunks

- C5/C6- Upper trunk
- C7- Middle Trunk
- C8-T1- Lower Trunk

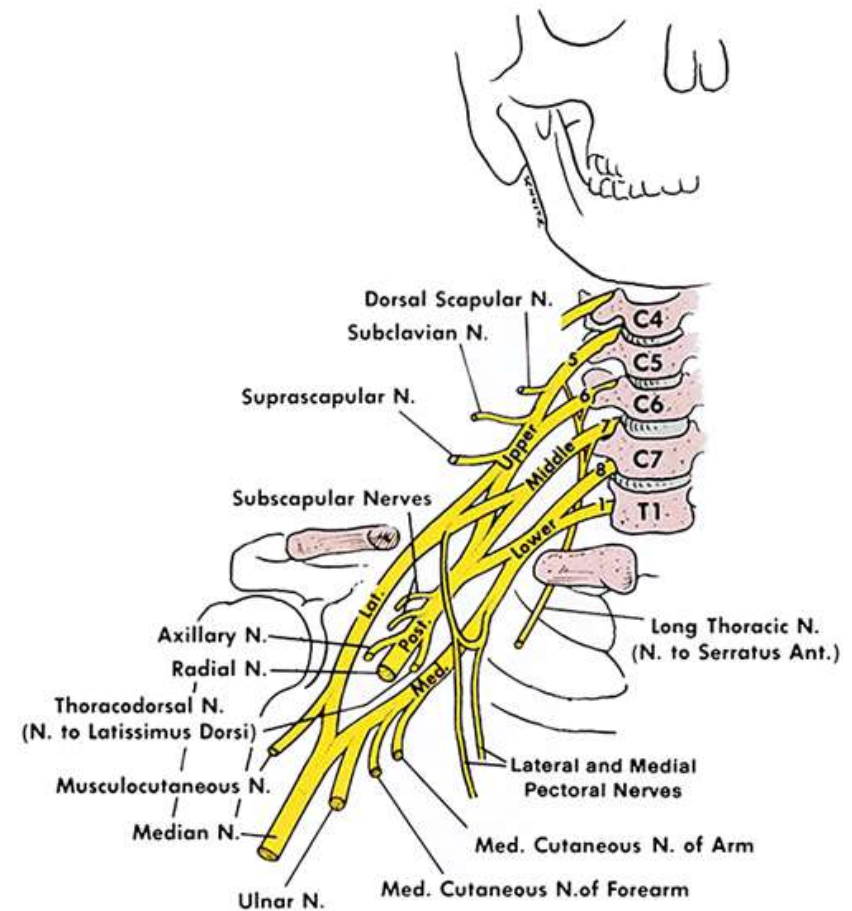
Plexus Anatomy- Trunks to Divisions

- Trunks then divide into divisions anterior and posterior.
This is where the BP differentiates between supraclavicular and infraclavicular



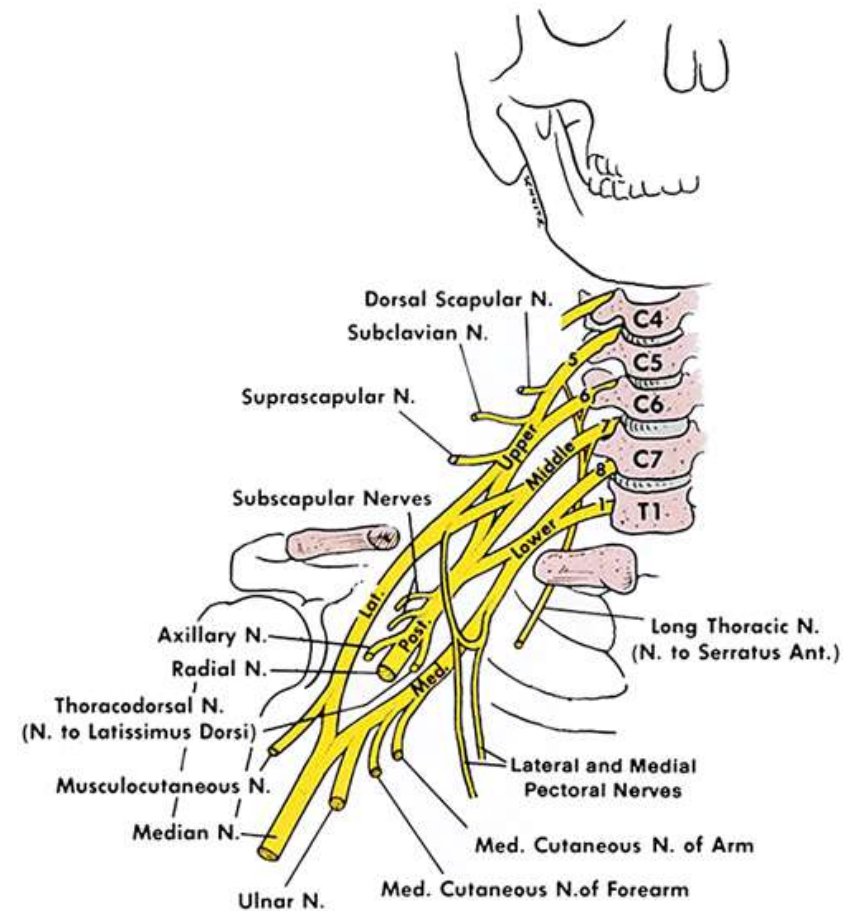
Plexus Anatomy- Divisions -> Cords

- Posterior cord formed by all 3 divisions C5-T1
- Lateral cord formed by anterior division of upper and middle trunk C5-C7
- Medial cord continuation of lower trunk C8-T1



Plexus Anatomy- Cords -> Nerves

- Lateral Cord- Musculocutaneous, Median S, LABC
- Posterior Cord- Axillary and entire radial n pathway
- Medial cord- Median M, Entire ulnar nerve pathway and MABC



Electrodiagnostic Evaluation Strategy

- **Where to start NCS?**
- Evaluate based on physical exam finding
- Standard NCS Screen:
 - Median sensory antidromic MF
 - Ulnar sensory antidromic SF
 - Radial Sensory antidromic dorsal compartment
 - Median M APB & Ulnar motor ADM
- Advanced NCS sensory screen
 - LABC/MABC
 - Volar thumb/ All digits to isolate different plexus divisions
- Advanced Motor Screen
 - Axillary & Musculo NCS
 - SSN NCS
 - Phrenic NCS

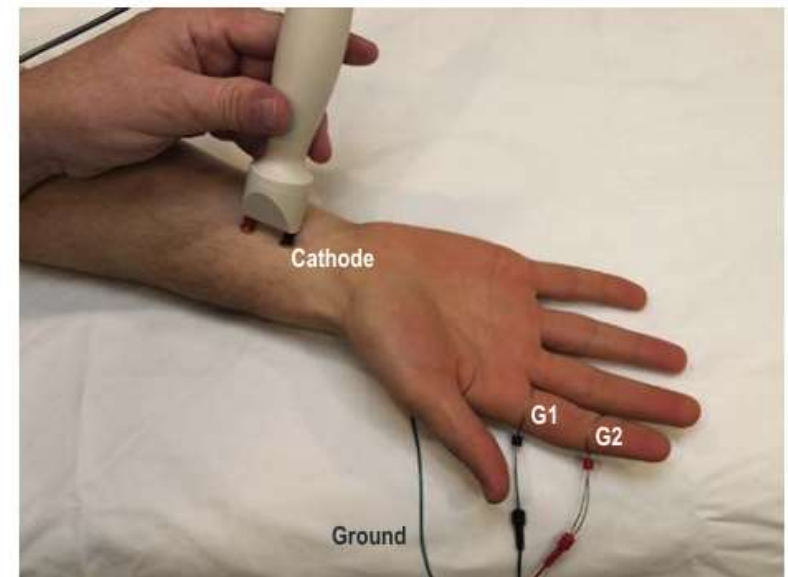


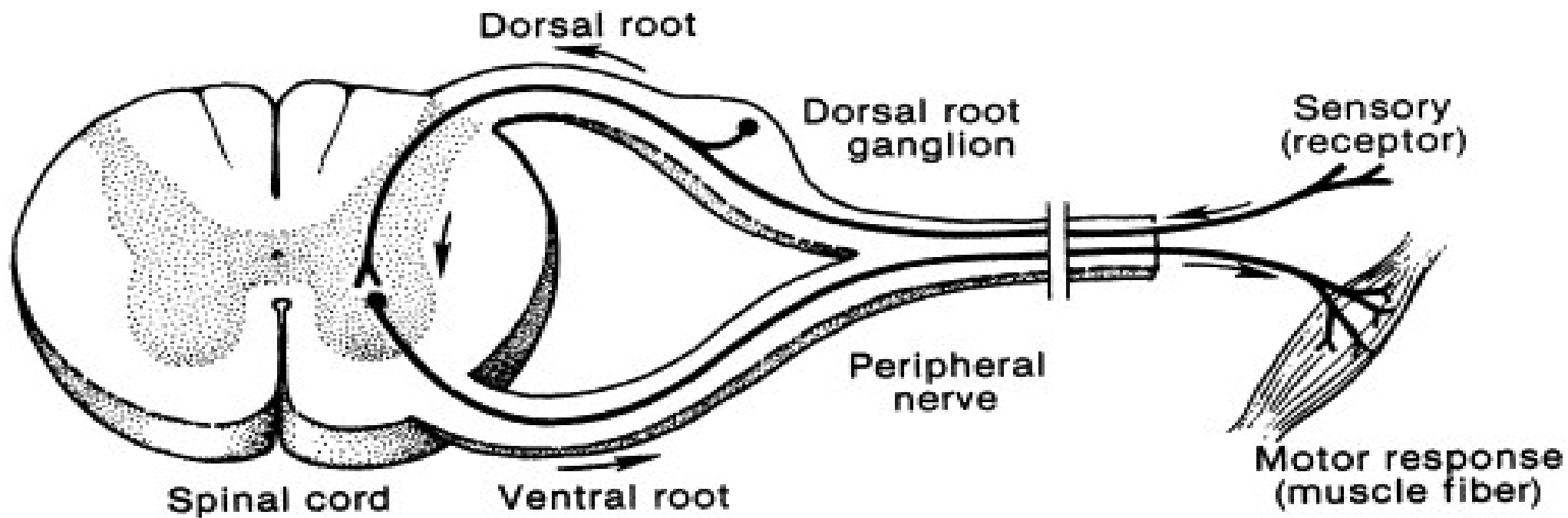
Fig. 10.3 Median sensory study. Stimulation site over the median nerve at the wrist, recording the index finger.

Slide 13

DA21 again going back to the type of injury and some of these nerves are motor only so difficult to delineate Post-pre ganglionic
Dylan Abshire, 3/12/2026

Pre Vs Post-Ganglionic lesion

- The DRG forms just distal to the intervertebral foramen. The dorsal root ganglion more recently referred to as the spinal ganglion, is **a collection of neuronal cell bodies of sensory neurons**. This is highly adventitious to EMGers as it provides information on whether a lesion is occurring pre-post ganglia.



Slide 14

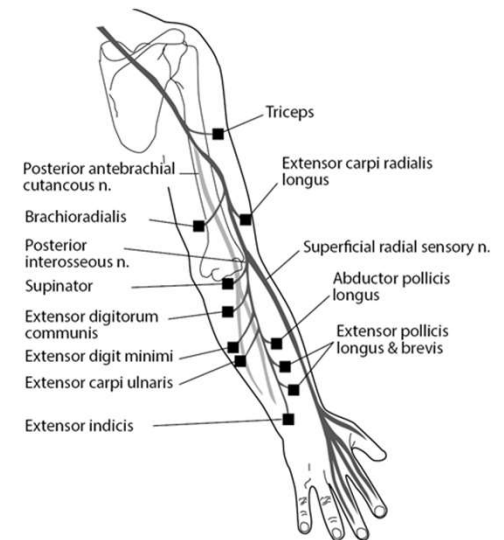
DA20 pre vs post ganglionic first temp in recognizing BP lesions
Dylan Abshire, 3/12/2026

DA22 Post ganglionic lesion indicating BPI--- this is true but in some cases its motor only or its root avulsed so the
snaps are deteriorated and the paraspinals are denervated
Dylan Abshire, 3/12/2026

Pre vs Post-ganglionic lesion

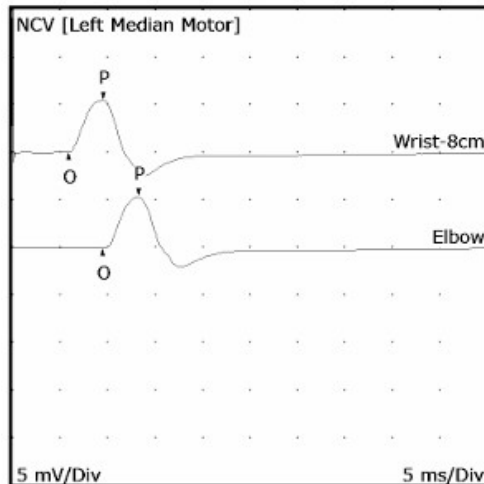
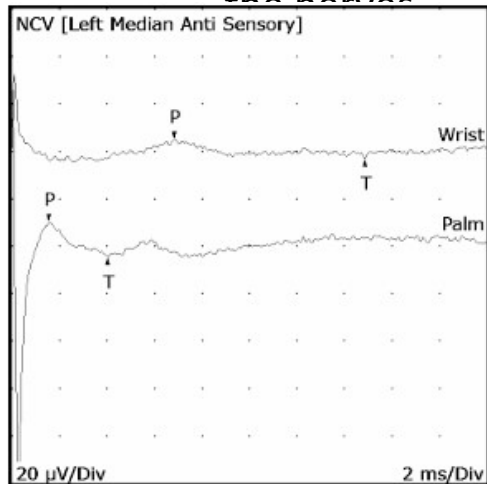
Latency ↑ → Myelin problem
 Amplitude ↓ → Axon problem
 Normal SNAP but abnormal
 EMG → Root lesion (proximal to
 DRG)
 Abnormal SNAP →
 Peripheral/plexus lesion (distal
 to DRG)

Structure	Location Relative to Dorsal Root Ganglion (DRG)	Contains
Preganglionic (root)	Proximal to DRG (spinal root level)	Motor fibers + sensory central processes
Postganglionic (peripheral nerve)	Distal to DRG (plexus, nerve trunks, etc.)	Motor fibers + sensory peripheral processes



- **Sensory NCS** measures **peripheral sensory axons**, which are **distal to the DRG**.
- The **cell body of the sensory neuron** resides in the **DRG**, not in the root or spinal cord.
- “If DRG is spared → sensory NCS is spared.”

- Order of fiber deterioration
 - Sensory fibers will deteriorate first as they are smaller diameter fibers and lay more peripheral
 - Motor fibers are larger fiber and lay more centrally within the nerve



Left Median Anti Sensory (3rd Digit)

Wrist		*6.8	<3.6	*8.2	>10	Wrist	3rd Digit	6.8	14.0	21	
Palm		1.6	<2.5	14.7	>10						

Left Median Motor (Abd Poll Brev)

Wrist-8cm		*5.9	<4.2	5.5	>3	Elbow	Wrist-8cm	3.6	22.0	61	>48
Elbow		9.5		5.5							

- Where to start EMG
- 26 g concentric
 - Screen peripheral nerve and roots
 - Deltoid, Bicep, Tricep, Pronator, FDI standard screen.
 - Then target areas of weakness or that correlate with physical exam and NCS findings.
 - Keep in mind atypical nerves that could be compromised in trauma cases. IE- motor nerves that give no sensory deficits only weakness.

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EMG

	Upper trunk (C5-6)	Middle trunk (C6-7-8)	Lower trunk (C8-T1)
Pre-cord	Supraspinatus (Sc) Infraspinatus (Sc)		
Lateral cord	Biceps (Mu) Brachialis (Mu)	Pronator teres (Me) FCR (Me)	
Posterior cord	Deltoid (A) Teres minor (A) Brachioradialis (R) Supinator (R) Triceps (R)	Triceps(R) Anconeus(R) ECR(R) EDC(R) ECU(R)	EIP(R) ECU(R)
Medial cord			APB (Me) FPL(Me) FDI (U) ADM(U) FCU(U) FDP(Me & U)

Daube JR, Rubin DI. Clinical Neurophysiology 3rd Ed. 2009

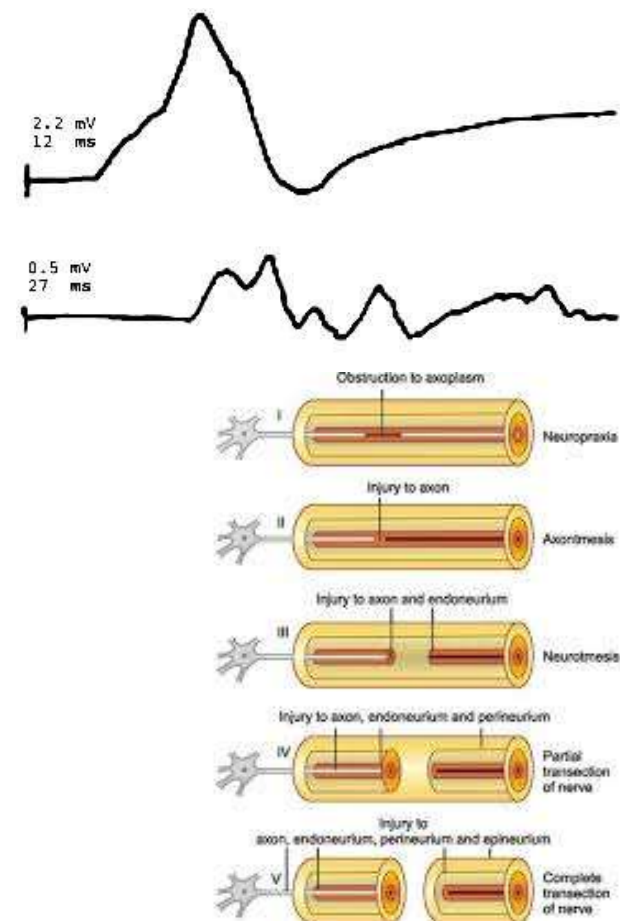
Nerve conduction studies table

	Upper trunk (C5-6)	Middle trunk (C6-7)	Lower trunk (C8-T1)
Pre-cord	Suprascapular		
Lateral cord	LABC Median sensory (I&II) Musculocutaneous	Median sensory (II&III)	
Posterior cord	Radial sensory Axillary	Radial sensory Radial motor	
Medial cord			Ulnar sensory DUC MABC Median motor Ulnar motor

Daube JR, Rubin DI. Clinical Neurophysiology 3rd Ed. 2009

Location to Nerve injury classifications

- Neuropraxia
- Axonotmesis
- Neurotmesis
- Root avulsion (most severe)
- **Real World Clinical emphasis:**
- **Conduction block across lesion site**
- Distal CMAP amplitudes
- BP injury with cervical paraspinal could indicate denervation.



Severity and prognosis depend more on **degree of axonal injury and avulsion** than mechanism alone.

Slide 19

DA19 once we have determined the location we can determine severity of location
Dylan Abshire, 3/12/2026

Case-1 BPI

- 42-year-old male presents 6 weeks after a motorcycle collision. Mechanism involved violent shoulder abduction with traction with associated dislocation. No humeral fracture. He reports progressive upper arm weakness and numbness along the upper lateral aspect of the arm.
- **Physical Examination**
- **Motor**
- Deltoid: 1/5
- Biceps: 1/5
- ER- 1/5
- Triceps: 5/5
- FDI: 5/5
- ADM: 5/5
- APB: 5/5
- EPL5/5
- Wrist extensors: 5/5
- Trapezius- 5/5
- No lower extremity symptoms.



Nerve Conduction Studies
Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	P-T Amp (µV)	Norm P-T Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Lat Ante Brach Cutan Anti Sensory (Lat Forearm)											
Lat Biceps	*NR					Lat Biceps	Lat Forearm		0.0		
Right Med Ante Brach Cutan Anti Sensory (Med Forearm)											
Elbow		3.1		18.4		Elbow	Med Forearm	3.1	0.0		
Right Median Anti Sensory Run #1 (3rd Digit)											
Wrist		3.6	<3.6	15.2	>10	Wrist	3rd Digit	3.6	14.0	39	
Right Median Anti Sensory Run #2 (1st digit volar thumb)											
Wrist	*NR		<3.6		>10	Wrist	1st volar thumb		14.0		
Right Radial Anti Sensory (Base 1st Digit)											
Wrist		1.9	<3.1	*9.9	>15	Wrist	Base 1st Digit	1.9	8.0	42	
Right Ulnar Anti Sensory (5th Digit)											
Wrist		3.5	<3.7	19.0	>15.0	Wrist	5th Digit	3.5	14.0	40	

Motor Summary Table

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Axillary Motor (Deltoid)											
Clavicle		4.3	<5	*1.9							
Right Median Motor (Abd Poll Brev)											
Wrist-8cm		3.8	<4.2	6.3	>3	Elbow	Wrist-8cm	4.4	22.0	50	>48
Elbow		8.2		6.2							
Right Ulnar Motor (Abd Dig Min)											
Wrist-8cm		3.0	<4.2	9.1	>3	B Elbow	Wrist-8cm	4.3	22.0	51	>48
B Elbow		7.3		8.4		A Elbow	B Elbow	1.3	8.0	62	>48
A Elbow		8.6		8.3							
Right Musculocutaneous Motor (Bicep)											
Clavicle		4.8	<5	2.0							

EMG



Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	Abd Poll Brev	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	PronatorTeres	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Biceps	Musculocut	C5-6	*Incr	*4+	*4+	*Decr	Nml	*4+	Nml	Nml	
Right	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Deltoid	Axillary	C5-6	*Incr	*4+	*4+	*Decr	Nml	*4+	Nml	Nml	
Right	BrachioRad	Radial	C5-6	*Incr	*4+	*4+	*Decr	Nml	*4+	Nml	Nml	
Right	Infraspinatus	SupraScap	C5-6	*Incr	*4+	*4+	*Decr	Nml	*4+	Nml	Nml	
Right	LatisDorsi	ThoracoDors	C6-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Trapezius	SpinalAcc	CN XI, C3-4	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Rhomboid Major	DorsalScap	C5	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	



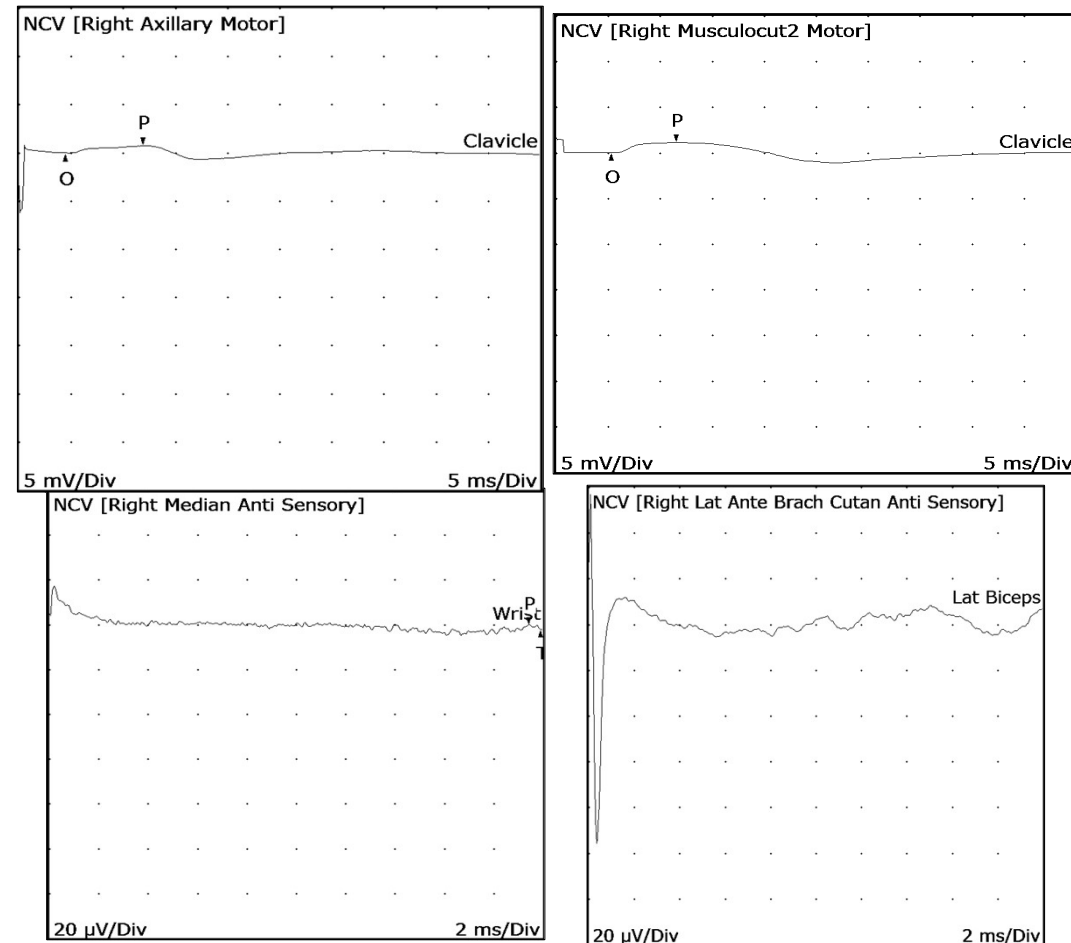
Upper Trunk Plexopathy



NCS Upper trunk findings---

- Axillary & Musculocutaneous nerve amplitude loss
- Median digit 1 sensory loss, LABC Snap loss and radial sensory snap loss

EMG-Fibs/PSWs – Low amp polyphasia
Deltoid, Bicep, BR, IF

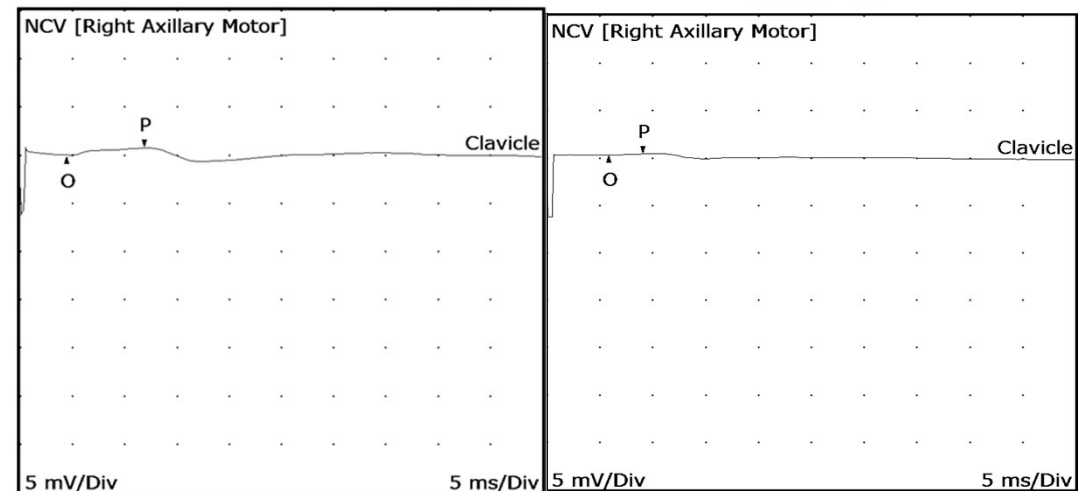
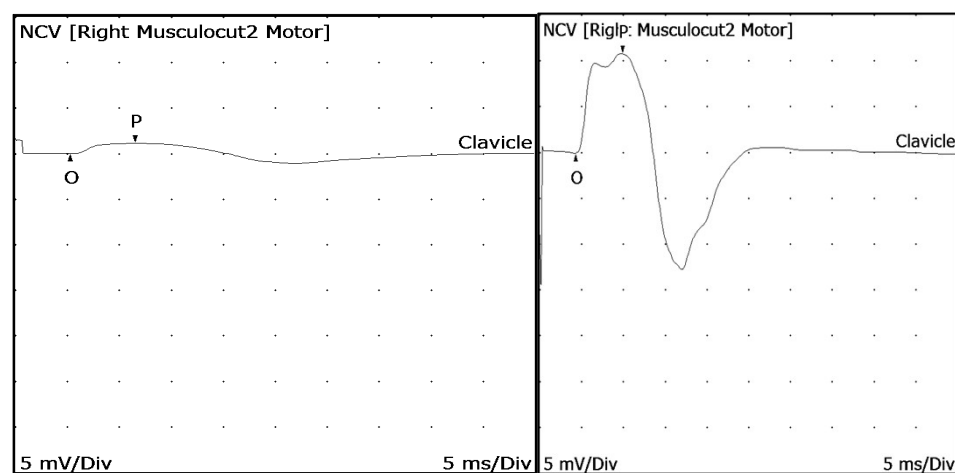
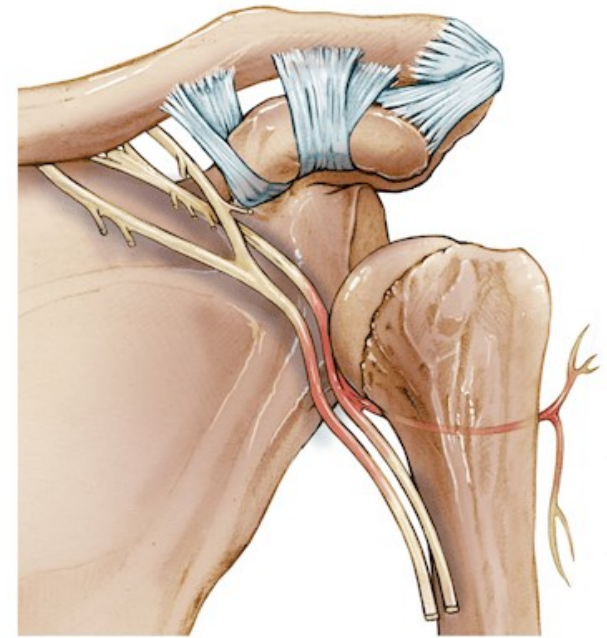


Slide 23

DA23 upper trunk plexopathy low amps with supraclavicular stim to deltoid and musculo
Dylan Abshire, 3/12/2026

Importance of Serial EMG

- In traumatic upper-plexus injuries, the axillary nerve can often demonstrate disproportionately severe involvement compared with other C5–C6 muscles. This likely reflects its fixed course around the surgical neck and vulnerability to traction and compression, and it is reported as one of the most commonly affected nerves in traumatic shoulder and plexus injuries.
- Axillary nerve importance- Essentially the entire control of the shoulder. If you lose the deltoid there is few available options to restore functional GH motion.
- Axillary N. Loss prevents a host of available surgeries for shoulder pain. Most surgeons won't perform total shoulders when there is no functional deltoid.
- Serial EMG- usually every 3 months to track CMAP



DA24 Serial EMG importance in high energy trauma
Dylan Abshire, 3/12/2026

BPI 1 Case conclusion

- **1. There is evidence of an upper trunk brachial plexus lesion involving the sensory and motor fibers to the right upper extremity.**
- **Upper trunk plexopathy → SNAPs reduced**
- **C5/C6 radiculopathy → SNAPs preserved**
- **Best sensory study:**
Lateral antebrachial cutaneous SNAP
If reduced → strongly supports upper trunk/post-ganglionic lesion.
- **Paraspinals normal**
- **Perform serial EMGs if need to assess CMAP amplitude improvement.**

Slide 25

DA25 upper trunk plexopathy LABC one of the best sensory nerves to study.
Dylan Abshire, 3/12/2026

Case 2 BPI

- 38-year-old male presents for electrodiagnostic evaluation following a high-speed motorcycle accident three months prior. He reports persistent weakness with shoulder abduction and difficulty extending the wrist and fingers. He describes numbness along the posterior arm and dorsum of the hand. He denies significant intrinsic hand weakness or numbness in the ring and small fingers. No prior cervical spine surgery. No lower extremity neurologic symptoms.

Case 2 BPI

- **Physical Examination**

- **Motor**

- Deltoid: 3/5
- Triceps: 3/5
- Wrist extensors: 1–2/5
- Finger extensors: 1/5
- FDI: 5/5
- APB: 5/5
- Biceps: 5/5

- **Atrophy**

- Mild posterior deltoid atrophy

- **Sensation**

- Diminished along posterior arm
- Dorsum of hand decreased sensation
- Median and ulnar digits largely intact

- **Reflexes**

- Triceps reflex diminished

Nerve Conduction Studies

Sensory

- Radial SNAP: reduced amplitude
- Median SNAP: normal
- Ulnar SNAP: normal
- LAC SNAP: normal
- MAC SNAP: normal

Motor

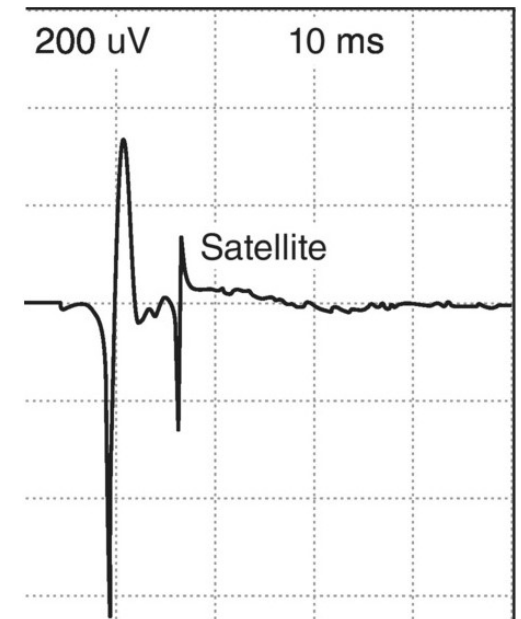
- Radial motor (EIP/EDC): low amplitude
- Axillary motor: reduced
- Median motor: normal
- Ulnar motor: normal

- **CASE 2**
- **Nerve Conduction Studies**
- **Sensory**
- Radial SNAP: reduced amplitude
- Median SNAP: normal
- Ulnar SNAP: normal
- LAC SNAP: normal
- MAC SNAP: normal
- **Motor**
- Radial motor (EIP/EDC): low amplitude
- Axillary motor: reduced
- Median motor: normal
- Ulnar motor: normal

EMG



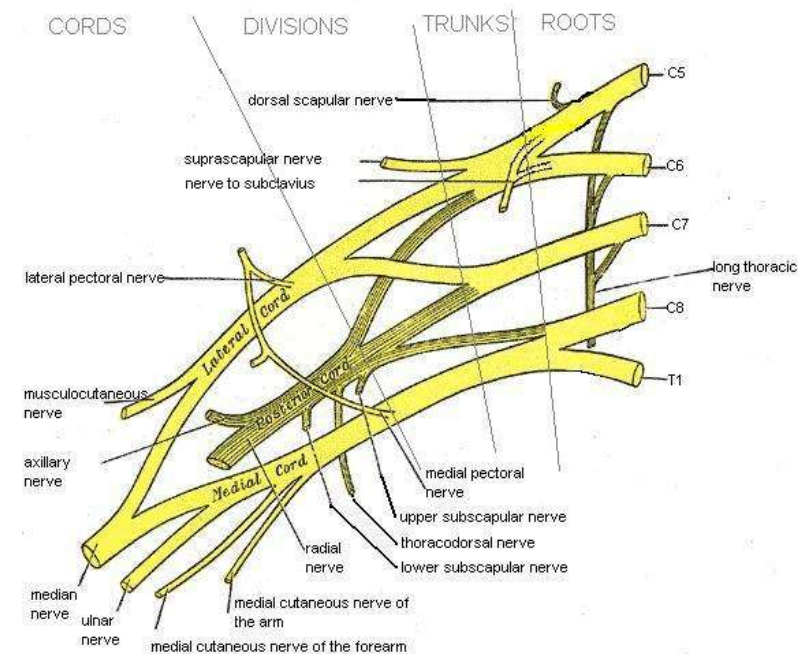
Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Amp	Dur	Poly	Recrt	Int Pat
Right	Abd Poll Brev	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	PronatorTeres	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	Biceps	Musculocut	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml
Right	Triceps	Radial	C6-7-8	*Incr	*2+	*2+	Nml	Nml	*3+	Nml	Nml
Right	Deltoid	Axillary	C5-6	*Incr	Nml	*1+	Nml	Nml	0	Nml	Nml
Right	Ext Digitorum	Radial (Post Int)	C7-8	*Incr	*2+	*2+	Nml	Nml	0	Nml	Nml
Right	BrachioRad	Radial	C5-6	*Incr	*3+	*2+	Nml	Nml	0	Nml	Nml
Right	ExtDigMin	Radial (Post Int)	C7-8	*Incr	*2+	*3+	Nml	Nml	0	Nml	Nml



Case 2

- **1. There is evidence of a brachial plexus lesion affecting the posterior cord that is actively affecting the sensorimotor fibers or the right upper extremity.**

- **Teaching Pearls**
- Entire Radial + axillary = posterior cord until proven otherwise
- Normal FDI/APB helps exclude lower trunk
- Radial SNAP abnormal supports postganglionic lesion
- Paraspinals normal = plexus, not root
- Lat Dorsi can be involved



Case 3 Brachial Plexopathies

- 45-year-old male presents for electrodiagnostic evaluation of progressive right hand numbness and weakness. Approximately 8 weeks ago, he sustained a displaced distal humeral fracture following a fall from a ladder that required open reduction and internal fixation. Since that time, he reports persistent numbness and tingling primarily involving the ring and small fingers of the right hand with gradual progression of hand weakness. He also reports difficulty with grip strength and fine motor tasks such as buttoning and key pinch. He has significant shoulder weakness.

Case 3 Brachial Plexus

- **Physical Examination**

- Severe visible atrophy of the first dorsal interosseous and hypothenar musculature on the right
- Weakness of first dorsal interosseous and interossei (3/5)
- Grip and key pinch strength reduced on the right
- Abductor pollicis brevis strength preserved at 5/5, EPL 5/5, Deltoid weak but firing.
- Sensation diminished to light touch along the ulnar distribution of the hand (ring and small fingers)
- No visible thenar atrophy
- Reflexes symmetric without long-tract signs
- Came to our nerve clinic with outside report already completed

DA17
DA18



Slide 31

DA17 Patient present with significant hypothenar and FDI atrophy. Numbness SF/RF. APB 5/5, EPL, 5/5, Has weakness in shoulder abduction.

Dylan Abshire, 3/8/2026

DA18 Came to our clinic with a completed nerve study already.

Dylan Abshire, 3/8/2026

Case 3 BP

Outside EMG NCS findings:

LABC- NR

MABC- NR

Ulnar sensory NR

Ulnar Motor NR

EMG Findings:

Tricep/PT/FDI/APB/FPL/BR

Impression- Widespread plexopathy with more severe involvement of the medial cord.

Nerve Conduction Studies
Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	P-T Amp (uV)	Norm P-T Amp	Site1	Site2	Delta-P (ms)	Dist (cm)
Left Lat Ante Brach Cutan Anti Sensory (Lat Forearm)									
Lat Biceps	NR		<3.0		>10	Lat Biceps	Lat Forearm		12.0
Left Med Ante Brach Cutan Anti Sensory (Med Forearm)									
Elbow	NR		<3.2		>5	Elbow	Med Forearm		12.0
		0.9		19.6					
Left Median Anti Sensory (2nd Digit)									
Wrist		3.5	<3.8	15.3	>12	Wrist	2nd Digit	3.5	13.0
Left Radial Anti Sensory (Base 1st Digit)									
Wrist		2.8	<2.9	11.8	>15	Wrist	Base 1st Digit	2.8	10.0
Left Ulnar Anti Sensory (5th Digit)									
Wrist	NR		<3.3		>10	Wrist	5th Digit		11.0

Motor Summary Table

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Motor (Abd Poli Brev)											
Wrist		4.8	<4.5	2.5	>4	Elbow	Wrist	5.3	28.0	53	>49
Elbow		10.1		1.6							
Left Ulnar Motor (Abd Dig Minimi)											
Wrist	NR		<4.0		>5.6						

Comparison Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	P-T Amp (uV)	Site1	Site2	Delta-P (ms)	Norm Delta (ms)
Left Median/Ulnar Palm Comparison (Wrist - 8cm)								
Median Palm		2.5	<2.2	14.1	Median Palm	Ulnar Palm	0.2	<0.4
Ulnar Palm		2.3	<2.2	4.3				

EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Pgw	Amp	Dur	Poly	Recrt	Comment
Left	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	
Left	Biceps	Musculocut	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	
Left	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	>12ms	2+	Nml
Left	Pronator Teres	Median	C6-7	Nml	Nml	Nml	Nml	>12ms	3+	Nml	
Left	1st Dors Int	Ulnar	C8-T1	Iner	Nml	Nml	Nml	>12ms	4+	Nml	
Left	Abd Poli Brev	Median	C8-T1	Nml	Nml	Nml	Nml	>12ms	4+	Nml	
Left	Ext Indicis	Radial (Post Int)	C7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	
Left	Flex Pol Long	Median (Ant Int)	C7,8,T1	Nml	Nml	Nml	Nml	>12ms	4+	Nml	
Left	Brachio Rad	Radial	C5-6	Nml	Nml	Nml	Nml	>12ms	3+	Nml	

- Normal MABC.
- Absent ulnar Snap.
- Ulnar M. Amp loss and focal slowing.
- FDI/FCU denervation.

Nerve Conduction Studies

Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	P-T Amp (µV)	Norm P-T Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left DorsCutan Anti Sensory (Dorsum 5th MC)											
Wrist	*NR		<2.5		>10	Wrist	Dorsum 5th MC		0.0		
Left Median Anti Sensory (3rd Digit)											
Wrist		3.6	<3.6	29.6	>10	Wrist	3rd Digit	3.6	14.0	39	
Left Radial Anti Sensory (Base 1st Digit)											
Wrist		1.7	<3.1	29.6	>15	Wrist	Base 1st Digit	1.7	8.0	47	
Left Ulnar Anti Sensory (5th Digit)											
Wrist		*NR	<3.7	*NR	>15.0	Wrist	5th Digit	6.0	14.0	23	
Left Med Ante Brach Cutan Anti Sensory (Med Forearm)											
Elbow		2.0		12.1		Elbow	Med Forearm	2.0	0.0		

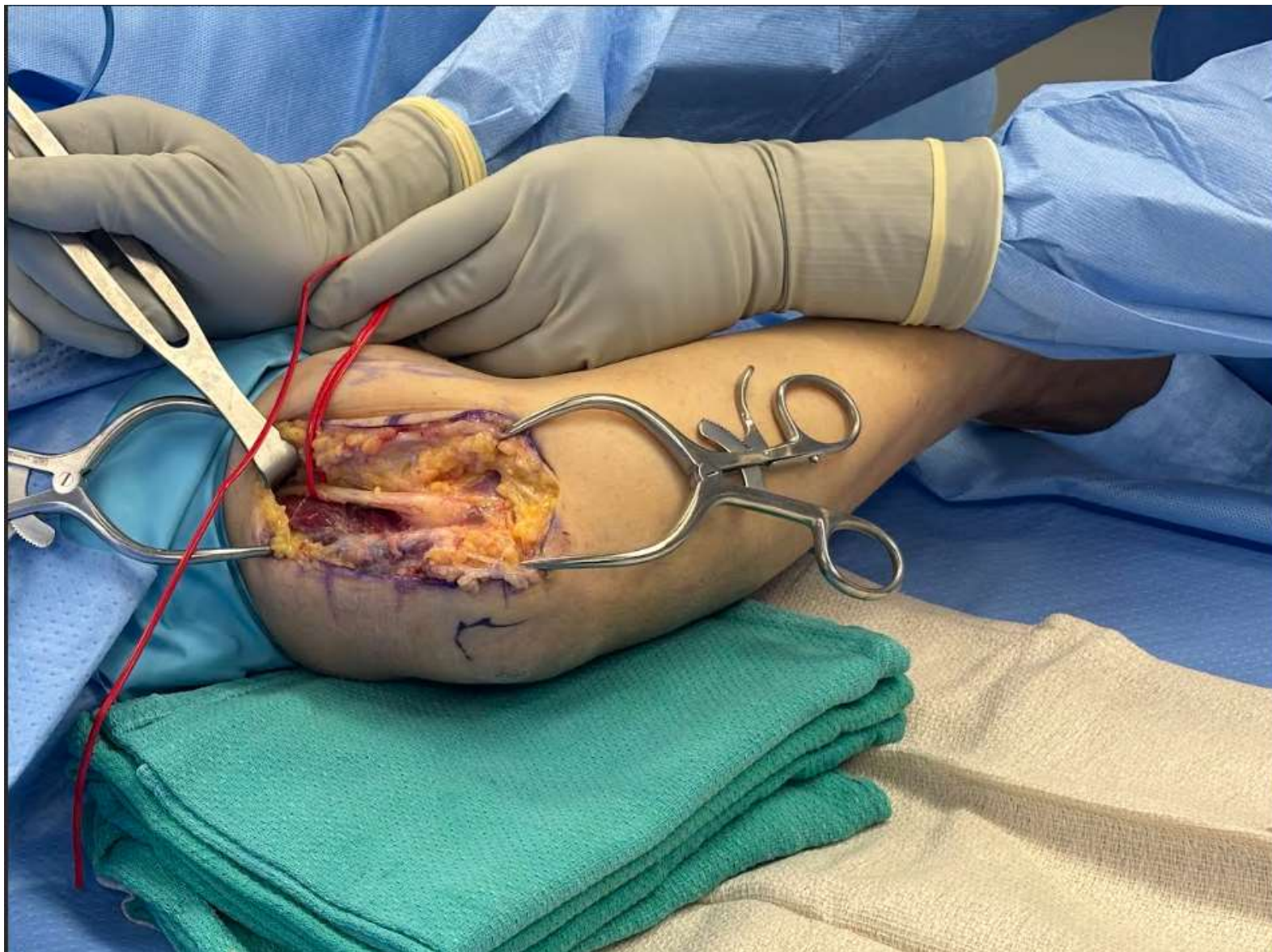
Motor Summary Table



Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Median Motor (Abd Poll Brev)											
Wrist-8cm		4.2	<4.2	3.7	>3	Elbow	Wrist-8cm	4.4	24.0	55	>48
Elbow		8.6		4.7							
Left Ulnar Motor (Abd Dig Min)											
Wrist-8cm		4.0	<4.2	*2.2	>3	B Elbow	Wrist-8cm	4.9	28.0	57	>48
B Elbow		8.9		1.5		A Elbow	B Elbow	4.4	8.0	*18	>48
A Elbow		13.3		1.5							

EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psy	Amp	Dur	Poly	Recrt	Int Pat	Comment
Left	Abd Poll Brev	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	1stDorInt/FCU	Ulnar	C8-T1	*Incr	*3+	*3+	Nml	Nml	0	*Reduced	Nml	
Left	PronatorTeres	Median	C6-7	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Biceps	Musculocut	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	EIP/ECRL	Radial (PIN)	C7-8	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	



Closing Comments

- Accurate assessment of brachial plexus pathology requires **deep anatomic mastery of the entire plexus** — from roots to trunks, divisions, cords, and terminal branches.
- Understanding **fiber contribution (C5–T1)** and overlap between peripheral nerves is essential for precise localization.
- High-level interpretation depends on strong **technical electrodiagnostic skills**, including proper muscle selection, correct needle placement, and high-quality waveform acquisition.
- Traumatic brachial plexopathy is a **time-sensitive diagnosis** where early localization and severity grading directly influence surgical and rehabilitation decision-making.
- Electrodiagnostic testing provides the **first objective physiologic baseline** following injury and distinguishes pre- vs post-ganglionic lesions, axonal loss, and early reinnervation.
- Pattern recognition is critical:** mechanism of injury + anatomy + EMG findings together allow precise localization to root, trunk, cord, or terminal nerve.

DIABETIC AMYOTROPHY OR LUMBAR RADICULOPATHY: CASE STUDIES AND IMPORTANT FEATURES WITH DIFFERENTIAL DIAGNOSIS.

Hulet R

ABSTRACT

BACKGROUND: Diabetes is typically associated with a distal sensorimotor polyneuropathic process. However, approximately 1% of diabetics may experience a more proximal neuropathic issue associated with hip and leg pain with weakness. Proximal leg pain and weakness in patients without sensory changes has been observed since 1890 and more research and observation has been done throughout the years showing a connection between diabetes with these types of symptoms. This condition has been called many names, but some of the more common names are diabetic amyotrophy, Bruns-Garfield syndrome, or proximal diabetic neuropathy.

CASE STUDIES: A 71-year-old female, diabetic of ~1 year, was referred for an NCS/EMG to assess for a lumbar radiculopathy after experiencing pain in the back radiating to the legs and more recent hip weakness. An 81-year-old female, diabetic of 15+ years, who was referred for an NCS/EMG to assess for a lumbar radiculopathy and leg weakness who has felt more unsteady over the last 9 months. A 67-year-old female, diabetic of 25+ years, who was referred for an NCS/EMG to assess for a lumbar radiculopathy and has had a spell of falls over the last month with her leg giving out on her. An 87-year-old female, diabetic of 30 years, referred for an NCS/EMG due to recent quad weakness and pain with the leg giving out often.

DISCUSSION: The main complaints behind diabetic amyotrophy can look very similar to a lumbar radiculopathic process, which makes it hard to diagnose. So, as a clinician it is important to know what information is helpful to understand and gather as a part of the history and clinical exam in the differential diagnosis. It is useful to know what the cause behind diabetic amyotrophy is. What are the signs and symptoms and risk factors for a diabetic amyotrophy versus a lumbar radiculopathy? What differences and similarities are there in the acuteness and mechanism of injury? What tests are useful to assist in the differential diagnosis? And lastly, are there any treatments that improve the prognosis?

CONCLUSION: When that a patient is referred for a lumbar radiculopathy as is also a diabetic, diabetic amyotrophy should be a part of the differential diagnosis. The cause of diabetic amyotrophy is considered to be a nonsystemic microvasculitis in origin. There is not one specific test that can be used to diagnose diabetic amyotrophy, but EMG/NCS testing along with lumbosacral plexus MRI together are useful in locating the problem. A clinician's medical history and clinical exam for diabetic amyotrophy patient will typically show that they are >60 years of age, male > female, have type 2 diabetes that has been diagnosed a median of 3 years ago, concurrent weight loss, have a painful onset unilaterally proximally with subacute and progressive weakness and atrophy within 2 weeks, and absent reflexes. The diagnosis of diabetic amyotrophy is typically one that is excluding other causes, namely a lumbosacral radiculopathy, but pattern recognition with symptoms and medical history will contribute to the diagnosis and patient care as we communicate concerns of diabetic amyotrophy with the referring provider.

Randall A. Hulet PT, DPT, ECS

Diabetic Amyotrophy or Lumbar Radiculopathy: Case Studies and Important Features with Differential Diagnosis

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History of Diabetic Amyotrophy

- First described in 1890 by a German neurologist Ludwig Bruns after seeing 3 cases (ages 57-70) with short-duration diabetes; all three developed severe pain in their hips and thighs, followed by a weakness and wasting of leg muscles without objective sensory loss
- Another case was described in 1890 by London physician Thomas Buzzard of a 55-year-old saying:
 - *'Began to get pain and tenderness in the front of the right thigh which extended presently down the leg to the foot. The pain was very severe and there was so much weakness that his knee often gave way and let him down. A month after the right the left leg was similarly attacked.'*



History of Diabetic Amyotrophy

- Later described by British neurologists Hugh Garland and Derek Taverner in 1953 describing 5 men (ages 56-73) with diabetes <4 years who experienced pain in the thigh/hips, atrophy in the quadriceps and areflexia, and no sensory changes.
- Addressed again in another article in 1955 with 7 more cases.
- In 1953 in Ohio physician John Goodman presented on diabetic motor neuropathy about 17 cases of femoral neuropathy- 16 who had diabetes.



Multitude of Names

- Bruns-Garland
- Diabetic Amyotrophy
- Diabetic Lumbosacral radiculoplexus neuropathy (DLRPN)
- Proximal Diabetic Neuropathy
- Lumbosacral Plexopathy
- Femoral-Sciatic Neuropathy
- Femoral Neuropathy
- Diabetic radiculo-plexo-neuropathy.



Prevalence and Incidence

- Prevalence of 1% of diabetics
 - The only available information comes from the Rochester Diabetic Neuropathy Study, which approximates the prevalence of DLRPN to be 1% of the diabetic community
- Incidence of 4.16/100,000 per year (Lumbosacral radiculoplexus neuropathy)
 - 3 times greater than other inflammatory conditions like GBS and CIDP, etc.



Signs and Symptoms

Common Symptoms

- Acute onset of pain in the proximal leg unilaterally
 - Deep and aching, tingling and possible burning
- Proximal or proximal >distal weakness
 - Typically unilateral, but some progression to bilaterally within 6 months
- Clinical Signs of a typical diabetic polyneuropathy
 - Not as likely with retinopathy and nephropathy

Less Common

- Autonomic involvement- orthostatic hypotension, diarrhea/constipation, changes in sweating



Signs and Symptoms

Common Signs

- Muscle Atrophy
 - Coincides with weight loss
- Absent Reflexes
 - Absent knee and ankle or just absent knee
- Sensory Impairment
 - Most usually related to distal sensorimotor polyneuropathy



Etiology

- Microvasculitis
 - Affects the arterioles and capillaries
 - Nerve biopsy shows:
 - Multi-focal fiber loss, neovascularization, and perineurial thickening that indicate an ischemic nerve injury
- Dysimmune microvascular dysfunction with secondary ischemic nerve injury



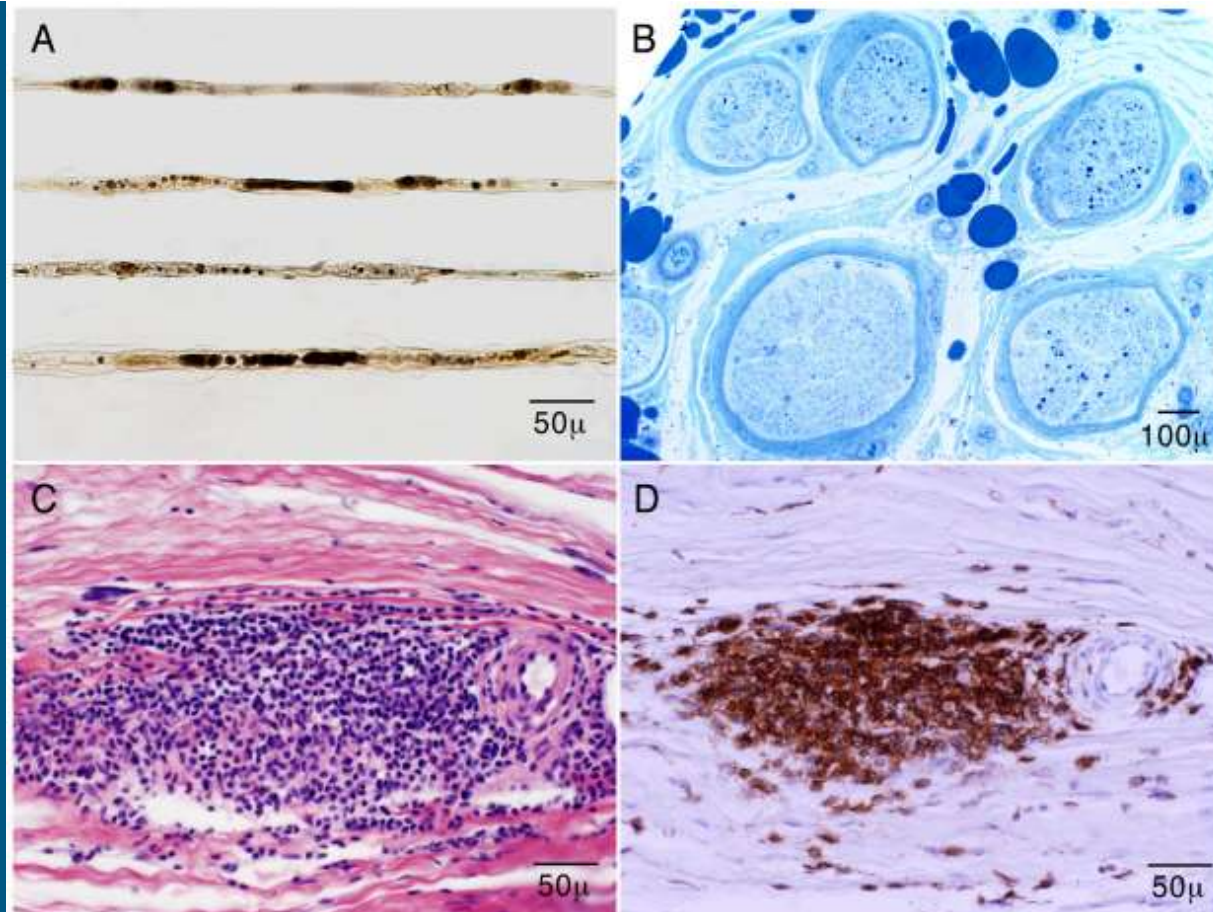


Figure 1.

Superficial peroneal nerve biopsy: Teased fiber preparation (osmium tetrachloride) shows that most fibers are undergoing active axonal degeneration (A). Semithin epoxy sections (methylene blue) show severe loss of myelinated fibers in a multifocal distribution (some fascicles have few remaining fibers whereas others have none) (B). Serial longitudinal paraffin sections show large epineurial perivascular (sheet-like) inflammatory collections on H&E staining (C) that react to a CD45 (leukocyte common antigen) immunostain. These findings demonstrate an active inflammatory neuropathy.

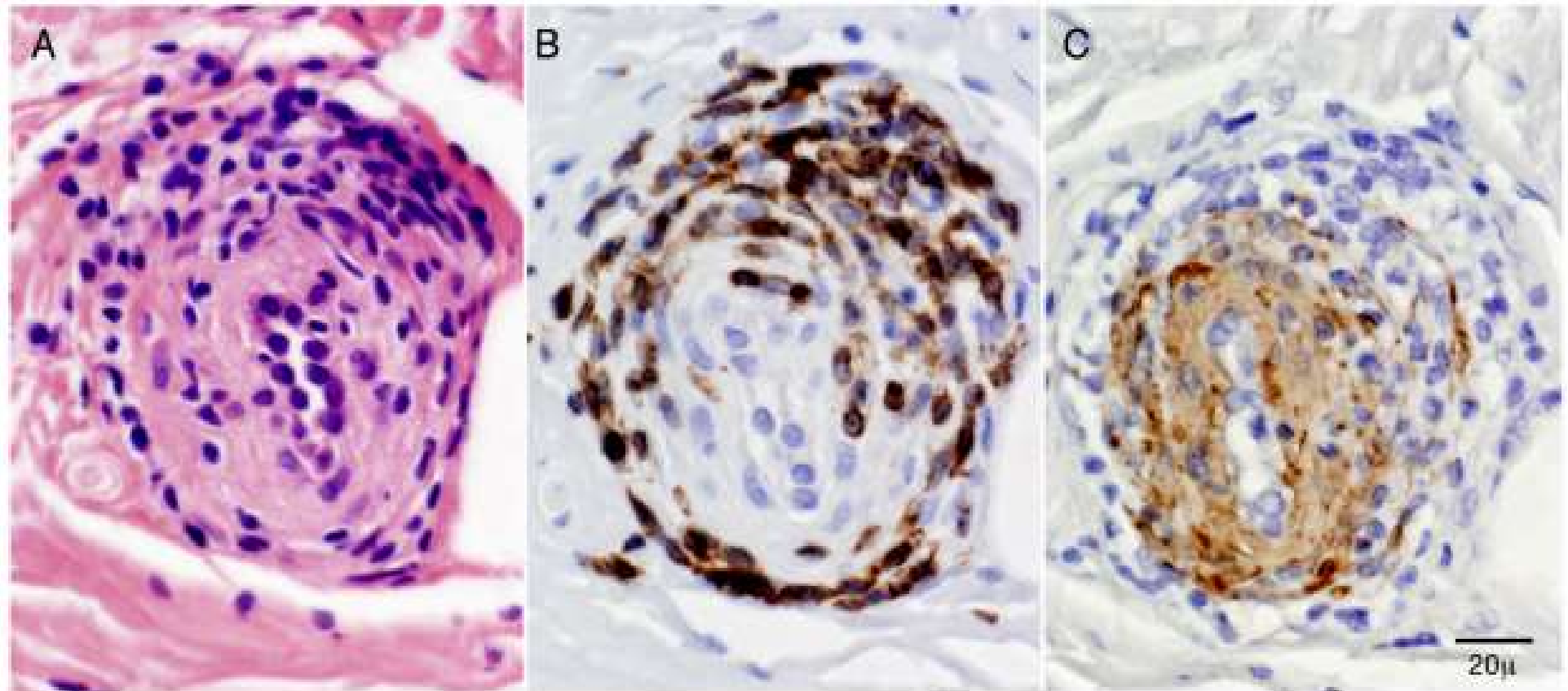
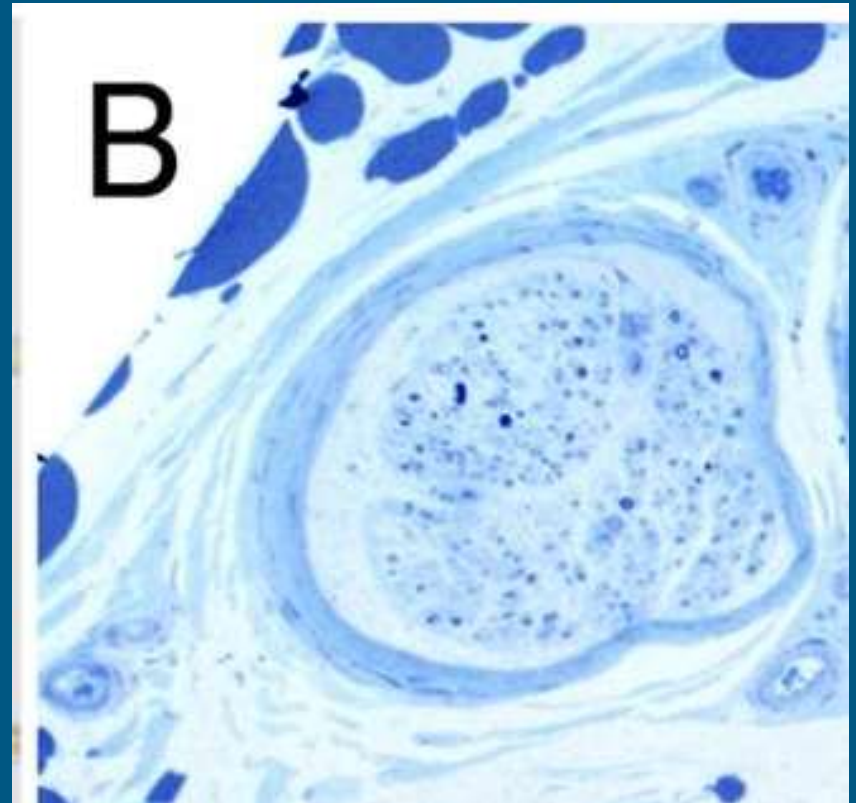
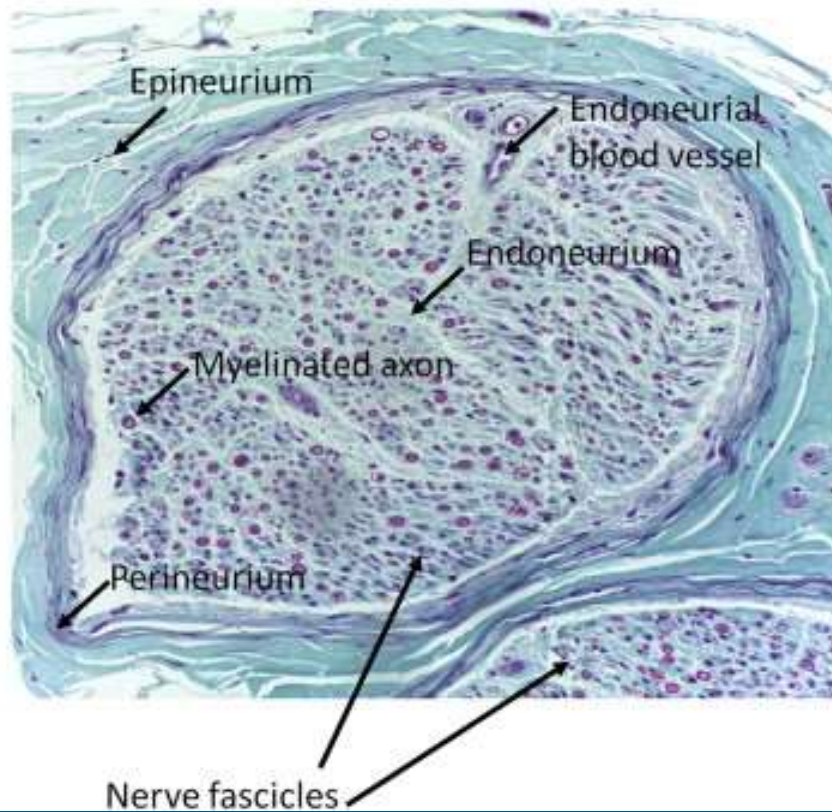


Figure 2.

Superficial peroneal nerve biopsy (serial epineurial paraffin sections): The sections show an epineurial vessel that has inflammatory cells involving the vessel wall (on H&E) (A), that immunostain with CD45 (B) and show disruption of muscle wall elements on smooth muscle actin preparation (C). These findings are diagnostic of microvasculitis.

Normal vs Abnormal

(B)



Not True Vasculitis

Feature	NSVN	Microvasculitis (true)	Diabetic Amyotrophy
Vessel wall destruction	Required	Required	Absent
Fibrinoid necrosis	Common	Common	Rare/Absent
Perivascular inflammation	Yes	Yes	Yes
Ischemic nerve injury	Yes	Yes	Yes
Systemic involvement	No	±	No
Course	Progressive	Progressive	Monophasic
Relapse	Common	Common	Rare



Comparison to DSPN

Feature	DSPN	Diabetic Amyotrophy
Vascular injury	Chronic microangiopathy	Acute microvascular failure
Distribution	Diffuse	Focal / patchy
Ischemia	Chronic hypoxia	Infarction
Inflammation	Minimal	Perivascular
Vessel wall destruction	No	No
Fiber loss	Gradual	Abrupt
Pattern	Length-dependent	Proximal / plexus
Course	Progressive	Monophasic



Risk Factors

- Type 2 Diabetes
 - Not usually severe
 - Often a recent diagnosis
 - Median A1C at 8.0%
- Typically 60+ years old



Differential Diagnoses

- Neoplastic Lumbosacral Plexopathy
- Lumbar Radiculopathy
- Cauda Equina Syndrome
- Lumbar Central Stenosis
- Guillan-Barre Syndrome
- Chronic Inflammatory Demyelinating Polyneuropathy
- Segmental Zoster Paresis



Lumbar Spinal Stenosis

- Herniated Nucleus Pulposus
 - Most Common
 - 95% of the time affects the L4/5 and/or L5/S1
 - Seen in 30–50-year-olds
- Osteoarthritis
 - Degeneration of disc space, osteophytes, facet joint arthritis leading to foraminal narrowing
 - Genetic predisposition, epigenetics, sex, lifestyle factors- diet, physical activity, and work-related habits.



Lumbar Spinal Stenosis

- Ligamentum Flavum Thickening
 - Pathophysiology is unclear
 - Possible risk factors:
 - Age
 - Disc degeneration
 - Hemodialysis



Differential Diagnosis

Symptomatic Presentation

Lumbar Spinal Stenosis

- Chronic Back Pain
- Neurogenic Claudication
- Mechanical aggravation/relief of symptoms
- Bilateral pain, unilateral radiating pain
- Burning pain
- Occasional Numbness
- MOI: Acute event or spinal movement

Diabetic Amyotrophy

- Acute unilateral pain to the proximal thigh
- Positional changes don't affect symptoms
- Possible sensory changes
- Muscle weakness/weight loss
- MOI: spontaneous, no spinal movement/event cause



Differential Diagnosis

Physical Examination

Lumbar Spinal Stenosis

- Changes to deep tendon reflexes
- Mechanical changes affect function/produce symptoms
- Myotomal weakness, dermatomal numbness
- Positive SLR and contralateral with HNP

Diabetic Amyotrophy

- Muscle Atrophy
- Absent reflexes
- Sensory changes- often distal related to a polyneuropathic process, but may also affect the anterolateral thigh and inner aspect of the leg
- Asymmetric proximal > distal weakness



Tests to Evaluate and Diagnose

- EMG
 - Denervation in a lumbosacral plexus pattern
 - Proximal > distal involvement
 - More acute/subacute axon loss than chronic proximally
 - Denervation can be seen in the paraspinals
 - Commonly see an underlying distal sensorimotor polyneuropathy
- MRI
 - Enhancement in root, plexus, and nerves- femoral, sciatic, and obturator
 - Better identification with diffusion tensor imaging



Tests to Evaluate and Diagnose

- Nerve biopsy
 - Not recommended for diagnosis
 - Can help prove signs of inflammation and ischemic injury
- Elevated protein in the CSF
 - Not typically significantly elevated
- Erythrocyte Sedimentary Rate(ESR)- not as useful



Treatment

- Immunotherapy (IVIG)
 - Often used to help with microvasculitis issues
- Corticosteroids
 - Research didn't show a significant with motor improvement
 - Better assisted with neuropathic symptoms
- Physical Therapy and assisted devices
- Medication or improvement with glycemic control



Prognosis

- People typically see eventual improvement in sensory and motor symptoms to normal or near normal
 - Process can take between 2 months and 2 years
- 1972 Study- excellent recovery at 18 months
 - Pain resolution by 1 year, followed by strength and then knee reflex.



Case 1

- Referral- BLE
 - Lumbar radiculopathy
- History/Complaints
 - 71-year-old female
 - Back pain radiating into the anterior thigh and medial lower leg to mid-calf after twisting while on a ladder that occurred 5 months ago
 - Hip weakness starting 1 week ago
 - Type 2 diabetic of 1-year, last A1C at 8%
 - Hypothyroidism- controlled with medication.
 - History of falls- last one a couple of months ago



Case 1

- Physical Exam
 - Reflexes: absent Achilles and patellar reflexes
 - Sensation: hyposensitivity to left medial thigh, medial knee, and medial lower leg above the ankle
 - MMT: 2+/5 left hip flexion, 3/5 left hip adduction, 4/5 left knee extension
 - Provocative testing: negative Tinel's, Babinski, and Clonus



Case 1: Data- NCS

Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	O-P* Amp (μV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Saphenous Anti Sensory (Anterior Medial Mall)											
Med Tibia		3.6		3.3							
Run #2		3.7		2.6							
Left Sup Fibular Anti Sensory (Ant Lat Mall)											
12 cm		3.6	<3.6	5.6	>2.0	12 cm	Ant Lat Mall	3.6	12.0	33	
Right Sup Fibular Anti Sensory (Ant Lat Mall)											
12 cm		3.2	<3.6	5.3	>2.0	12 cm	Ant Lat Mall	3.2	12.0	38	
Left Sural Anti Sensory (Lat Mall)											
Calf		4.1	<4.2	5.3	>5.0	Calf	Lat Mall	4.1	14.0	34	
Right Sural Anti Sensory (Lat Mall)											
Calf		3.8	<4.2	6.2	>5.0	Calf	Lat Mall	3.8	14.0	37	



Case 1: Data- NCS Continued

Motor Summary Table

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P* Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Fibular Motor (Ext Dig Brev)												
Ankle 8cm		4.5	<5.6	2.0	>1.0	6.72	B Fib	Ankle 8cm	7.1	26.5	*37	>38
B Fib		11.6		1.6		7.03	Poplt	B Fib	2.2	10.0	45	>39
Poplt		13.8		1.6		7.34						
Right Fibular Motor (Ext Dig Brev)												
Ankle 8cm		4.2	<5.6	1.6	>1.0	5.78	B Fib	Ankle 8cm	6.7	26.0	39	>38
B Fib		10.9		1.3		7.03	Poplt	B Fib	2.5	10.0	40	>39
Poplt		13.4		1.3		7.81						
Left Tibial Motor (Abd Hall Brev)												
Ankle 8cm		4.2	<6.1	10.0	>2.8	5.31	Knee	Ankle 8cm	9.9	38.5	39	>38
Knee		14.1		6.9		5.78						
Right Tibial Motor (Abd Hall Brev)												
Ankle 8cm		4.5	<6.1	7.0	>2.8	6.41	Knee	Ankle 8cm	8.9	39.0	44	>38
Knee		13.4		3.9		8.75						



Case 1: Data- NCS Continued

F Wave Studies

NR	F-Lat (ms)	Lat Norm (ms)	L-R F-Lat (ms)	M-Lat (ms)	FLat-MLat (ms)
Left Fibular (Mrkrs) (EDB) A-waves					
*NR		<60			
Right Fibular (Mrkrs) (EDB)					
	56.79	<60		4.55	52.24
Left Tibial (Mrkrs) (Abd Hallucis) A-waves					
	59.42	<61	0.67	4.67	54.75
Right Tibial (Mrkrs) (Abd Hallucis) A-waves					
	60.09	<61	0.67	5.33	54.76

H Reflex Studies

NR	H-Lat (ms)	Lat Norm (ms)	L-R H-Lat (ms)
Left Tibial (Gastroc)			
*NR		<36	
Right Tibial (Gastroc)			
*NR		<36	



Case 1: Data- EMG

EMG													
Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Left	Iliopsoas	Femoral	L2-3	Nml	*2+	*3+	Nml	Nml	*>12ms	*2+	*Reduced	*2-3 MU	
Left	RectFemoris	Femoral	L2-4	Nml	*2+	*3+	Nml	Nml	Nml	0	Nml	Nml	
Left	AdductorLong	Obturator	L2-4	Nml	*2+	*3+	Nml	Nml	Nml	*1+	*Reduced	*2-3 MU	
Left	VastusLat	Femoral	L2-4	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	VastusMed	Femoral	L2-4	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	AntTibialis	Dp Br Fibular	L4-5	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Ext Dig Brev	Dp Br Fibular	L5, S1	Nml	Nml	Nml	Nml	*Incr	Nml	0	*Reduced	Nml	
Left	Fibularis Long	Sup Br Fibular	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	BicepsFemS	Sciatic (Fibular)	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	MedGastroc	Tibial	S1-2	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	VastusMed	Femoral	L2-4	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	AntTibialis	Dp Br Fibular	L4-5	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Ext Dig Brev	Dp Br Fibular	L5, S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	BicepsFemS	Sciatic (Fibular)	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Fibularis Long	Sup Br Fibular	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	MedGastroc	Tibial	S1-2	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	



Case 1: Data- EMG Paraspinals

Paraspinal EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Left	Upper Lumbar	DPR	L2-4	Nml	Nml	*2+	Nml	NTd	NTd	NTd	NTd	NTd	
Left	Middle Lumbar	DPR	L4-5	Nml	*2+	*3+	Nml	NTd	NTd	NTd	NTd	NTd	
Left	Lower Lumbar	DPR	L5-S2	Nml	*1+	*1+	Nml	NTd	NTd	NTd	NTd	NTd	
Right	Upper Lumbar	DPR	L2-4	Nml	Nml	Nml	Nml	NTd	NTd	NTd	NTd	NTd	
Right	Middle Lumbar	DPR	L4-5	Nml	Nml	Nml	Nml	NTd	NTd	NTd	NTd	NTd	
Right	Lower Lumbar	DPR	L5-S2	Nml	*1+	*2+	Nml	NTd	NTd	NTd	NTd	NTd	



Impression

- Electrophysiologic evidence was recorded consistent with an **ACTIVE on SUBACUTE LUMBOSACRAL RADICULOPATHIC PROCESS** most likely involving the **LEFT L2/L3 SPINAL NERVE ROOT LEVELS**. See Comment #1
- Possible beginning stages of a length dependent polyneuropathic process
- Possible left L5/S1 radiculopathic process



Additional Imaging

- MRI 2 weeks before EMG Testing
 - Moderate left L5-S1 facet osteoarthritis with no facet edema or foraminal stenosis.
- X-ray
 - Decreased disc space between L5-S1



Diabetic Amyotrophy?

- Favors DA:
 - Type 2 Diabetes- recent diagnosis
 - Age >60
 - Proximal weakness and acute axon loss
 - Minimal abnormal MRI findings
- Doesn't Favor:
 - MOI- pain started with twisting on ladder
 - Still reports having pain
 - Some chronic EMG changes to proximal muscles
- Questionable:
 - Absent reflexes



Diabetic Amyotrophy?

- What do you think?
- I think it is an example of diabetic amyotrophy



Case 2

- Referral- RLE
 - Leg weakness and lumbar radiculopathy
- History/Complaints:
 - 81-year-old female
 - Unsteadiness on her feet over the last 9 months with many falls
 - Pain in the right knee and leg, some pain in the back, and numbness from the knees to the toes.
 - Had a minor stroke 6 years ago- mainly affecting speech.
 - Type 2 diabetic of 15+ years, with last A1C at 10%.



Case 2

- Physical Exam
 - Reflexes: absent Achilles and patellar reflexes
 - Sensation: hyposensitivity to medial lower legs and dorsum of the feet bilaterally
 - MMT: 2/5 right knee extension, 3/5 right hip flexion
 - Provocative testing: negative Tinel's, Babinski, and Clonus



Case 2: Data- NCS Continued

Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	O-P* Amp (μV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Radial Anti Sensory (Base 1st Digit)											
Wrist		2.7	<2.8	11.1	>7.0	Wrist	Base 1st Digit	2.7	10.0	37	
Right Sup Fibular Anti Sensory (Ant Lat Mall)											
12 cm	*NR		<3.6		>2.0	12 cm	Ant Lat Mall		12.0		
Left Sural Anti Sensory (Lat Mall)											
Calf	*NR		<4.2		>5.0	Calf	Lat Mall		14.0		
Right Sural Anti Sensory (Lat Mall)											
Calf	*NR		<4.2		>5.0	Calf	Lat Mall		14.0		
Right Ulnar D5 Anti Sensory (5th Digit)											
Wrist		*3.8	<3.7	13.0	>10	Wrist	5th Digit	3.8	14.0	37	



Case 2: Data- NCS

Motor Summary Table

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P* Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Fibular Motor (Ext Dig Brev)												
Ankle 8cm		5.0	<5.6	*0.4	>1.0	6.72	B Fib	Ankle 8cm	10.0	25.5	*26	>38
B Fib		15.0		0.5		6.41	Poplt	B Fib	2.5	10.0	40	>39
Poplt		17.5		0.5		6.41						
Right Fibular Motor (Ext Dig Brev)												
Ankle 8cm		5.6	<5.6	*0.5	>1.0	7.97	B Fib	Ankle 8cm	9.1	25.5	*28	>38
B Fib		14.7		0.5		8.75	Poplt	B Fib	2.3	10.0	43	>39
Poplt		17.0		0.4		9.22						
Right Tibial Motor (Abd Hall Brev)												
Ankle 8cm		5.3	<6.1	*2.3	>2.8	9.69	Knee	Ankle 8cm	15.0	37.0	*25	>38
Knee		20.3		0.6		8.91						
Right Ulnar Motor (Abd Dig Minimi)												
Wrist 8cm		2.8	<3.7	7.1	>4.3	5.47	B Elbow	Wrist 8cm	3.8	18.5	49	>49
B Elbow		6.6		7.2		5.47	A Elbow	B Elbow	2.2	10.0	*45	>46
A Elbow		8.8		6.8		5.63	Axilla	A Elbow	1.4	9.0	64	>50
Axilla		10.2		5.6		5.63						



Case 2: Data- NCS Continued

F Wave Studies

NR	F-Lat (ms)	Lat Norm (ms)	L-R F-Lat (ms)	M-Lat (ms)	FLat-MLat (ms)
Left Fibular (Mrkrs) (EDB)					
	*65.98	<60	1.33	6.00	59.98
Right Fibular (Mrkrs) (EDB)					
	*64.65	<60	1.33	6.67	57.98
Right Tibial (Mrkrs) (Abd Hallucis)					
	*75.42	<61		8.00	67.42
Right Ulnar (Mrkrs) (Abd Dig Min)					
	29.33	<36		2.67	26.66

H Reflex Studies

NR	H-Lat (ms)	Lat Norm (ms)	L-R H-Lat (ms)	M-Lat (ms)	HLat-MLat (ms)
Right Tibial (Gastroc)					
	*41.72	<36		6.67	35.05



Case 2: Data- EMG

EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	VastusLat	Femoral	L2-4	Nml	*3+	*3+	Nml	Nml	*>12ms	*2+	*Reduced	*1-2 MU	
Right	VastusMed	Femoral	L2-4	Nml	*2+	*3+	Nml	Nml	*-	*-	*-	*-	No MUPs
Right	AdductorLong	Obturator	L2-4	Nml	*3+	*3+	Nml	Nml	*>12ms	*2+	*Reduced	*25%	
Right	AntTibialis	Dp Br Fibular	L4-5	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Ext Dig Brev	Dp Br Fibular	L5, S1	Nml	Nml	Nml	Nml	Nml	*>12ms	0	*Reduced	*1-2 MU	
Right	PostTibialis	Tibial	L5, S1	Nml	Nml	Nml	Nml	*Incr	Nml	0	*Reduced	Nml	
Right	Fibularis Long	Sup Br Fibular	L5-S1	Nml	Nml	Nml	Nml	*Incr	Nml	0	*Reduced	*2-3 MU	
Right	BicepsFemS	Sciatic (Fibular)	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	MedGastroc	Tibial	S1-2	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	VastusMed	Femoral	L2-4	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	AntTibialis	Dp Br Fibular	L4-5	Nml	Nml	Nml	Nml	*Incr	Nml	0	*Reduced	Nml	
Left	PostTibialis	Tibial	L5, S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Ext Dig Brev	Dp Br Fibular	L5, S1	Nml	Nml	Nml	Nml	Nml	*>12ms	0	*Reduced	*1-2 MU	
Left	BicepsFemS	Sciatic (Fibular)	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Fibularis Long	Sup Br Fibular	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	MedGastroc	Tibial	S1-2	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	



Case 2: Data- EMG Paraspinals

Paraspinal EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	Upper Lumbar	DPR	L2-4	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Right	Middle Lumbar	DPR	L4-5	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Right	Lower Lumbar	DPR	L5-S2	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Left	Upper Lumbar	DPR	L2-4	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Left	Middle Lumbar	DPR	L4-5	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Left	Lower Lumbar	DPR	L5-S2	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	



Impression

- Electrophysiologic evidence was recorded consistent with an **ACUTE on CHRONIC LUMBOSACRAL RADICULOPATHIC PROCESS** most likely involving the **RIGHT L2-4 SPINAL NERVE ROOT LEVEL**. See Comment #1.
- Electrophysiologic evidence recorded was consistent with a **SENSORIMOTOR NERVE PERIPHERAL POLYNEUROPATHIC PROCESS** characterized by **chronic axonal loss and demyelination involving the lower > upper extremities**. See Comment #1.



Additional Imaging

- X-ray- about 2 years ago
 - Old T12 compression fracture with degenerative changes below and a grade 1 degenerative listhesis at L4-5



Diabetic Amyotrophy?

- Favors DA:
 - Type 2 Diabetes
 - Age >60
 - Proximal Weakness with acute axon loss
 - Polyneuropathy Present
 - Minimal X-ray abnormalities
- Doesn't Fit:
 - Continued pain in the leg- but some from falls
 - Not recent diabetes diagnosis and not as well managed
 - Chronic EMG findings in proximal muscles
- Questionable:
 - Absent reflexes



Diabetic Amyotrophy?

- What would you think?
- I think it is an example diabetic amyotrophy superimposed on a polyneuropathy



Case 3

- Referral- RLE
 - Weakness and “rule out HNP”
- History/Complaints:
 - 67-year-old female
 - A spell of falls 1 month ago with the leg giving out
 - Numbness and tingling in bilateral feet
 - Type 2 diabetic of 25 years, with last A1C at 9%.
 - No back pain



Case 3

- Physical Exam
 - Reflexes: absent Achilles and patellar reflexes
 - Sensation: hyposensitivity to medial lower legs and dorsum of the feet bilaterally
 - MMT: 2/5 right knee extension, 3/5 right hip flexion
 - Provocative testing: negative Tinel's, Babinski, and Clonus



Case 3: Data- NCS

Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	O-P* Amp (µV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Radial Anti Sensory (Base 1st Digit)											
Wrist		2.4	<2.7	15.6	>10.0	Wrist	Base 1st Digit	2.4	10.0	42	
Left Sup Fibular Anti Sensory (Ant Lat Mall)											
12 cm	*NR		<3.6		>2.0	12 cm	Ant Lat Mall		12.0		
Right Sup Fibular Anti Sensory (Ant Lat Mall)											
12 cm		*4.1	<3.6	3.2	>2.0	12 cm	Ant Lat Mall	4.1	12.0	29	
Left Sural Anti Sensory (Lat Mall)											
Calf		*4.3	<4.2	*4.7	>5.0	Calf	Lat Mall	4.3	14.0	33	
Right Sural Anti Sensory (Lat Mall)											
Calf		*4.4	<4.2	*3.2	>5.0	Calf	Lat Mall	4.4	14.0	32	
Left Ulnar D5 Anti Sensory (5th Digit)											
Wrist		*4.0	<3.6	*4.1	>10	Wrist	5th Digit	4.0	14.0	35	
B Elbow		7.3		2.0		B Elbow	Wrist	3.3	18.5	56	>50
A Elbow		9.7		1.4		A Elbow	B Elbow	2.4	10.0	*42	>53



Case 3: Data- NCS Continued

Motor Summary Table

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P* Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Fibular Motor (Ext Dig Brev)												
Ankle 8cm		*6.1	<5.6	1.0	>1.0	5.63	B Fib	Ankle 8cm	8.4	27.0	*32	>38
B Fib		14.5		1.1		6.09	Poplt	B Fib	1.9	10.0	53	>39
Poplt		16.4		1.0		6.25						
Right Fibular Motor (Ext Dig Brev)												
Ankle 8cm		*6.1	<5.6	*0.5	>1.0	7.66	B Fib	Ankle 8cm	7.8	27.5	*35	>38
B Fib		13.9		0.5		8.13	Poplt	B Fib	1.7	10.0	59	>39
Poplt		15.6		0.4		7.03						
Left Tibial Motor (Abd Hall Brev)												
Ankle 8cm		5.8	<6.1	*1.1	>2.8	7.50	Knee	Ankle 8cm	11.1	40.0	*36	>38
Knee		16.9		0.9		7.97						
Right Tibial Motor (Abd Hall Brev)												
Ankle 8cm		4.7	<6.1	*2.2	>2.8	6.88	Knee	Ankle 8cm	10.9	39.0	*36	>38
Knee		15.6		0.7		7.66						
Left Ulnar Motor (Abd Dig Minimi)												
Wrist 8cm		3.1	<3.6	7.4	>4.2	7.19	B Elbow	Wrist 8cm	4.1	20.0	49	>49
B Elbow		7.2		5.0		7.50	A Elbow	B Elbow	2.6	10.0	*38	>47
A Elbow		9.8		4.9		7.50	Axilla	A Elbow	1.8	10.0	56	>50
Axilla		11.6		4.5		7.66						



Case 3: Data- NCS Continued

F Wave Studies

NR	F-Lat (ms)	Lat Norm (ms)	L-R F-Lat (ms)	M-Lat (ms)	FLat-MLat (ms)
Left Fibular (Mrkrs) (EDB)					
	*62.02	<60	1.87	5.76	56.26
Right Fibular (Mrkrs) (EDB)					
	*60.15	<60	1.87	5.89	54.26
Left Tibial (Mrkrs) (Abd Hallucis)					
	*65.36	<61	0.67	6.16	59.20
Right Tibial (Mrkrs) (Abd Hallucis)					
	*64.69	<61	0.67	5.49	59.20
Left Ulnar (Mrkrs) (Abd Dig Min)					
	29.64	<36		2.61	27.03

H Reflex Studies

NR	H-Lat (ms)	Lat Norm (ms)	L-R H-Lat (ms)	M-Lat (ms)	HLat-MLat (ms)
Left Tibial (Gastroc)					
	35.81	<36	0.24	4.10	31.71
Right Tibial (Gastroc)					
	35.57	<36	0.24	4.34	31.23



Case 3: Data- EMG

EMG													
Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	VastusLat	Femoral	L2-4	Nml	*2+	*3+	Nml	Nml	Nml	0	Nml	Nml	
Right	RectFemoris	Femoral	L2-4	Nml	*2+	*2+	Nml	Nml	Nml	0	Nml	Nml	
Right	AdductorLong	Obturator	L2-4	Nml	Nml	*2+	Nml	Nml	Nml	0	Nml	Nml	
Right	VastusMed	Femoral	L2-4	Nml	Nml	*1+	Nml	*Incr	Nml	0	*Reduced	Nml	
Right	AntTibialis	Dp Br Fibular	L4-5	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Ext Dig Brev	Dp Br Fibular	L5, S1	Nml	Nml	Nml	Nml	Nml	Nml	0	*Reduced	*2-3 MU	
Right	BicepsFemS	Sciatic (Fibular)	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Right	Fibularis Long	Sup Br Fibular	L5-S1	Nml	Nml	Nml	Nml	*Incr	Nml	0	*Reduced	Nml	
Right	MedGastroc	Tibial	S1-2	Nml	Nml	Nml	Nml	Nml	Nml	0	*Reduced	*75%	
Left	Deltoid	Axillary	C5-6	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Triceps	Radial	C6-7-8	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	ExtDigCom	Radial (Post Int)	C7-8	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Abd Poll Brev	Median	C8-T1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	1stDorInt	Ulnar	C8-T1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	VastusMed	Femoral	L2-4	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	AntTibialis	Dp Br Fibular	L4-5	Nml	Nml	Nml	Nml	*Incr	Nml	0	*Reduced	*75%	
Left	Ext Dig Brev	Dp Br Fibular	L5, S1	Nml	Nml	Nml	Nml	Nml	Nml	0	*Reduced	*2-3 MU	
Left	BicepsFemS	Sciatic (Fibular)	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Fibularis Long	Sup Br Fibular	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	AbdHallucis	MedPlantar	S1-2	Nml	Nml	Nml	Nml	Nml	Nml	0	*Reduced	*1-2 MU	
Left	MedGastroc	Tibial	S1-2	Nml	Nml	Nml	Nml	*Incr	Nml	*1+	*Reduced	*2-3 MU	



Case 3: Data- EMG Paraspinals

Paraspinal EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Right	Upper Lumbar	DPR	L2-4	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Right	Middle Lumbar	DPR	L4-5	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Right	Lower Lumbar	DPR	L5-S2	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Left	Upper Lumbar	DPR	L2-4	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Left	Middle Lumbar	DPR	L4-5	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Left	Lower Lumbar	DPR	L5-S2	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	



Impression

- Electrophysiologic evidence was recorded consistent with an **ACTIVE LUMBOSACRAL RADICULOPATHIC PROCESS** most likely involving the **RIGHT L2-L4 SPINAL NERVE ROOT LEVELS**. See Comment #1.
- Electrophysiologic evidence recorded was consistent with a **SENSORIMOTOR NERVE PERIPHERAL POLYNEUROPATHIC PROCESS** characterized by **chronic axonal loss and demyelination involving the lower and upper extremities**. See Comment #2.
- Mild Left Ulnar Nerve @ Elbow



Additional Imaging

- MRI:

- Multilevel degenerative changes of the lumbar spine, superimposed upon mild levoconvex curvature and congenitally short pedicles. At L4-5, small left posterolateral/foraminal disc protrusion, encroaching upon the **traversing left L5 and exiting left L4 nerve roots**. Mild to moderate central stenosis as well as moderate left and mild to moderate right subarticular zone stenosis. **Severe left and moderate right foraminal stenosis with encroachment of the exiting right L4 nerve root**. At L3-4, small right foraminal/far lateral disc protrusion, **encroaching upon the exiting right L3 nerve root**. Mild to moderate (right slightly greater than left) subarticular zone stenosis and **contact of the traversing bilateral L4 nerve roots**. **Mild to moderate right and mild left foraminal stenosis**. At L2-3, small left posterolateral/foraminal disc extrusion with slight superior and inferior migration, but no significant nerve root displacement.



Diabetic Amyotrophy?

- Favors DA:
 - Type 2 diabetic
 - Age >60
 - Polyneuropathy present
 - Proximal weakness and acute axon loss in those muscles
- Doesn't Favor:
 - MRI findings supporting
 - No onset of severe hip/leg pain
 - Not recent diabetes diagnosis
- Questionable:
 - Absent reflexes



Diabetic Amyotrophy?

- What do you think?
- I think it is a lumbar radiculopathy superimposed on the polyneuropathy



Case 4

- Referral- LLE
 - Femoral Neuropathy
- History/Complaints:
 - 87-year-old female
 - In hospital for 2 days with UTI 5-6 weeks before testing
 - Pain in the hips and left leg
 - Left leg giving out
 - Numbness to medial lower leg
 - Type 2 diabetic of 30 years, with last A1C at <6%, always controlled with diet
 - Chronic low back pain
 - Heart Surgery for myxoma, vein removal for varicose veins



Case 4

- Physical Exam
 - Reflexes: absent left patellar reflex
 - Sensation: hyposensitivity to the whole left leg
 - MMT: 2/5 left hip flexion and knee extension, 3+/5 left ankle plantarflexion, 4/5 right hip flexion and left knee flexion
 - Provocative testing: negative Tinel's, Babinski, and Clonus
 - Extensive ankle/foot arthritis and deformity- left worse than right
 - Charcot-type foot deformity on the left



Case 4: Data- NCS

Anti Sensory Summary Table

Stim Site	NR	Peak (ms)	Norm Peak (ms)	O-P* Amp (µV)	Norm O-P Amp	Site1	Site2	Delta-P (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Saphenous Anti Sensory (Anterior Medial Mall)											
Med Tibia	*NR										
Right Saphenous Anti Sensory (Anterior Medial Mall)											
Med Tibia	*NR										
Left Sup Fibular Anti Sensory (Ant Lat Mall)											
12 cm		3.0	<3.6	4.2	>2.0	12 cm	Ant Lat Mall	3.0	12.0	40	
Run 2		3.0		4.9							
Left Sural Anti Sensory (Lat Mall)											
Calf		3.3	<4.2	*3.6	>5.0	Calf	Lat Mall	3.3	14.0	42	
Run 2		3.6		3.7							
Right Sural Anti Sensory (Lat Mall)											
Calf		3.5	<4.2	*4.1	>5.0	Calf	Lat Mall	3.5	14.0	40	
Run 2		3.6		4.6							



Case 4: Data- NCS Continued

Motor Summary Table

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P* Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Left Femoral Motor (Vastus Med)												
8 cm		2.5		0.8		13.13						
Right Femoral Motor (Vastus Med)												
8 cm		1.9		4.6		11.88						
Left Fibular Motor (Ext Dig Brev)												
Ankle 8cm		3.4	<5.6	2.0	>1.0	6.25	B Fib	Ankle 8cm	5.4	23.0	43	>38
B Fib		8.8		1.9		5.94	Poplt	B Fib	2.0	10.0	50	>39
Poplt		10.8		2.0		5.78						
Left Tibial Motor (Abd Hall Brev)												
Ankle 8cm		3.6	<6.1	*0.6	>2.8	11.09	Knee	Ankle 8cm	8.9	36.5	41	>38
Knee		12.5		0.2		10.78						
Right Tibial Motor (Abd Hall Brev)												
Ankle 8cm		4.5	<6.1	*1.2	>2.8	6.72	Knee	Ankle 8cm	7.1	35.0	49	>38
Knee		11.6		1.2		7.03						



Case 4: Data- NCS Continued

F Wave Studies

NR	F-Lat (ms)	Lat Norm (ms)	L-R F-Lat (ms)	M-Lat (ms)	FLat-MLat (ms)
Left Fibular (Mrkrs) (EDB)					
	41.98	<60		3.33	38.65
Left Tibial (Mrkrs) (Abd Hallucis)					
	50.76	<61	2.00	5.33	45.43
Right Tibial (Mrkrs) (Abd Hallucis)					
	52.76	<61	2.00	4.67	48.09

H Reflex Studies

NR	H-Lat (ms)	Lat Norm (ms)	L-R H-Lat (ms)	M-Lat (ms)	HLat-MLat (ms)
Left Tibial (Gastroc)					
	26.68	<36		3.35	23.33



Case 4: Data- EMG

EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Left	Iliopsoas	Femoral	L2-3	Nml	Nml	Nml	Nml	Nml	Nml	0	*Reduced	*50%	
Left	AdductorLong	Obturator	L2-4	Nml	Nml	Nml	Nml	Nml	*>12ms	*2+	*Reduced	*25%	
Left	VastusMed	Femoral	L2-4	Nml	Nml	*3+	Nml	*-	*-	*-	*-	*-	No MUPs
Left	RectFemoris	Femoral	L2-4	Nml	Nml	*3+	Nml	Nml	*>12ms	*1+	*Reduced	*1-2 MU	
Left	VastusLat	Femoral	L2-4	Nml	*1+	*2+	Nml	Nml	*-	*-	*-	*-	No MUPs
Left	AntTibialis	Dp Br Fibular	L4-5	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	PostTibialis	Tibial	L5, S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	BicepsFemS	Sciatic (Fibular)	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	Fibularis Long	Sup Br Fibular	L5-S1	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	
Left	MedGastroc	Tibial	S1-2	Nml	Nml	Nml	Nml	Nml	Nml	0	Nml	Nml	

Paraspinal EMG

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Other	Amp	Dur	Poly	Recrt	Int Pat	Comment
Left	Upper Lumbar	DPR	L2-4	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Left	Middle Lumbar	DPR	L4-5	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	
Left	Lower Lumbar	DPR	L5-S2	Nml	Nml	Nml	Nml	NT'd	NT'd	NT'd	NT'd	NT'd	



Impression

- Electrophysiologic evidence was recorded consistent with a **LEFT FEMORAL** nerve compromise of motor nerve fibers with **evidence of ACTIVE and SUBACUTE axonal disruption** at this time. See Comment #1.
 - There was evidence of subacute/chronic abnormalities in the adductor longus and reduced recruitment in the iliopsoas muscle with needle EMG. This could possibly indicate a L2-4 radiculopathic process, but it could also possible be related to diabetic amyotrophy (Bruns-Garland Syndrome or diabetic radiculo-plexo neuropathy). Correlation to imaging is recommended.



Impression Continued

- The left and right saphenous nerve failed to respond to stimulation bilaterally, the sural sensory nerve and tibial motor nerve amplitudes were diminished, but each of these are likely just related to patient age and/or natural wear and tear without other correlative abnormal findings outside of finding #1.



Additional Imaging

- CT of Abdomen- 2 months previous at ER
 - 1. No acute abnormality.
 - 2. Cirrhosis with portal hypertension.
 - 3. Colonic diverticulosis with no evidence of diverticulitis.
- CT of Lumbar Spine
 - No acute findings. Multilevel lumbar spondylosis. Grade 1 anterolisthesis of L3 on L4, L4 on L5 and L5 on S1.
- MRI Lumbar Spine:
 - Multilevel degenerative disc disease and facet osteoarthritis causing varying degrees of neural foraminal narrowing and central stenosis. **Central stenosis is most evident at the level of L4-L5.**
Neural foraminal narrowing is most evident at the level of L4-L5



Diabetic Amyotrophy?

- Favors DA:
 - Type 2 diabetic
 - Age
 - Proximal weakness-pain more asymmetric
 - Absent patellar reflex
 - MRI/CT not remarkable
 - Starting to have some weakness contralaterally
- Doesn't Favor DA:
 - Not recent diabetes diagnosis
 - Chronic low back pain
 - Recent hospitalization prior to weakness



Diabetic Amyotrophy?

- What do you think?
- I think this is an example of diabetic amyotrophy primarily affecting the femoral nerve



Takeaways

- Know the key characteristics to look for will help with getting the correct diagnosis
 - Type 2 DM of more recent diagnosis, Over 65, acute proximal leg pain followed by weakness
- Better history and more details about their diabetes, the mechanism of injury, and aggravating/relieving symptoms
- Don't be afraid to communicate the possibility of DA with the referring provider, so they can order additional testing to help rule it in/rule out other causes



Takeaways

- EMG/NCS findings
 - Looks more like a lumbosacral plexus
 - Acute axon loss in proximal muscles
 - Asymmetric findings
 - May have a distal sensorimotor polyneuropathy as well
 - Could show paraspinal involvement



Questions



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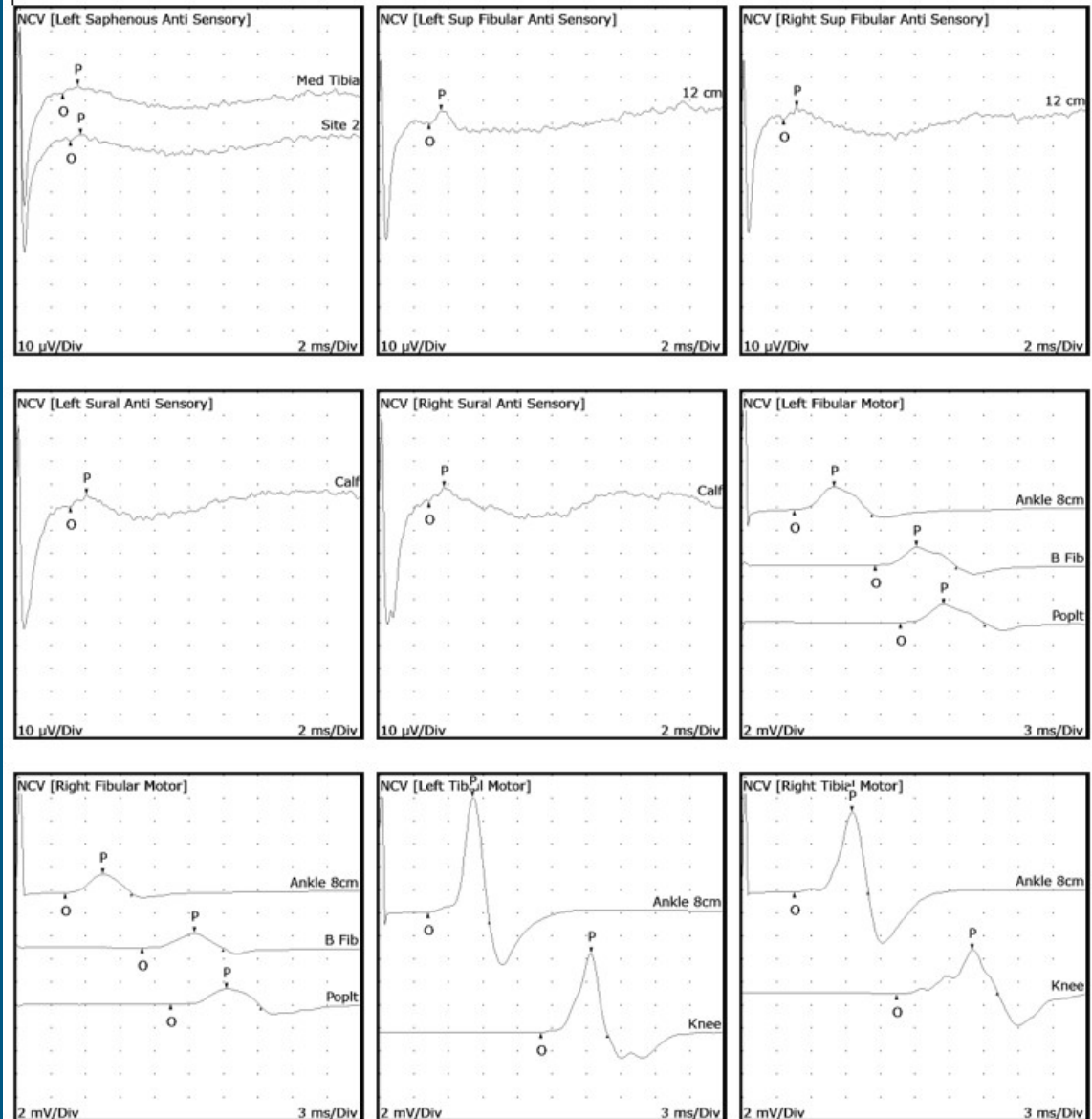


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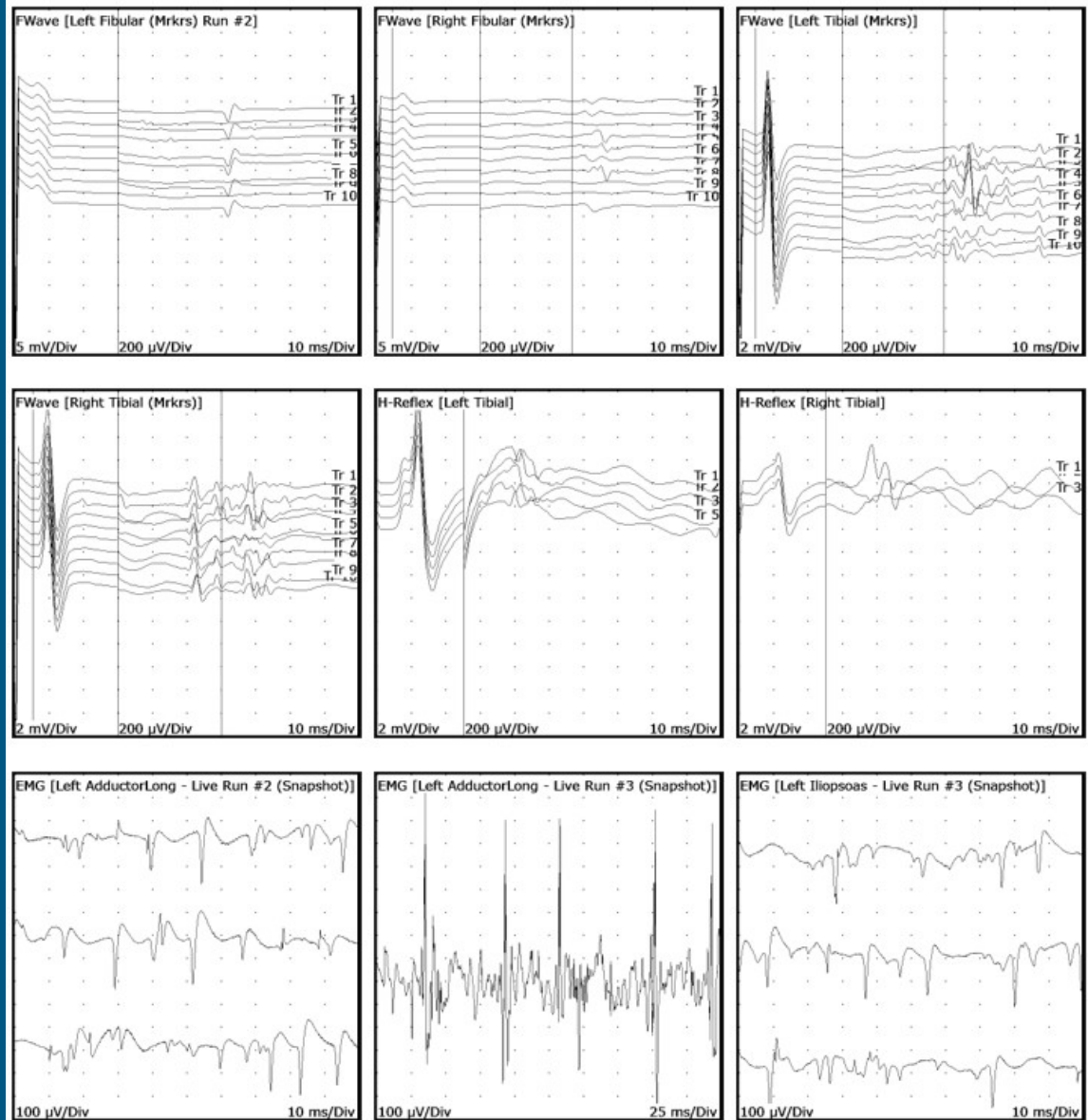


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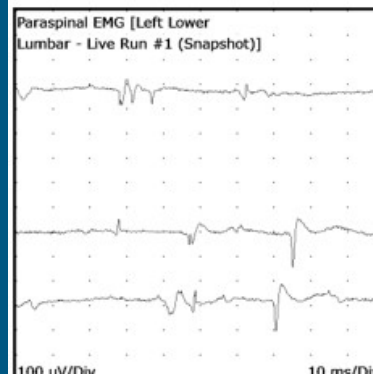
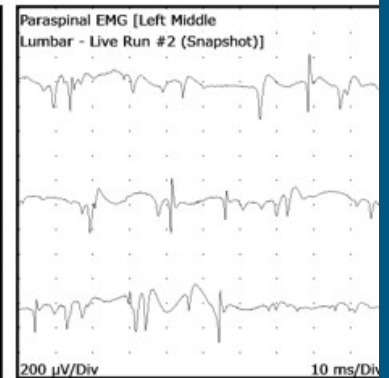
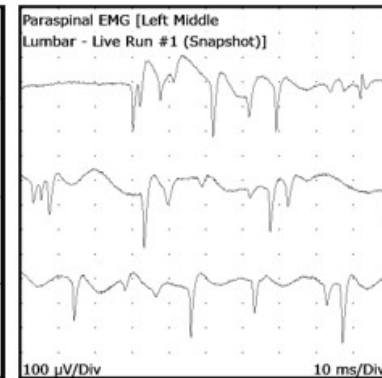
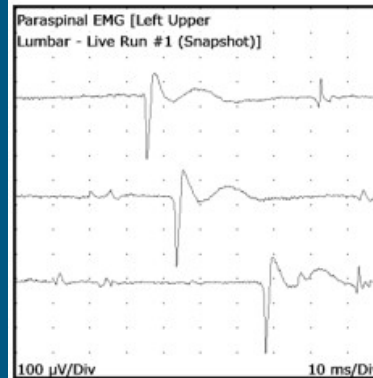
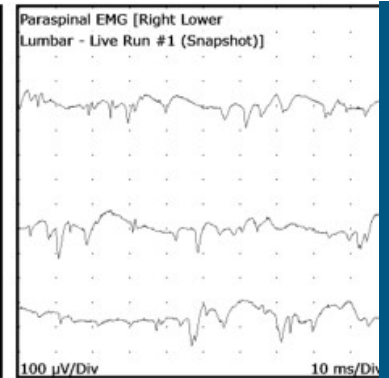
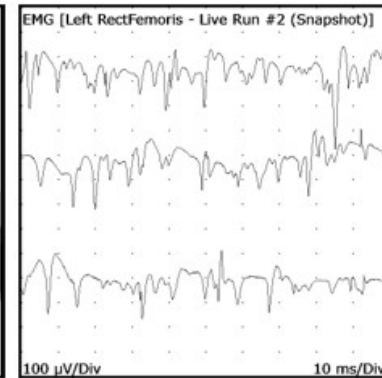
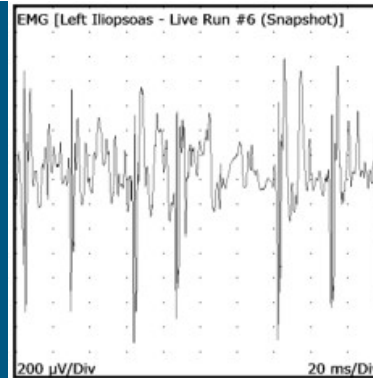
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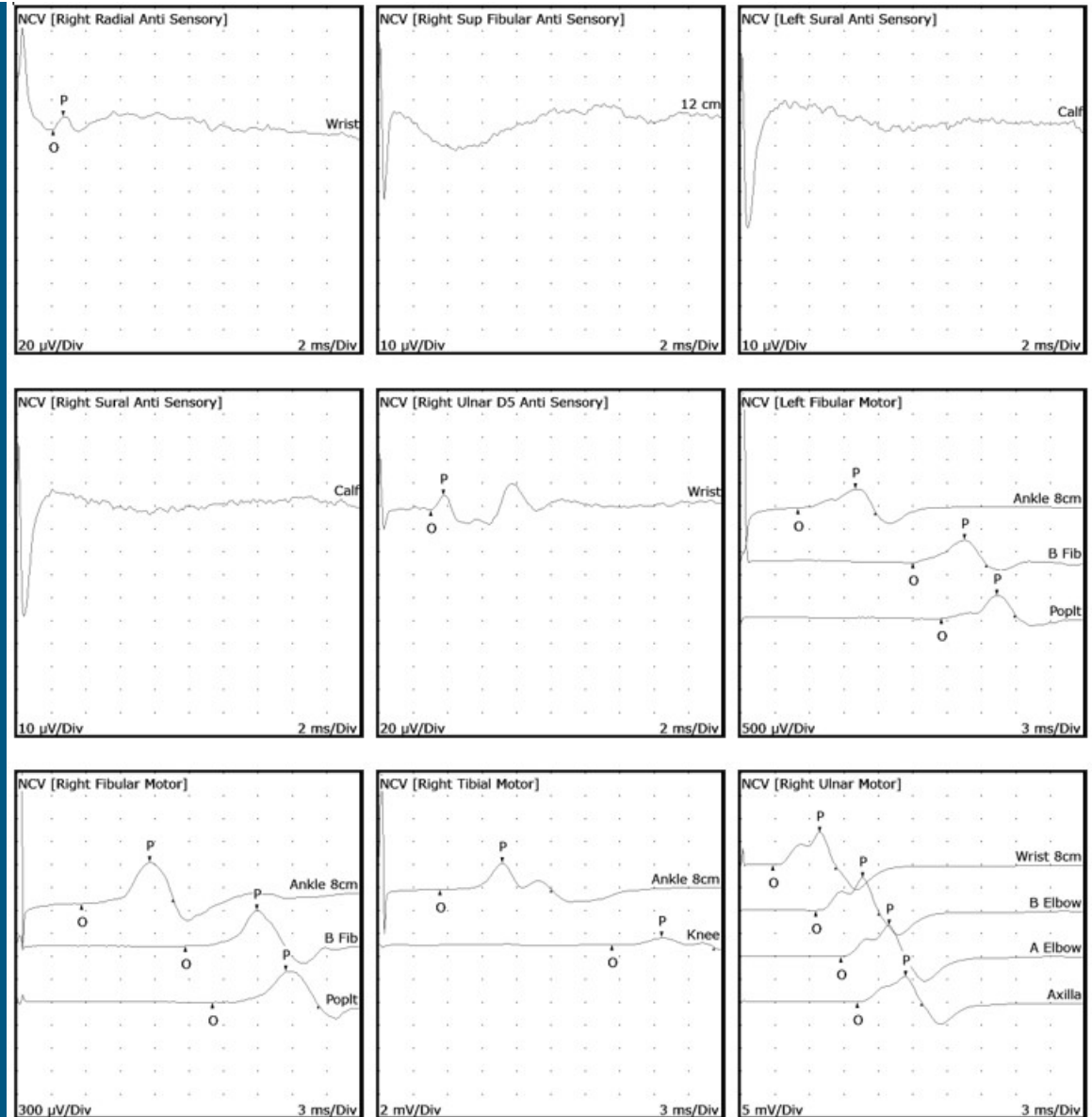
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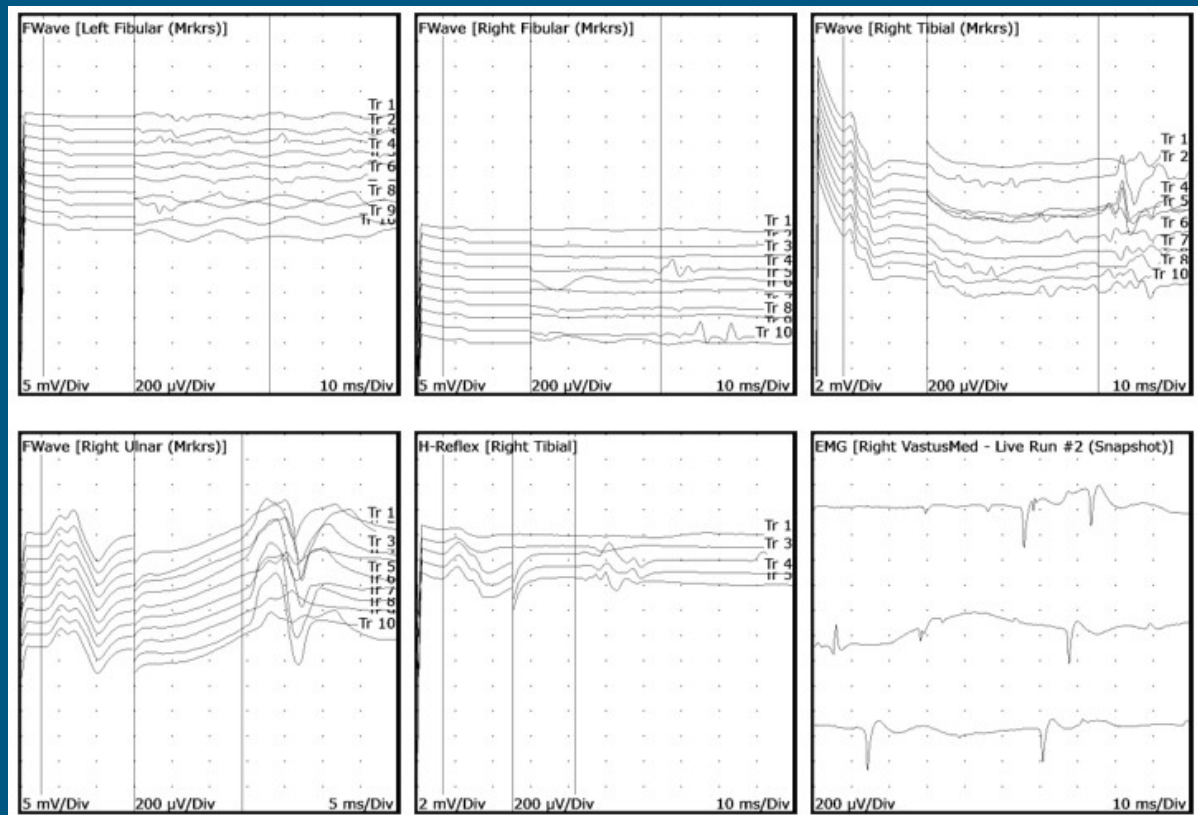
Case 2 Waveforms



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Case 2 Waveforms

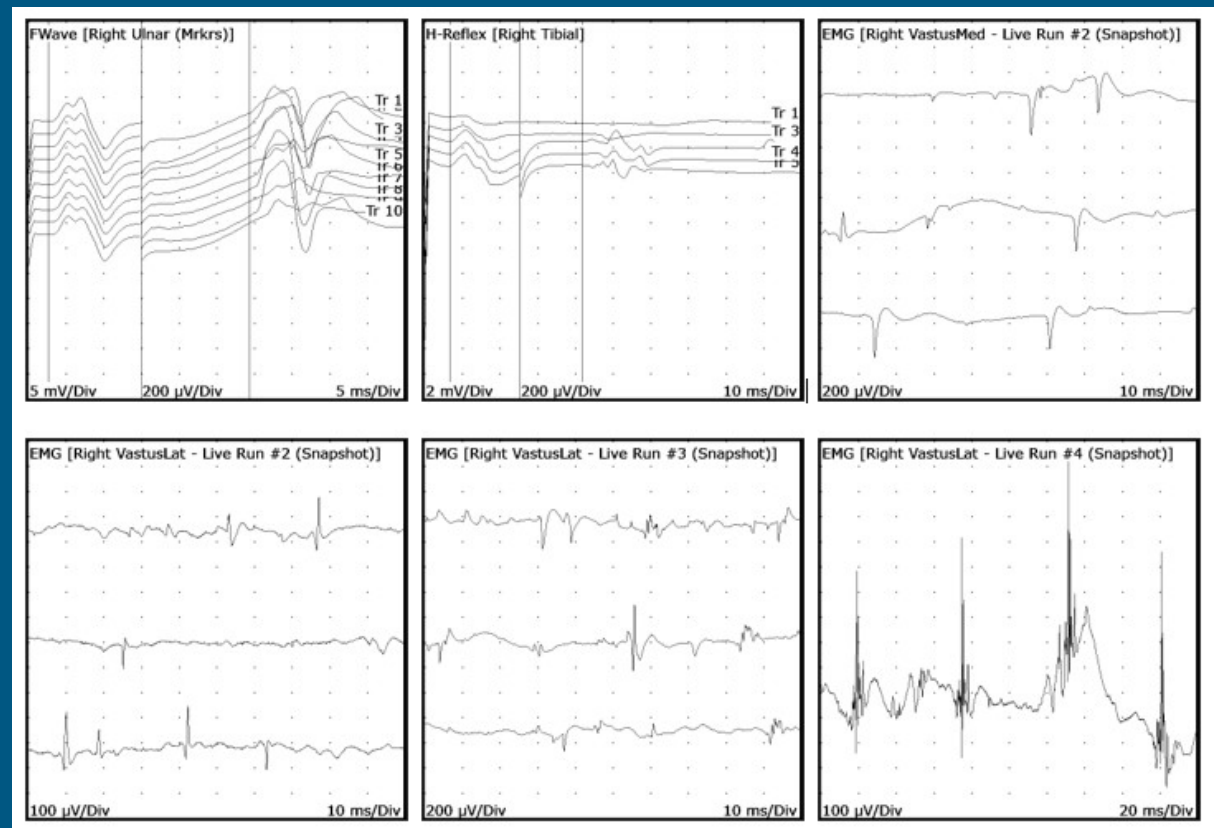


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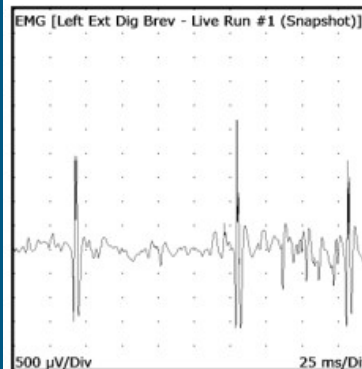
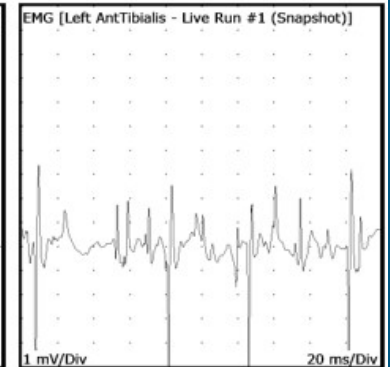
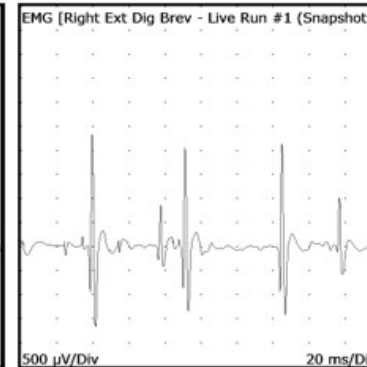
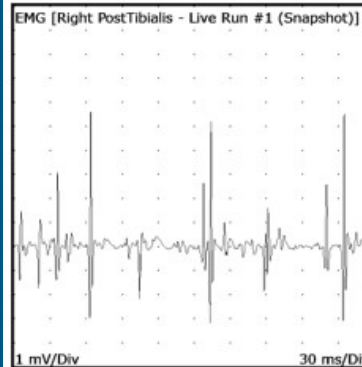
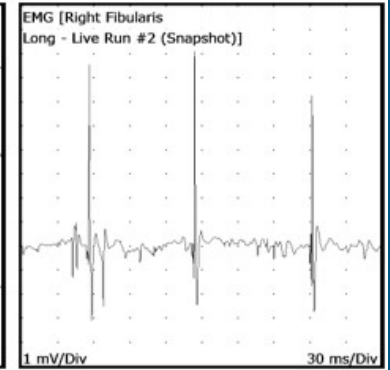
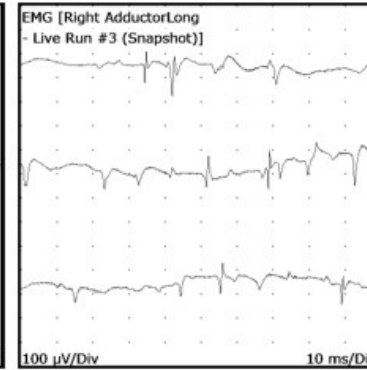
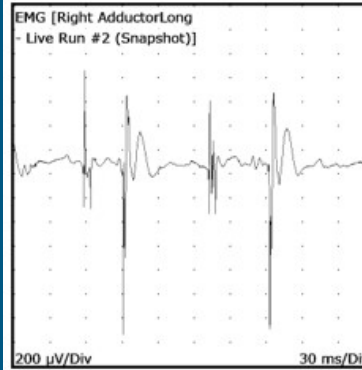
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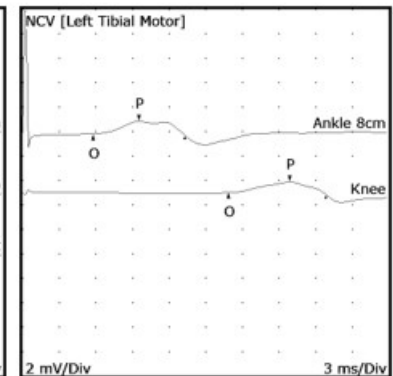
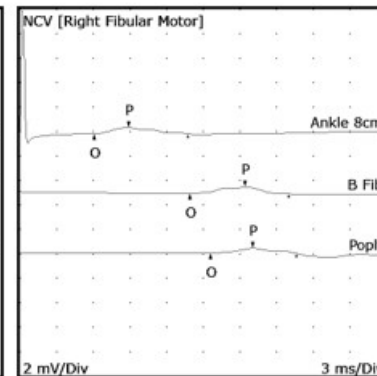
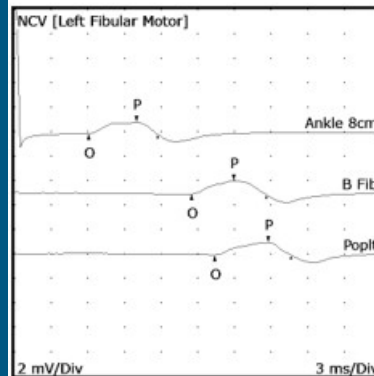
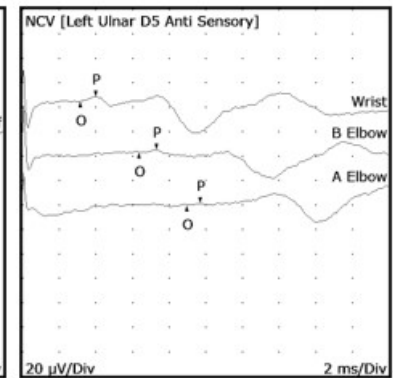
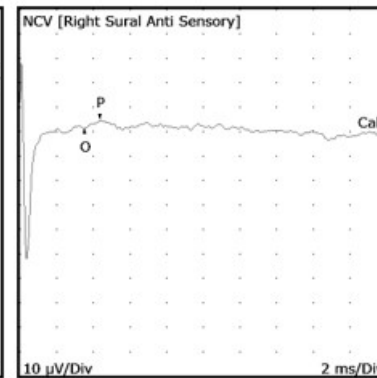
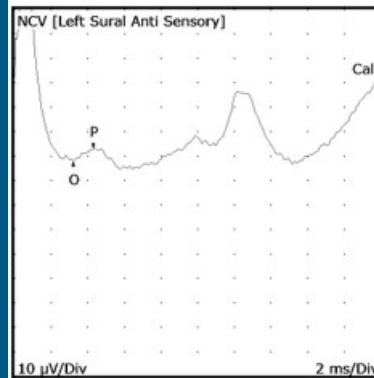
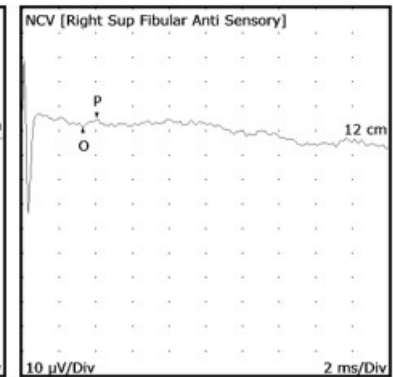
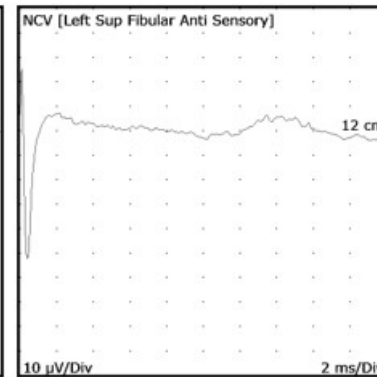
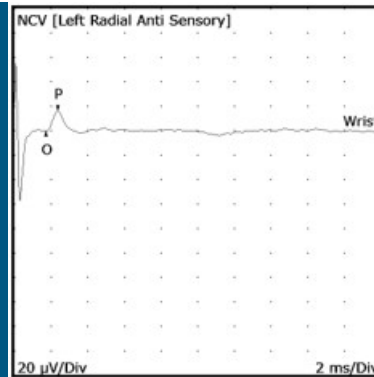
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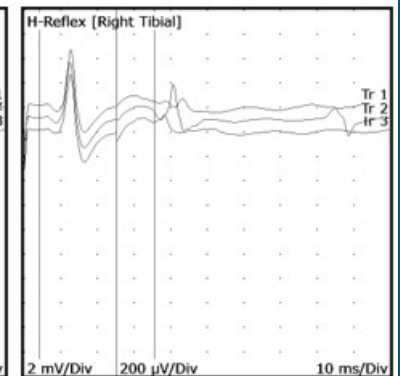
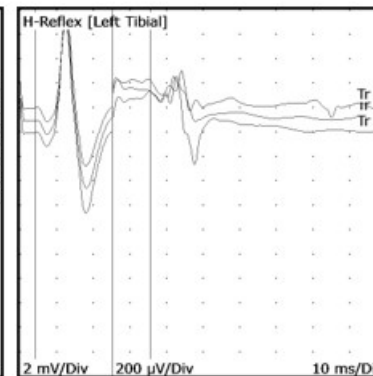
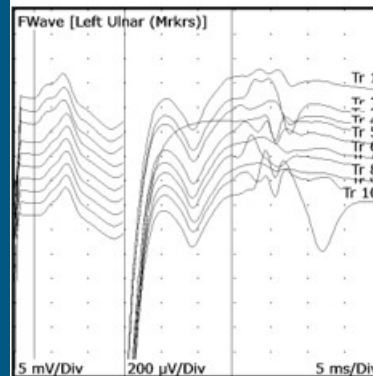
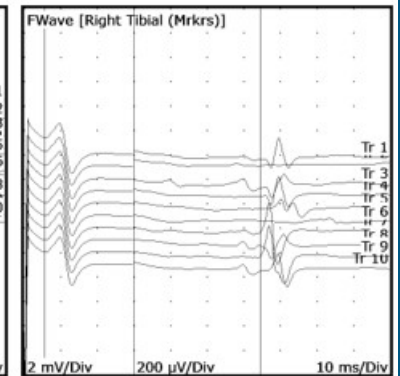
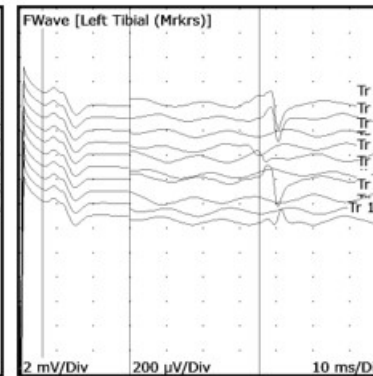
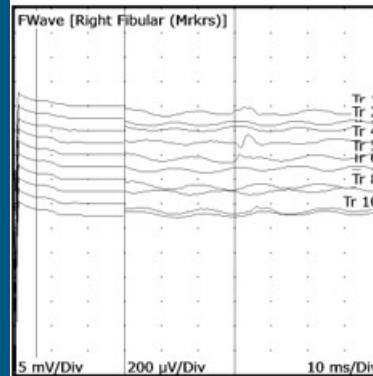
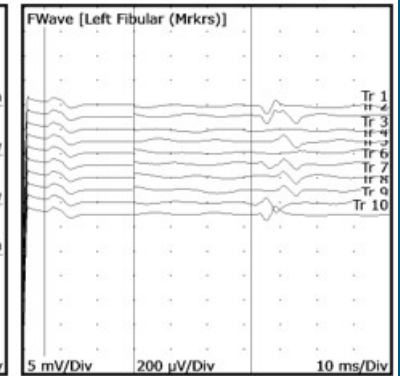
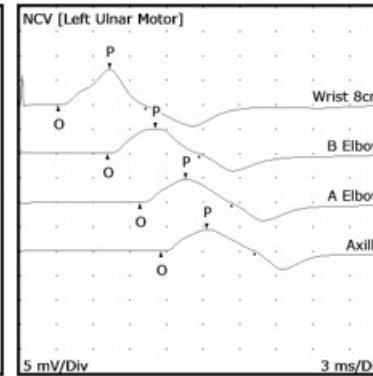
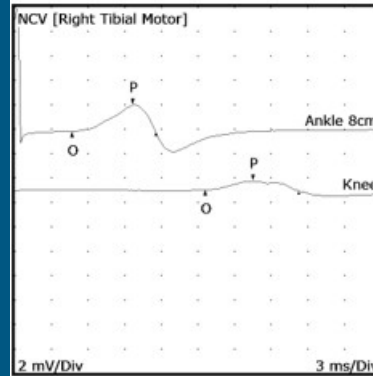
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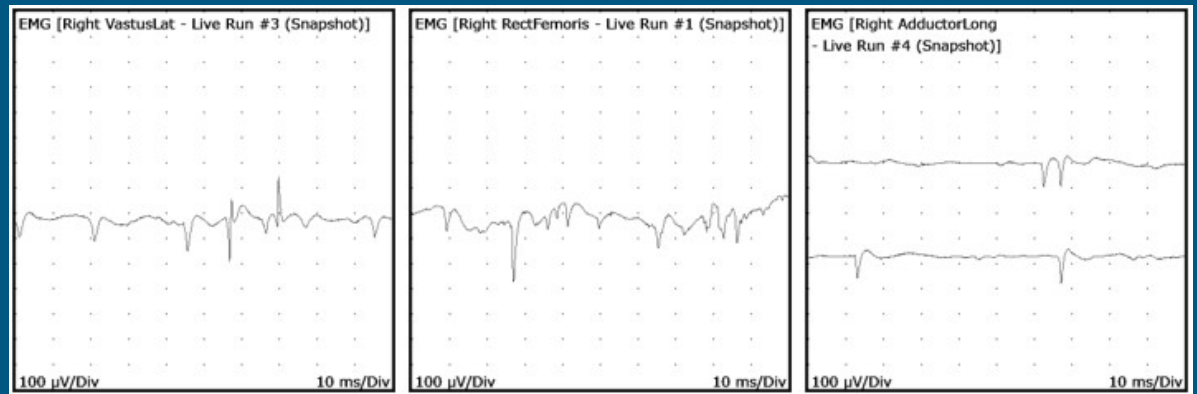
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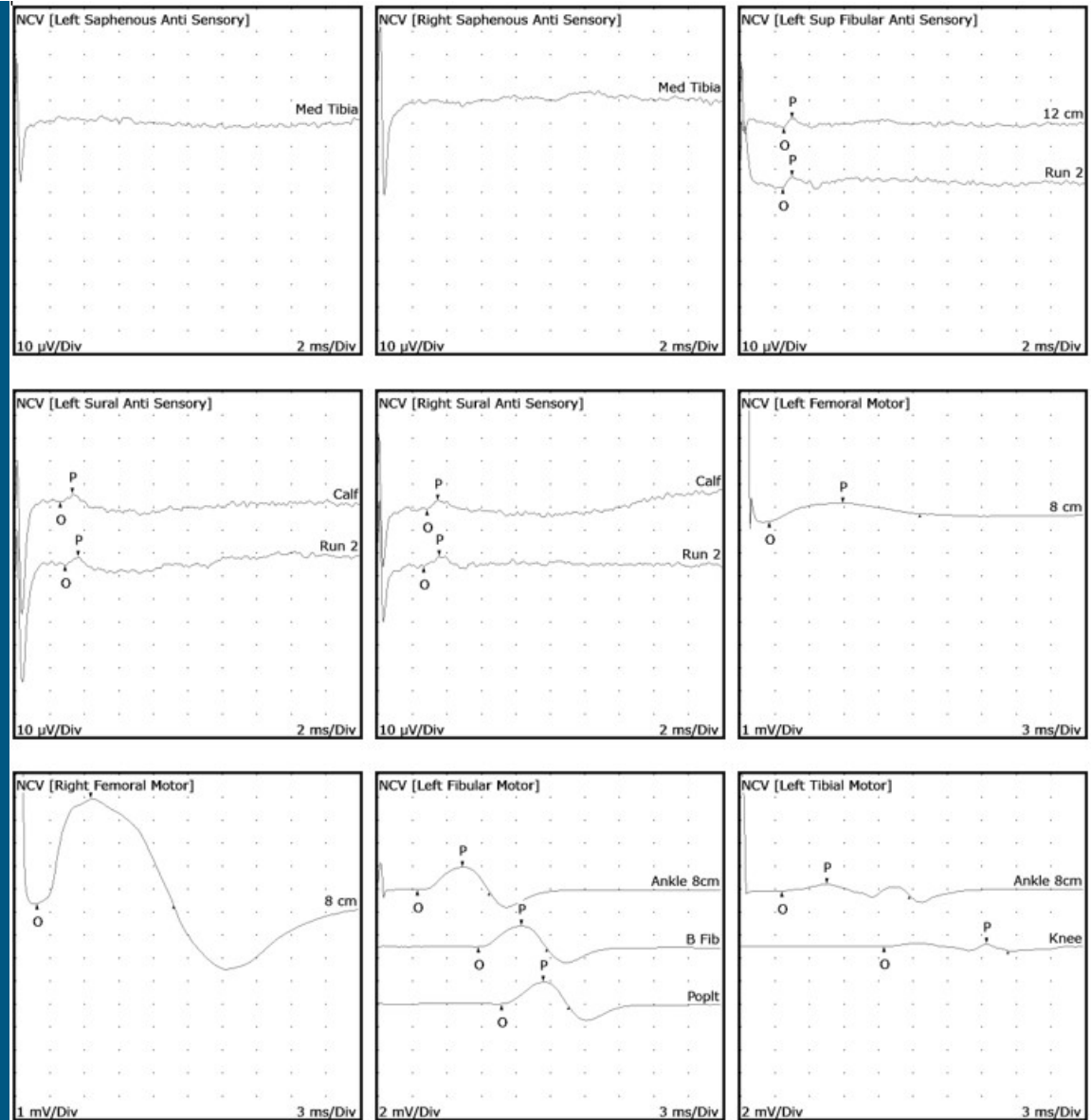
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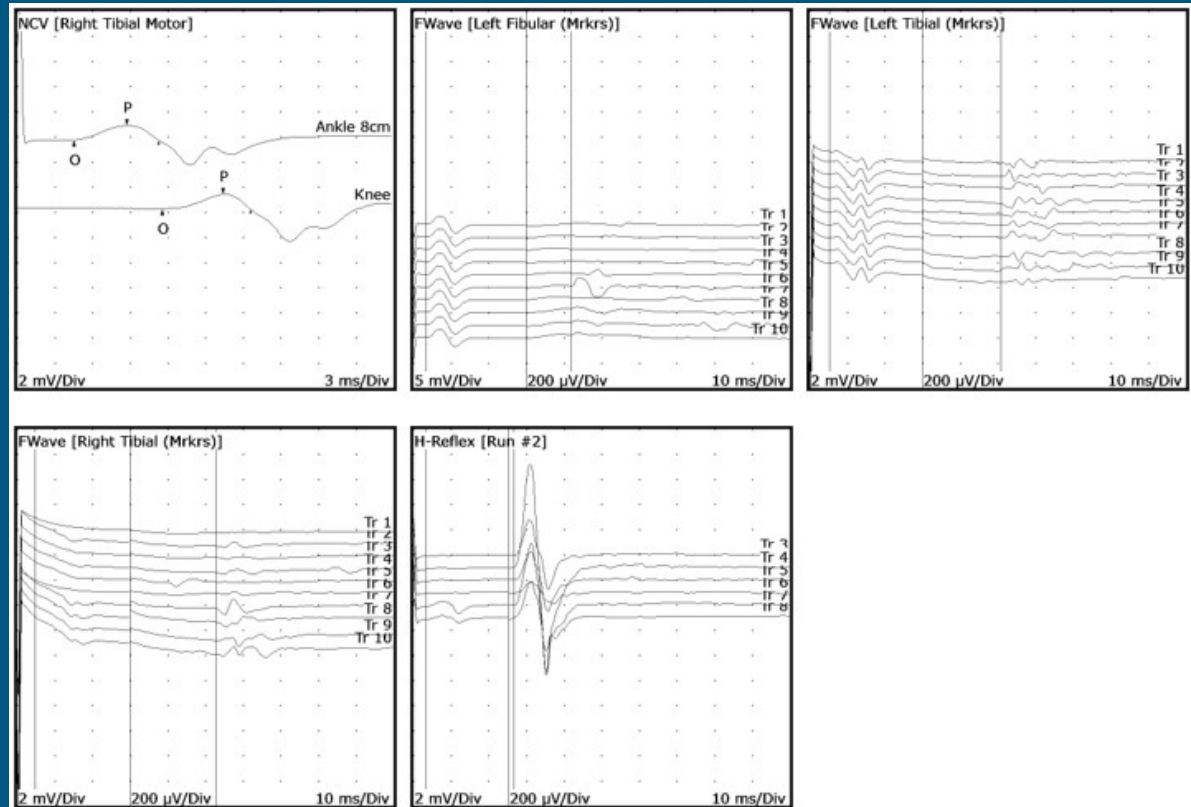
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Case 4 Waveforms

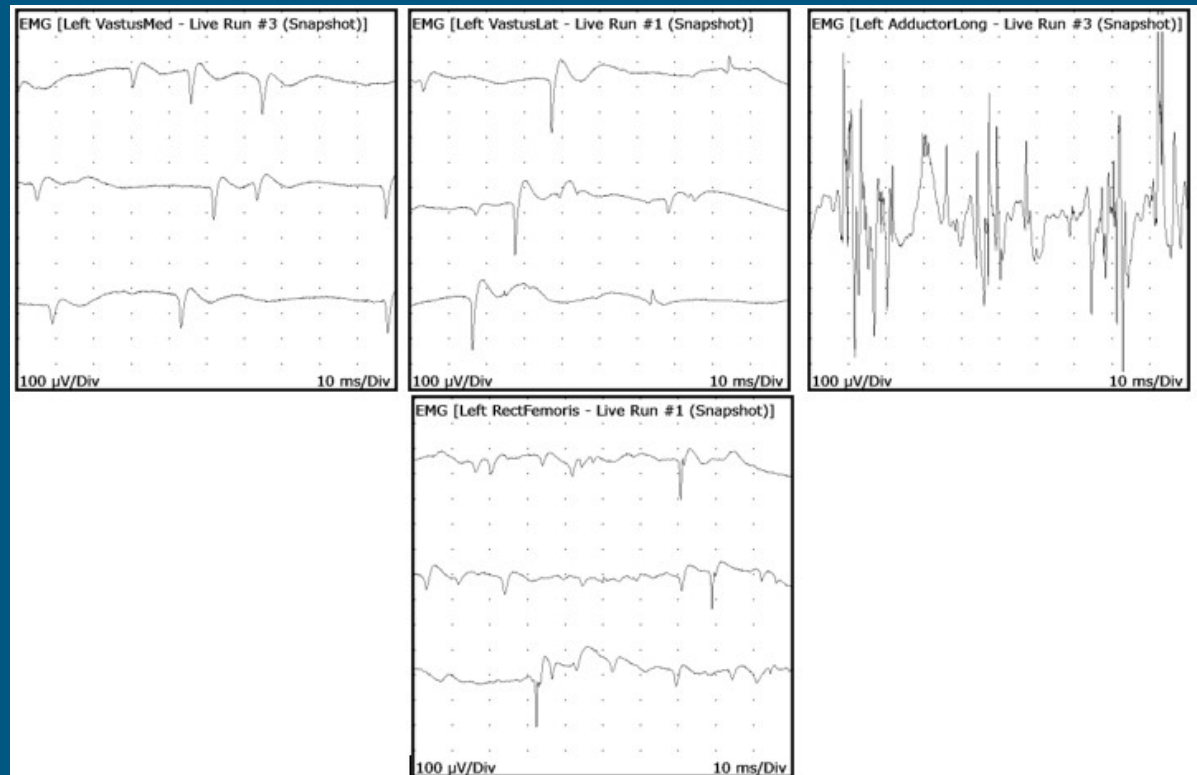


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Case 4

Waveforms



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Nerve Transfers: Past, Present and Future

David A. Fuller, MD
Professor of Orthopaedic Surgery

Rocky Mountain University
2026 Annual Meeting
Provo, Utah
March 28, 2026

- No financial disclosures relevant

Objectives

- Focus on Some Active Areas of Nerve Surgery that are newer to practices:
 - Nerve Transfers
- Discuss the need for early and accurate assessments related to decision making
- Brief Review Nerve Trauma
- Consider Future Areas of Growth for Surgical Care of Nerve Injuries

Core Values

Collaborative

We learn and create within and across teams to innovate, foster change, implement strategy, ensure accountability, and celebrate achievement.



**Rocky Mountain
University**
of Health Professions

New Jersey: Cooper Hospital



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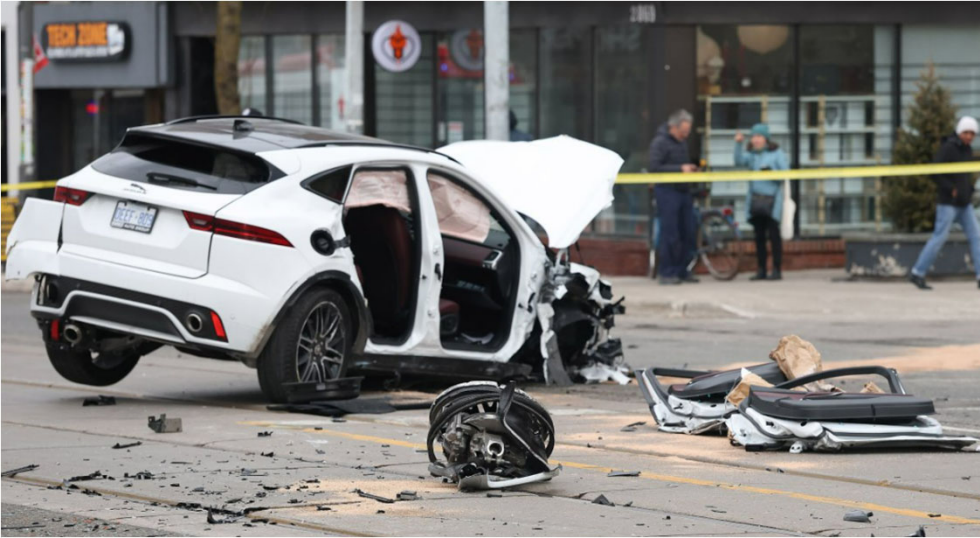
Cooper Today



Cooper Hospital – 3 new towers (\$3 billion plan)



Accidents





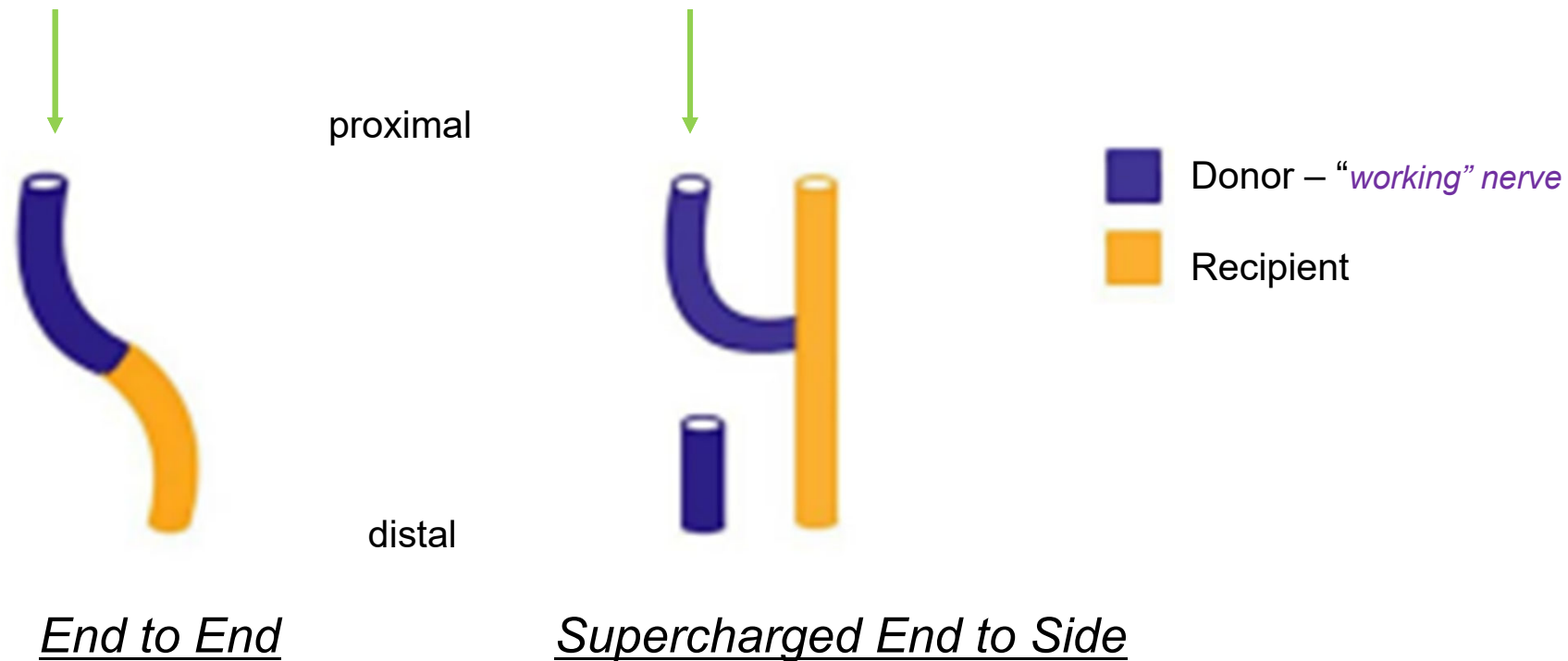
Nerve Trauma

4:45	5:25	Ting Oh, PT, ECS	<i>Severe Brachial Plexopathy Post Fall and Fracture Dislocation of the Shoulder</i>
------	------	------------------	--

9:40	10:20	Robert Sellin, PT DSc, ECS	<i>Severe Brachial Plexopathy following Shoulder Hemiarthroplasty: A Study that Includes the Effect of Contextual Factors on Outcomes</i>
------	-------	-------------------------------	---

A surgical procedure that redirects a functional donor nerve to reinnervate a paralyzed or denervated recipient nerve

- Originally developed for severe brachial plexus injuries with root avulsions, but now widely used for various peripheral nerve injuries
- Indications increasingly expanding for Spinal Cord Injury and Stroke

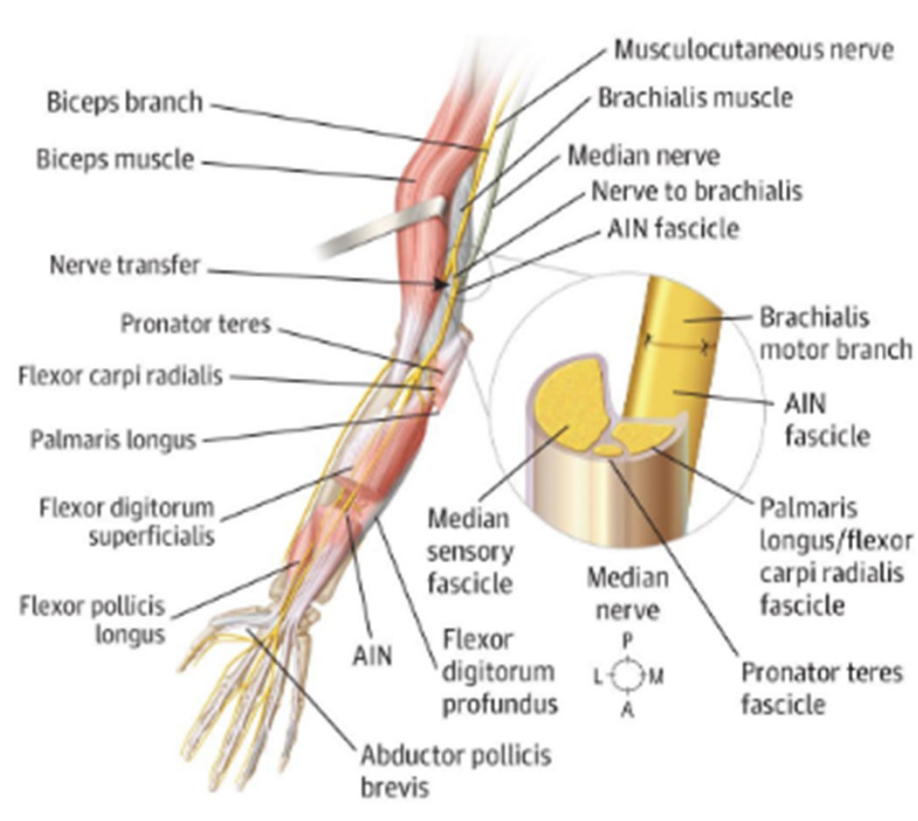


End to End: Recipient nerve has no chance of spontaneous recovery

Supercharged End to Side: Recipient nerve has some chance of spontaneous recovery or has some current limited function

Example of Nerve Transfer: Spinal Cord Injury C7 Paraplegia

Brachialis Branch of Musculocutaneous Nerve to AIN



Goal: Restoration of Hand Grasp and Pinch

Nerve transfer principles

- The donor nerve must be expendable
- The nerve coaptation should be performed as close to the target muscles as possible
- Synergistic innervation patterns desirable
- One motor axon can reinnervate multiple recipient axons thru collateral sprouting
- Cortical plasticity plays a role in functional recovery

Nerve Transfer Challenges

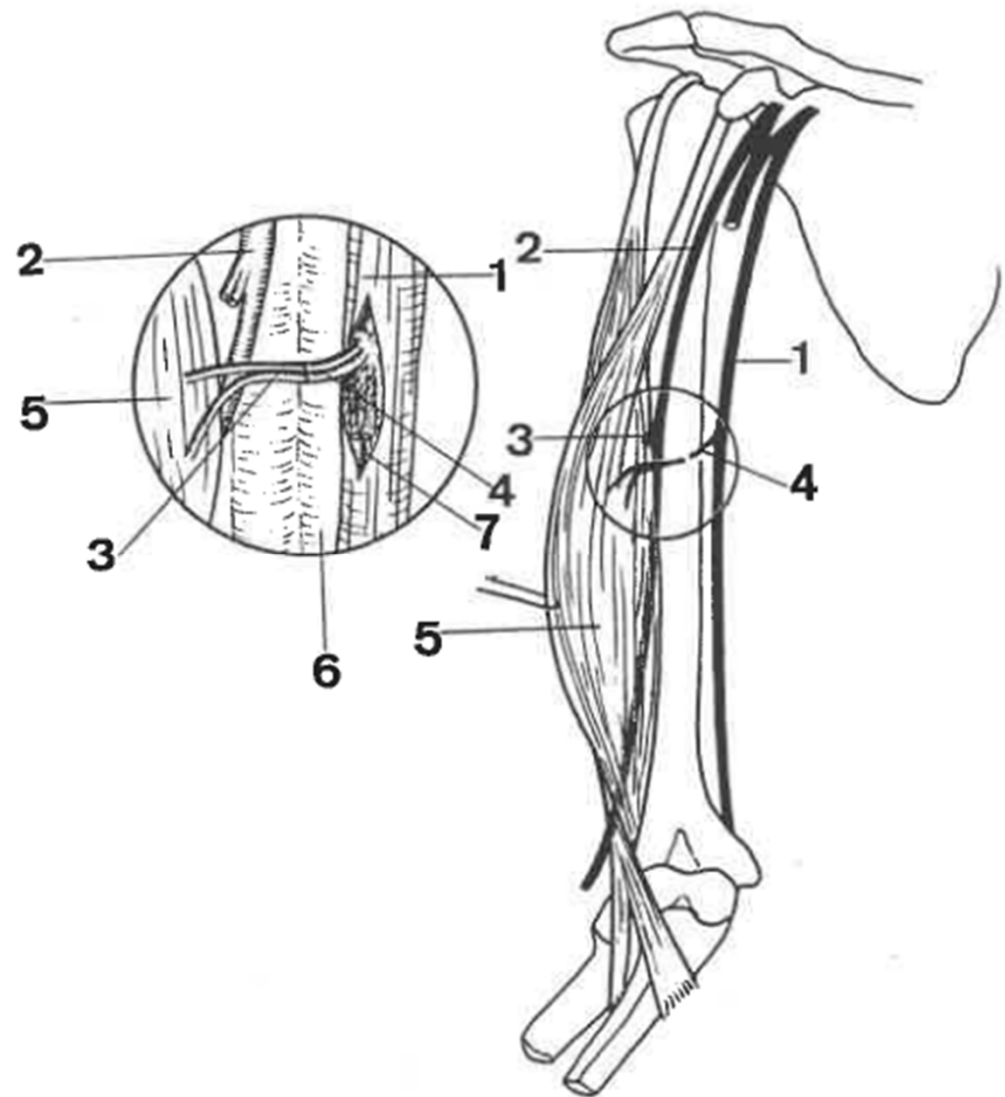
- Donor Morbidity
- Axon Counts
- Predictability of Recovery

Oberlin Transfer 1994

Nerve transfer to biceps muscle using a part of ulnar nerve for C5–C6 avulsion of the brachial plexus: anatomical study and report of four cases

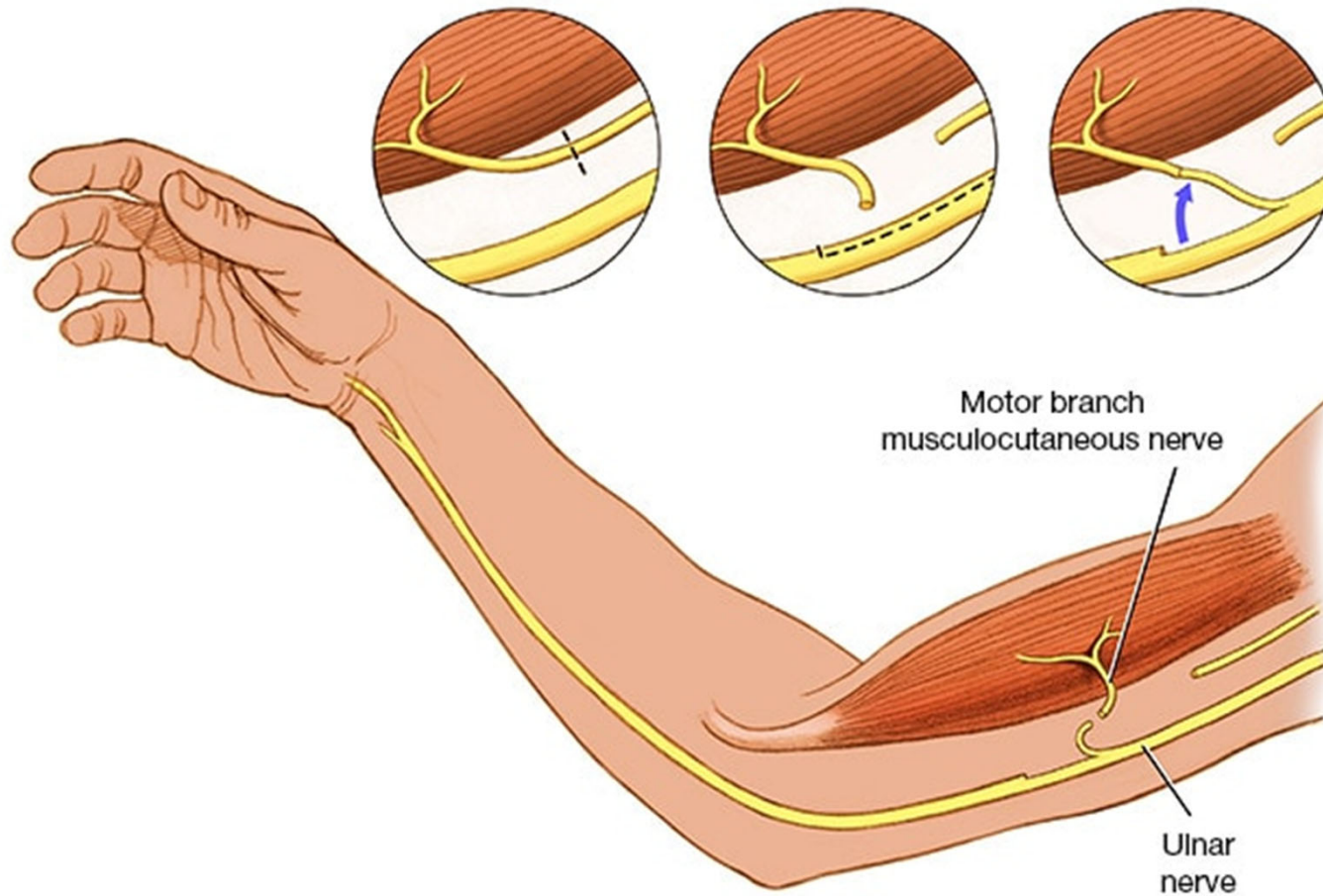
C Oberlin¹, D Béal, S Leechavengvongs, A Salon, M C Dauge, J J Sarcy

JHS 1994



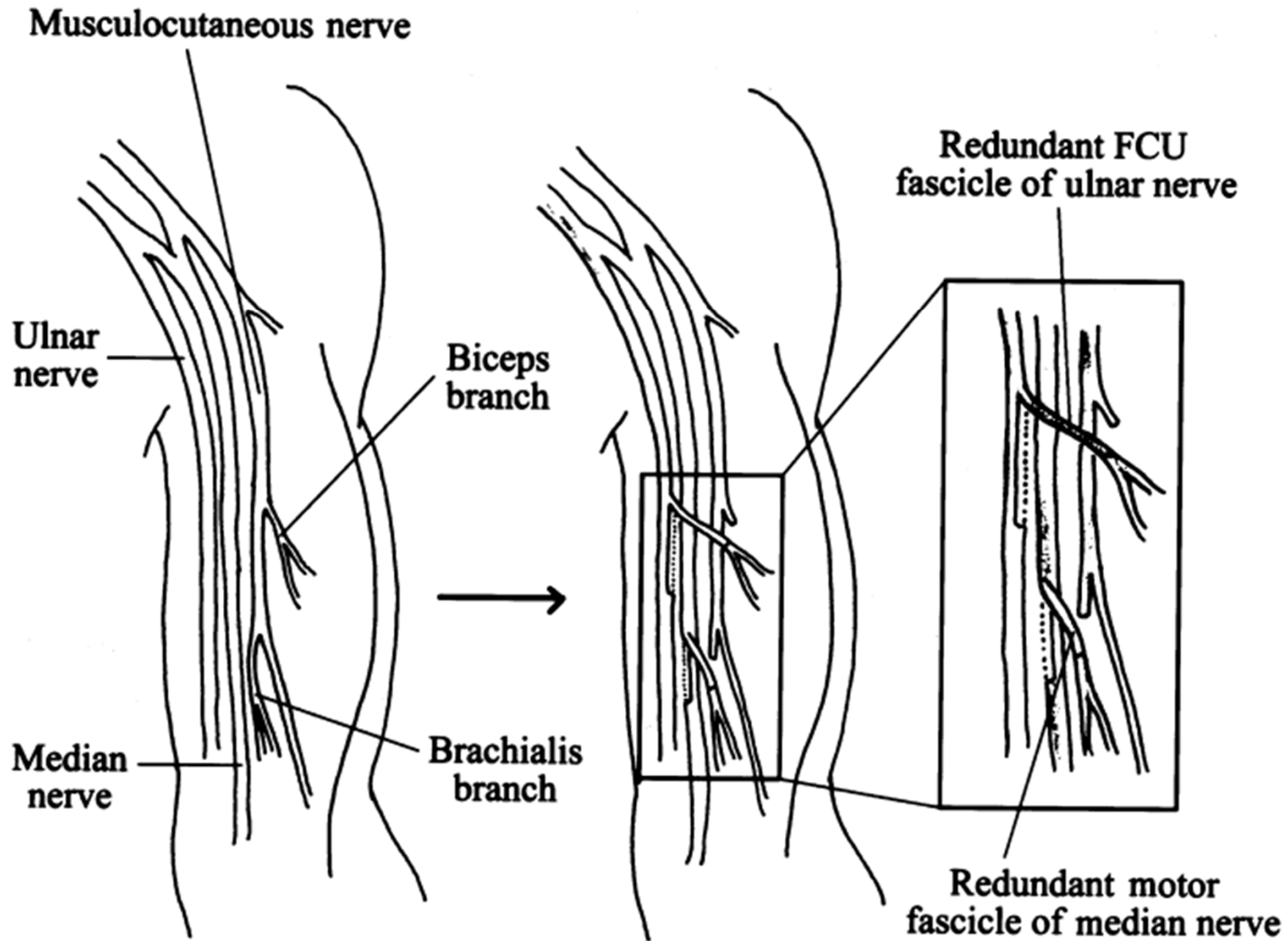
“10% of Ulnar Nerve Transferred”

Oberlin: Technique

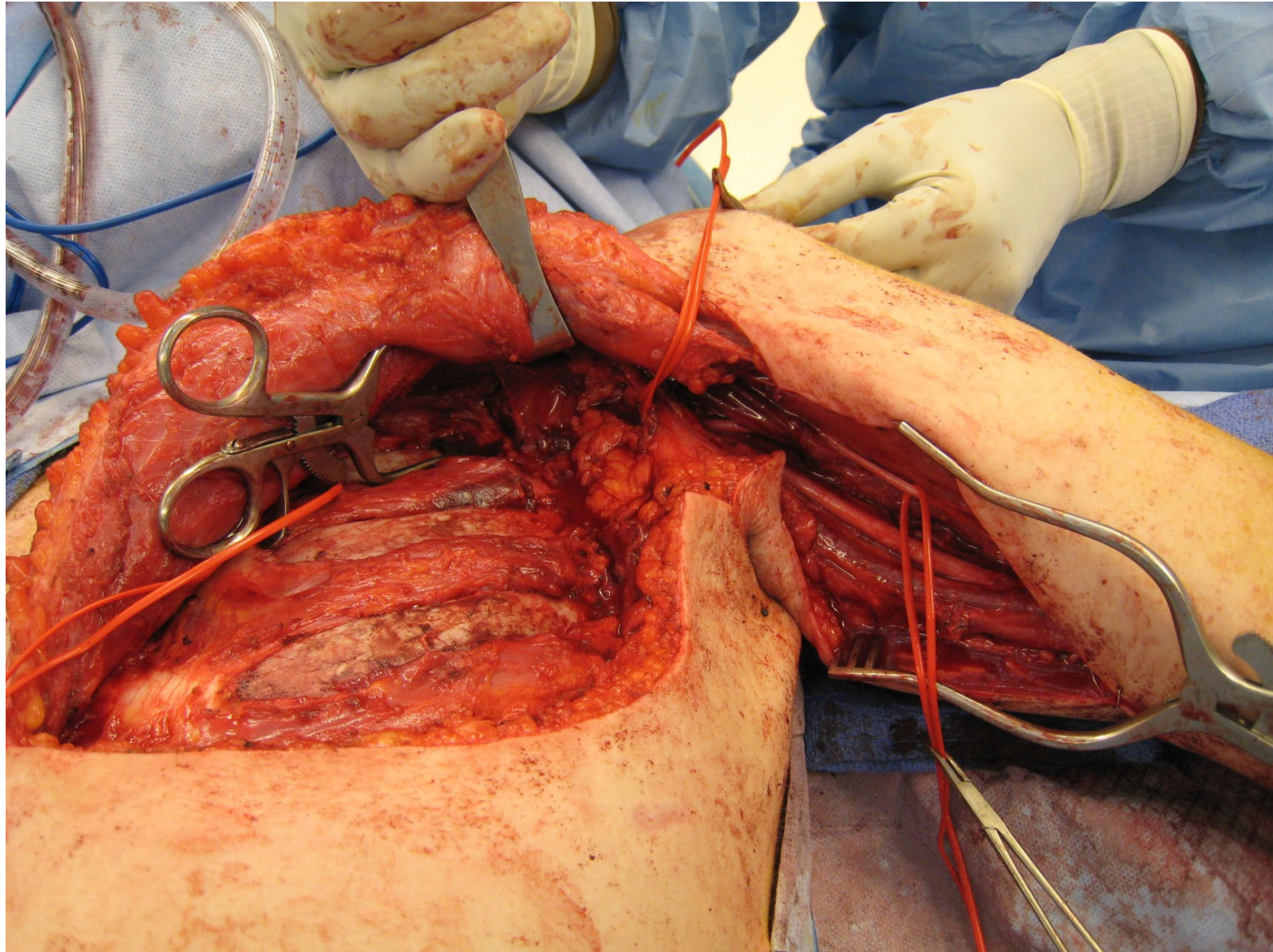


An “End to End” Nerve Transfer

Double Fascicular Transfer to Restore Elbow Flexion

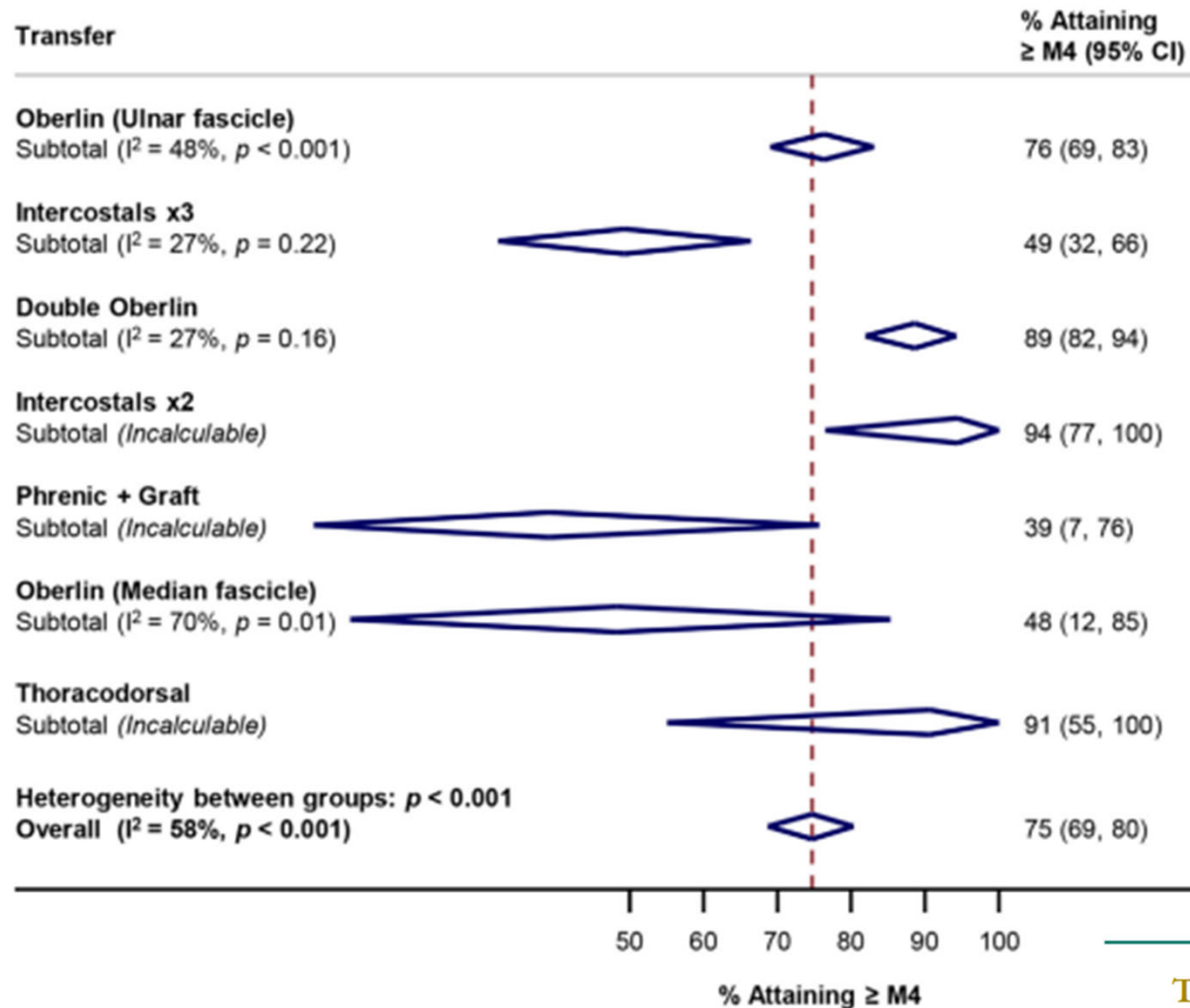


Intercostal Transfer to Restore Elbow Flexion



Nerve Transfer for Elbow Flexion: Outcomes

Nerve Transfers to Restore Elbow Flexion in Partial Brachial Plexus Injuries



EDITOR'S CHOICE

The Effectiveness of Different Nerve Transfers
in the Restoration of Elbow Flexion in Adults
Following Brachial Plexus Injury: A Systematic
Review and Meta-Analysis

Chung Yan Vernon Lee, MBBS,* Elliott Cochrane, MBChB, BSc,* Misha Chew, MBBS,†
Robert D. Bains, MBChB, BSc,* Gráinne Bourke, MBBCh, BAO,*‡ Ryckie G. Wade, MSc, PhD*‡

5/27/2025



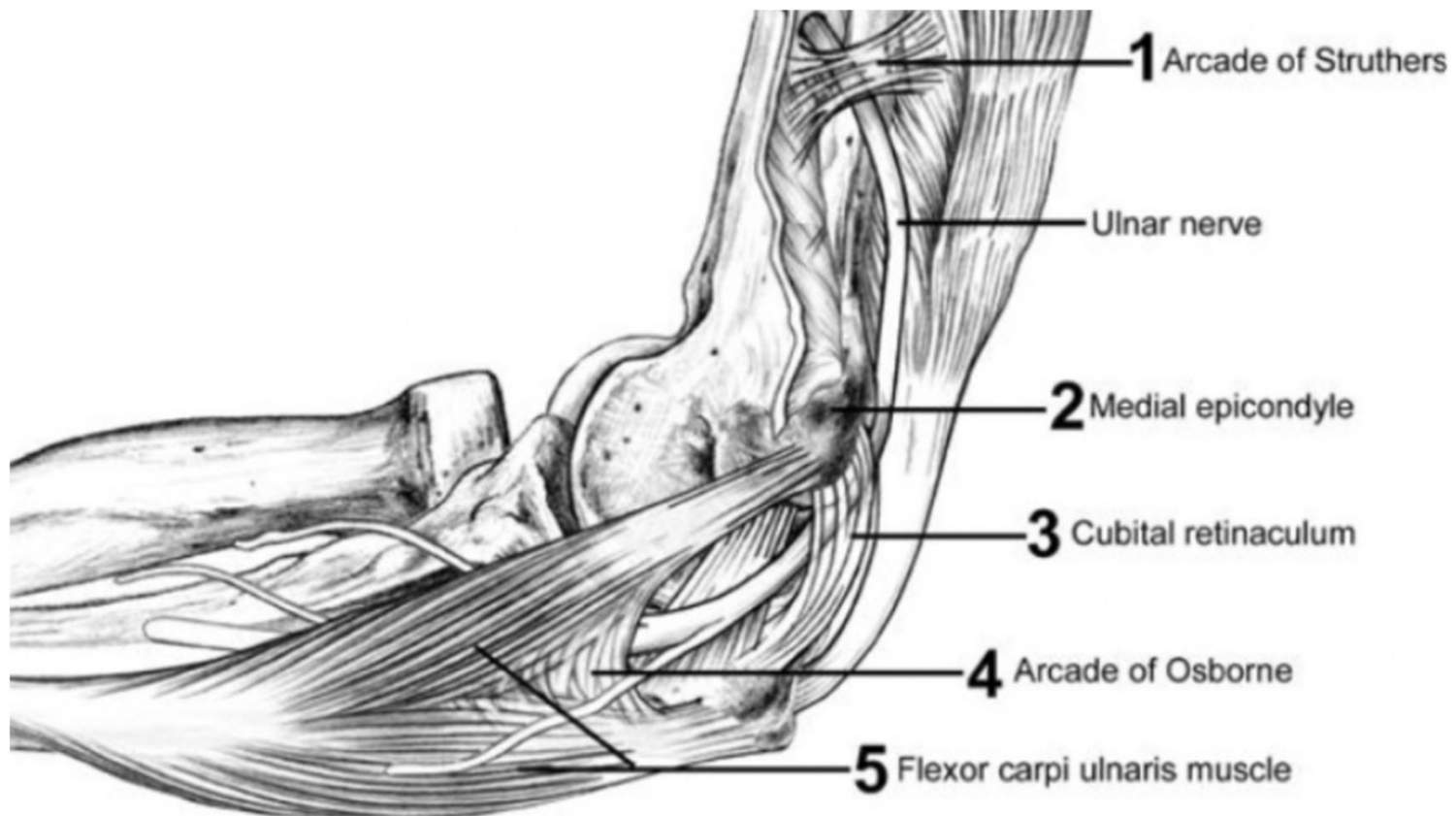
Ulnar Neuropathy at Elbow

12:50

1:30

Nathan Savage, PT,
DPT, PhD, ECS

*Relationship Between Electrodiagnostic and Sonographic
Categorizations of Ulnar Neuropathy at the Elbow*



Ulnar Clawing



- High Grade Trauma or Laceration ?
- Compressive Neuropathy ?

Anterior Interosseous Nerve Transfer
has become an established treatment
choice

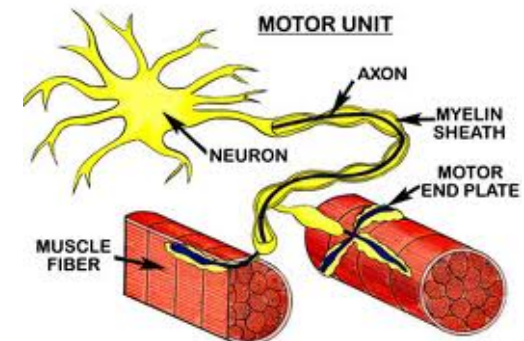
AIN Transfer for High Ulnar Neuropathy

Supercharged End-to-Side Anterior Interosseous to Ulnar Motor Nerve Transfer for Intrinsic Musculature Reinnervation

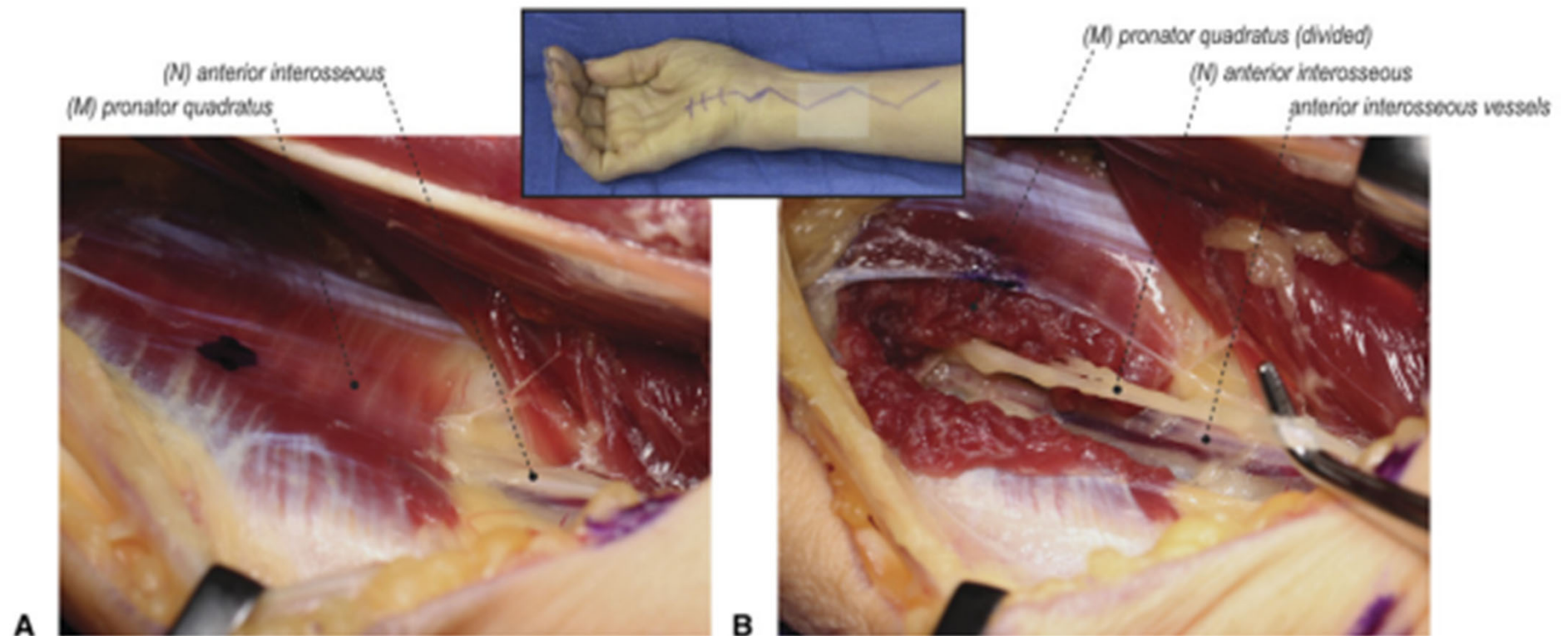
John Barbour, MD, Andrew Yee, BS, Lorna C. Kahn, BS, Susan E. Mackinnon, MD

JHS, 2012

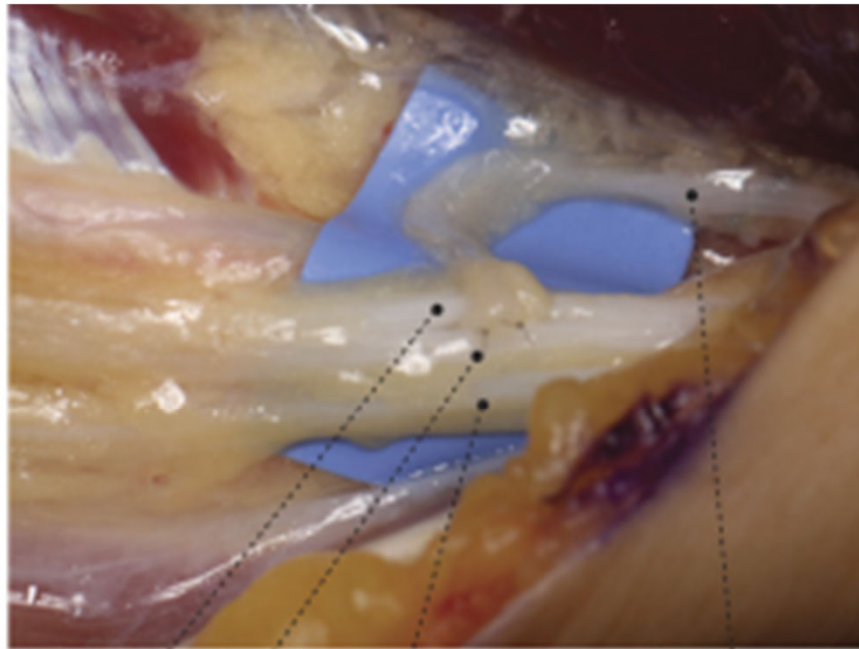
“...we believe that the SETS procedure might have broad clinical utility for 2nd and 3rd degree axonotmesis nerve injuries, to augment partial recovery and/or “babysit” motor end plates until the native parent axons regenerate to target ...”



AIN Transfer: Technique

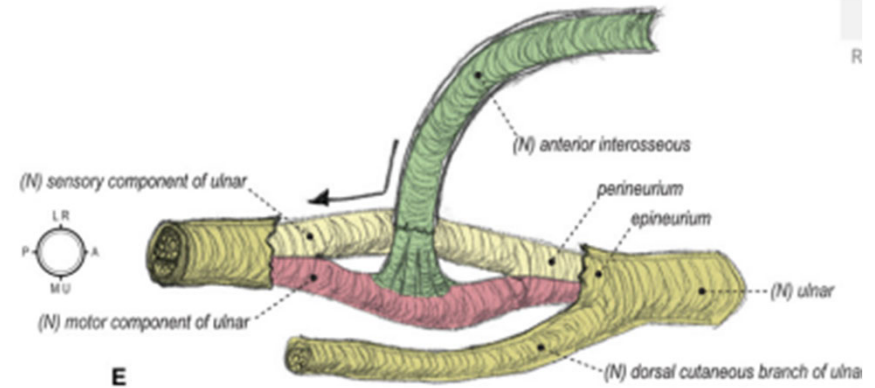


AIN Transfer: Technique End to Side



C

(N) sensory component of ulnar
(N) motor component of ulnar
(N) dorsal cutaneous branch of ulnar
(N) anterior i



E

Supercharged end-to-side anterior interosseous nerve transfer to restore the intrinsic motor function in osteoarthritis-induced cubital tunnel syndrome

Soo Min Cha, MD, PhD^{a,*}, Seung Hoo Lee, MD^b, In Ho Ga, MD^c, Yong Hwan Kim, MD^d

JSES 2025

27 Patients, mean age 54, high ulnar nerve lesions with intrinsic wasting
Grip Strength, Key Pinch and DASH scores all improved

“ in cases of compressive ulnar nerve palsy associated with elbow arthritis, SETS AIN transfer should be considered ...”

HAND/PERIPHERAL NERVE

Five Operations That Give the Best Results after Brachial Plexus Injury

Maldonado et al,
Plastic and Reconstructive Surgery, 2017

- Oberlin (Ulnar → MSC)
- Leechavengvongs Procedure (Radial → Axillary)
- Free Functional Muscle Transfer for Elbow Flexion and Rudimentary Hand Function
- Radial Nerve Tendon Transfers after Posterior Cord Injury
- C5-C6 Nerve Grafting after Obstetric Brachial Palsy Injury

HAND/PERIPHERAL NERVE

Five Operations That Give the Best Results after Brachial Plexus Injury

Maldonado et al,
Plastic and Reconstructive Surgery, 2017

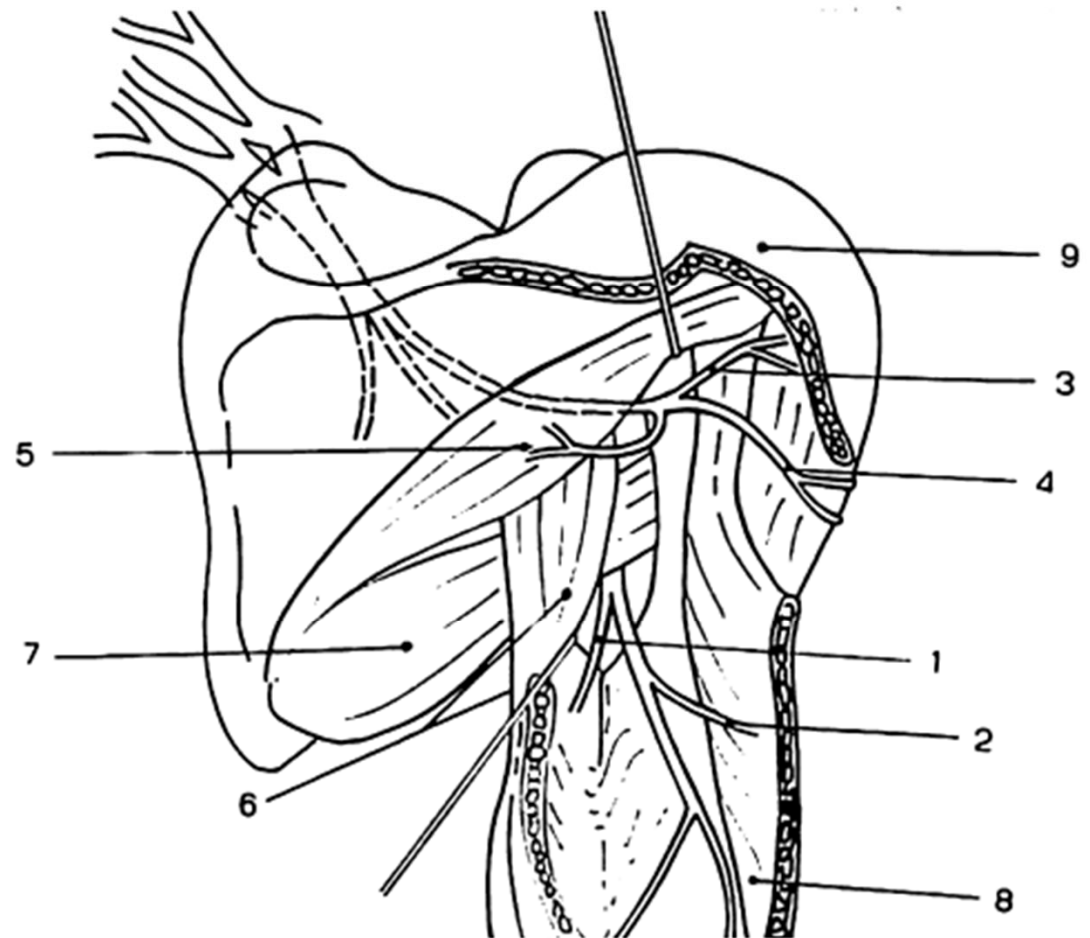
- Oberlin (Ulnar → MSC)
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- Radial Nerve Tendon Transfers after Posterior Cord Injury
- C5-C6 Nerve Grafting after Obstetric Brachial Palsy Injury

Leechavengvongs Procedure

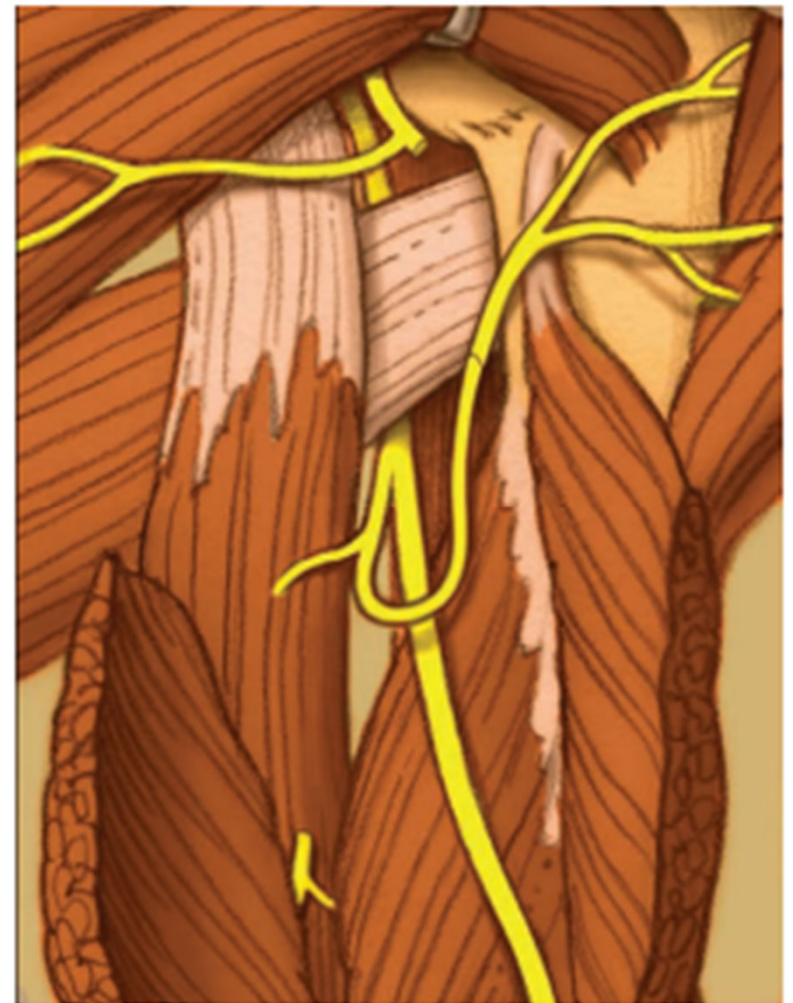
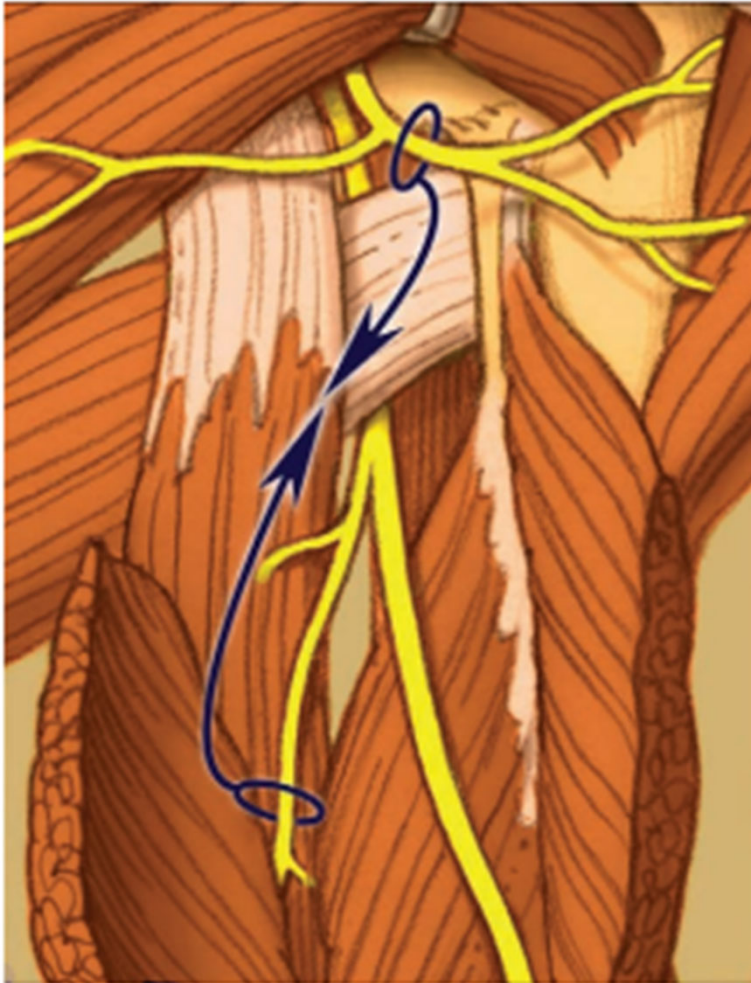
Nerve Transfer to Deltoid Muscle Using the Nerve to the Long Head of the Triceps, Part I: An Anatomic Feasibility Study

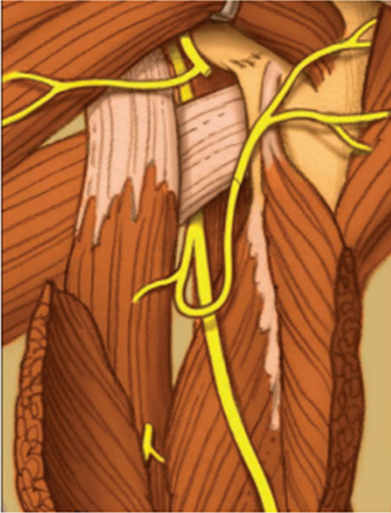
Kiat Witoonchart, MD, Somsak Leechavengvongs, MD,
Chairoj Uerpaiojkit, MD, Phairat Thuvasethakul, MD,
Vudthipong Wongnopsuwan, MD, *Bangkok, Thailand*

The Journal of Hand Surgery / Vol. 28A No. 4 July 2003



Leechavengvongs Procedure





80% success rate

Nerve transfers for axillary nerve repair in brachial plexus injuries: results from 206 patients

Pavel Haninec, Jan Hradecky, Martin Ouzky, Filip Samal, Libor Mendl

J Neurosurg Spine. 2025

3 Favored Sets of Transfers:

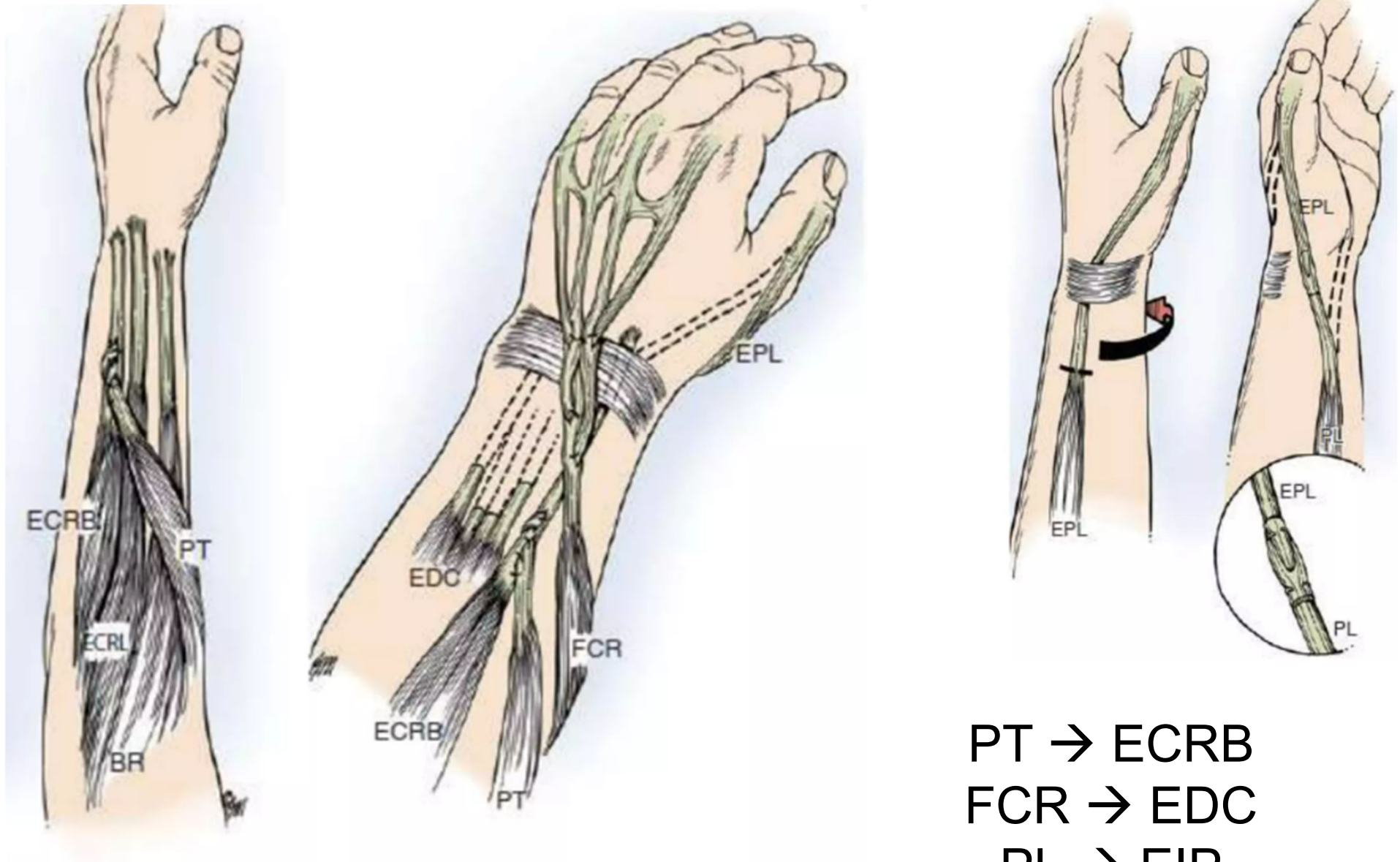
→ FCR

→ Superficialis

→ FCU

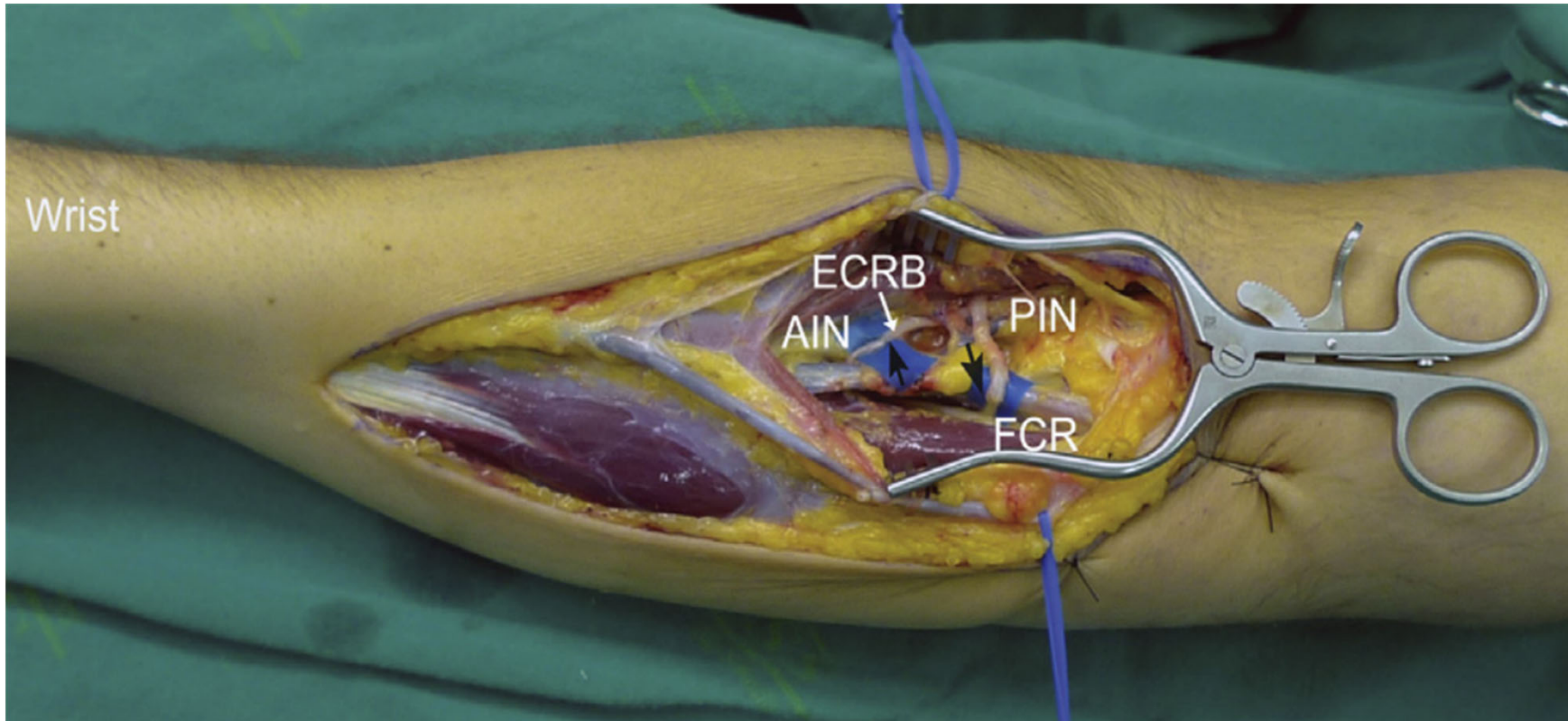
BEST COMBINATIONS OF TENDON TRANSFERS FOR RADIAL NERVE PALSY
FCR Transfer (Starr,¹⁰¹ Brand,¹⁷ Tsuge and Adachi¹⁰⁹)
PT to ECRB
FCR to EDC
PL to rerouted EPL
Superficialis Transfer (Boyes,¹³ Chuinard et al²³)
PT to ECRL and ECRB
FDS III to EDC
FDS IV to EIP and EPL
FCR to APL and EPB
FCU Transfer
PT to ECRB
FCU to EDC
PL to reroute EPL

Radial Nerve Tendon Transfers



PT → ECRB
FCR → EDC
PL → EIP

Nerve Transfers for Radial Nerve Palsy



Nerve Transfers:

- 1) AIN → ECRB Motor Branch
- 2) Motor Branch of FCR → PIN

Nerve Versus Tendon Transfer for Radial Nerve Paralysis Reconstruction

Jayme Augusto Bertelli, MD, PhD*†

14 patients nerve transfers, 13 patients tendon transfers (all chronic)

Conclusions:

1. we observed better outcomes in those that underwent nerve transfers”
2. room for improvement for improved thumb motion in both groups”

Nerve Transfers:

Composite Wrist and Finger extension possible
Better thumb CMC extension

Thumb Extension



Nerve Transfer example



Tendon Transfer example

Case Example: Cervical trauma

- 36 yo mother of 4
- Low energy trauma (fall) in setting of congenital cervical stenosis
- 2 months s/p fusion

Physical Exam:

Shoulder 0/5

Elbow Flexion 0/5

Elbow Extension 5-/5

WE/WF 5/5

Finger F/E/Abd 5/5



Cervical trauma

- 36 yo mother of 4
- Low energy trauma (fall) in setting of congenital cervical stenosis
- 2 months s/p fusion

Physical Exam:

Shoulder 0/5

Elbow Flexion 0/5

Elbow Extension 5-/5

WE/WF 5/5

Finger F/E/Abd 5/5



Consider Nerve Transfers to restore Shoulder and Elbow Function ?

Cervical trauma

- 36 yo mother of 4
- Low energy trauma (fall) in setting of congenital cervical stenosis
- 2 months s/p fusion

Physical Exam:

Shoulder 0/5

Elbow Flexion 0/5

Elbow Extension 5-/5

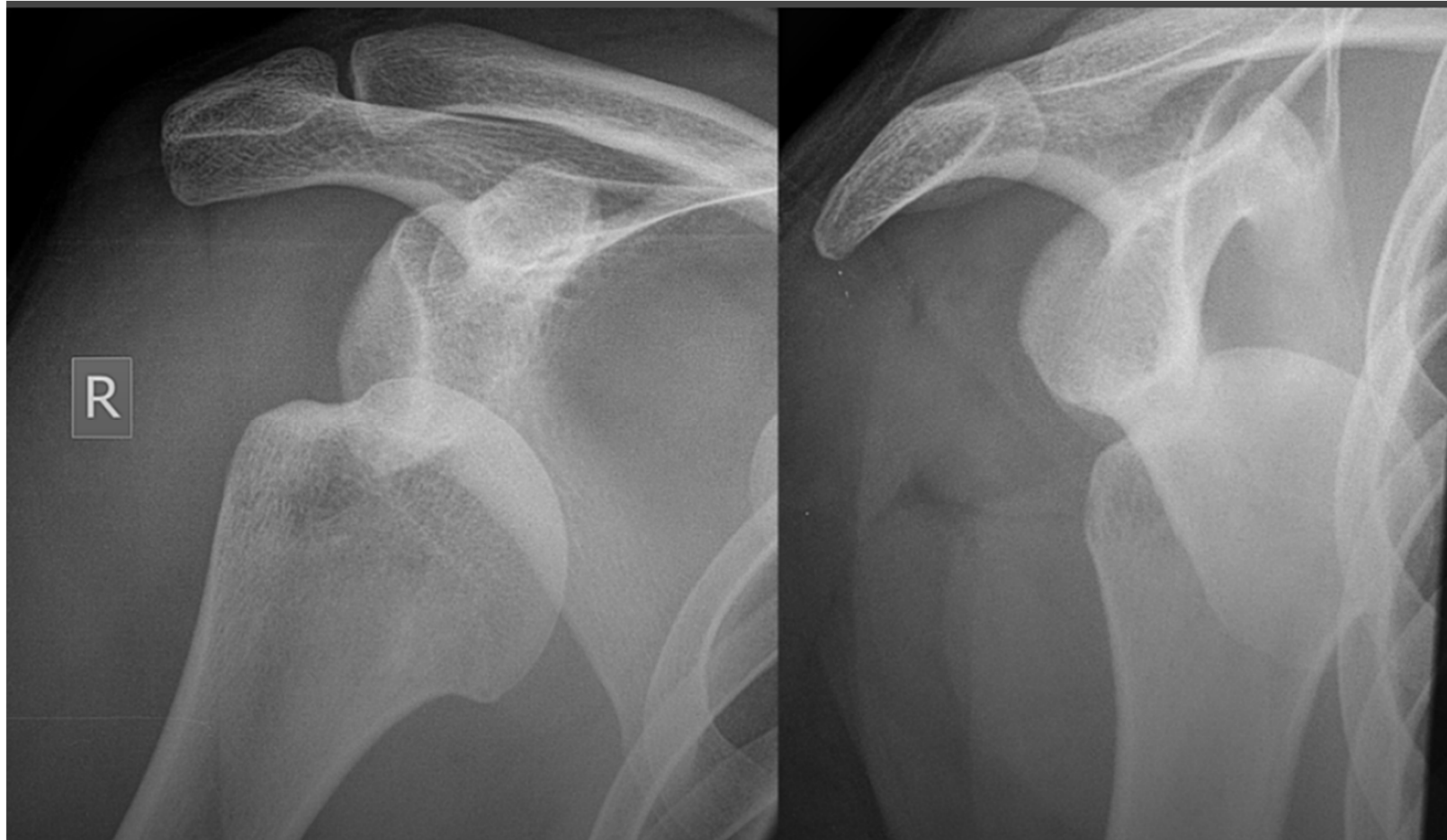
WE/WF 5/5

Finger F/E/Abd 5/5

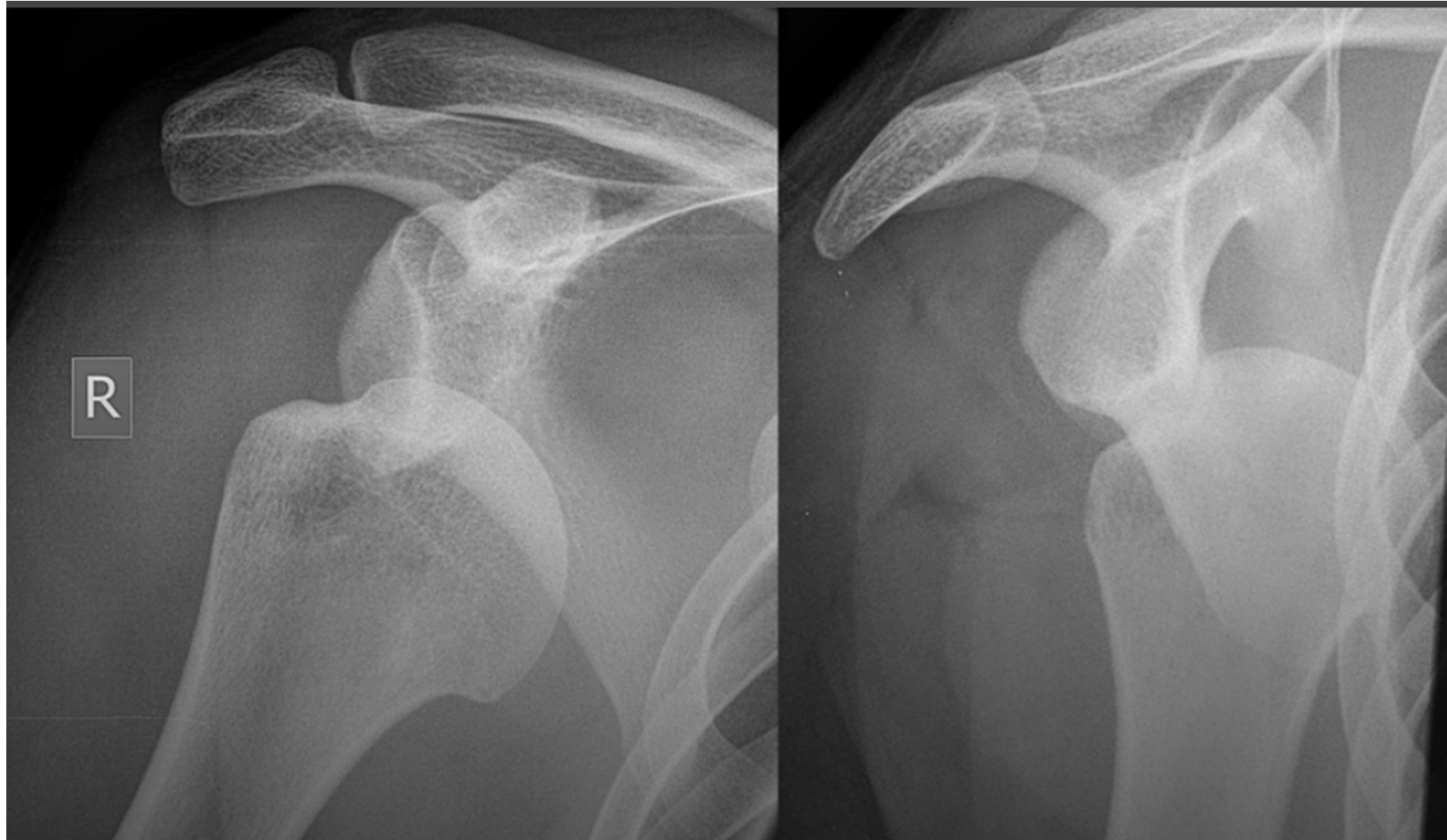


Consider Nerve Transfers to restore Shoulder and Elbow Function ?
(Donors, Timing, Technique)

Dislocated Shoulder

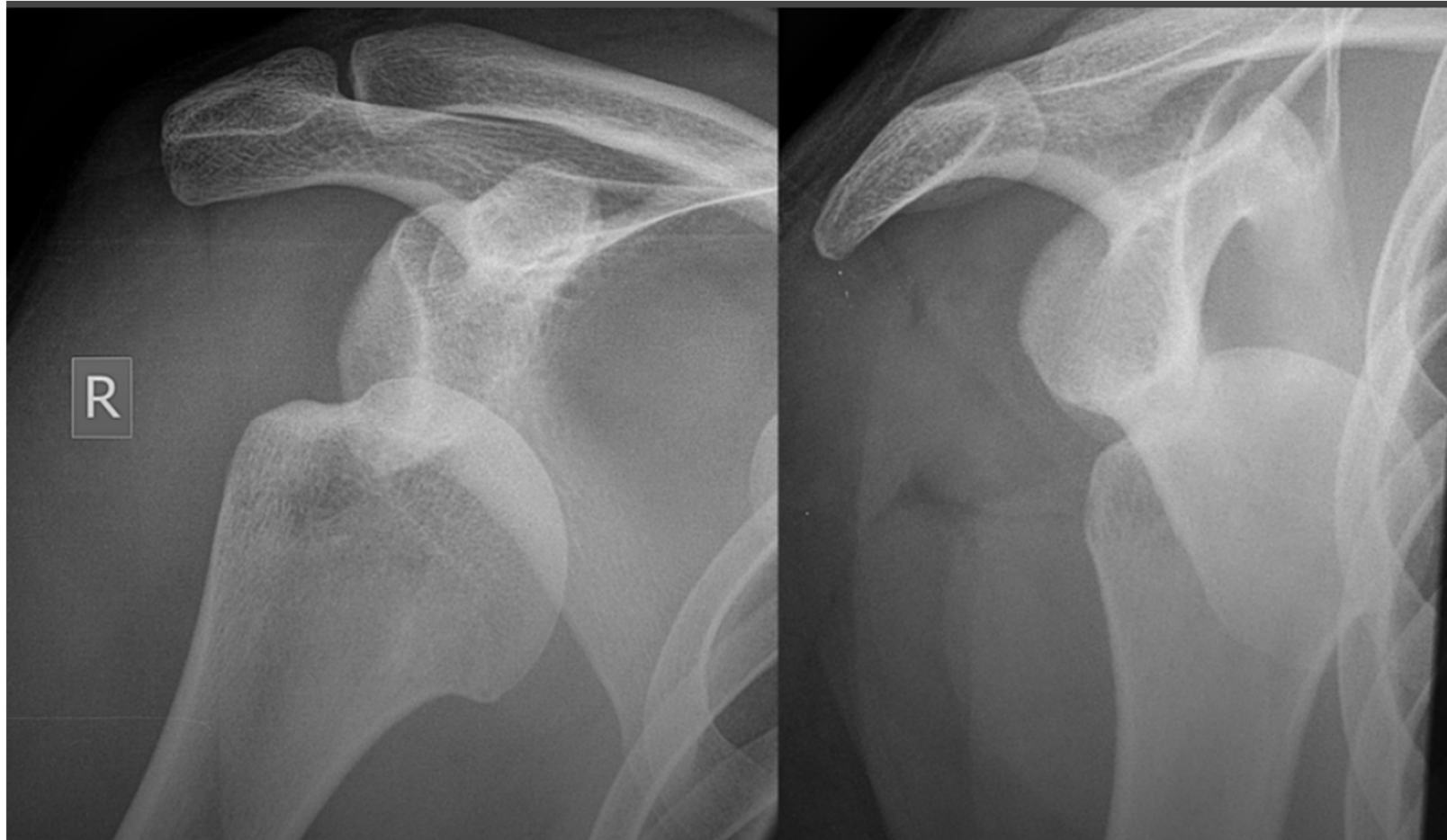


Case Example: Dislocated Shoulder



with Axillary Nerve Palsy – consider Radial to Axillary Nerve Transfer ?

Dislocated Shoulder



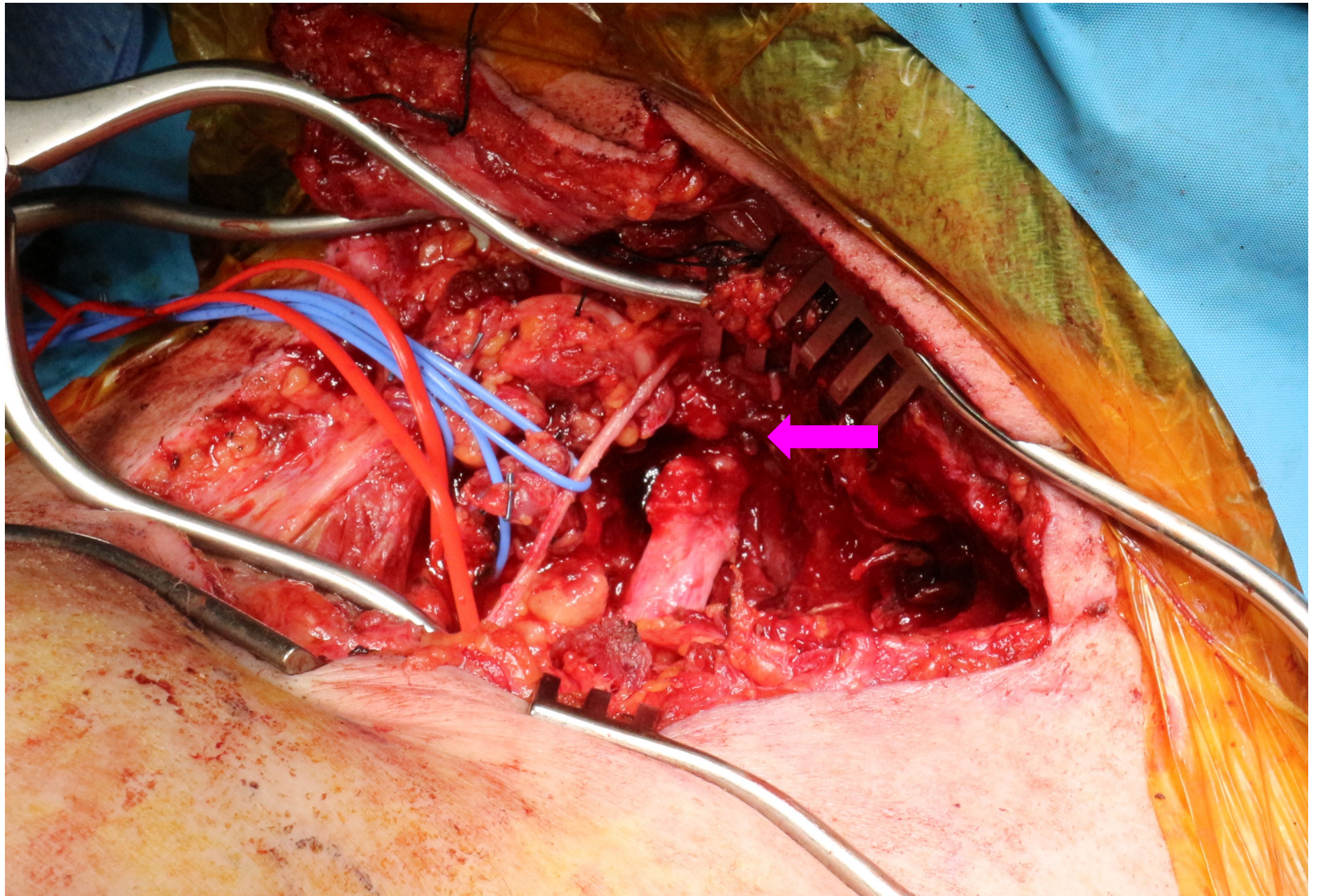
with Musculocutaneous Nerve Palsy – consider Oberlin Nerve Transfer ?

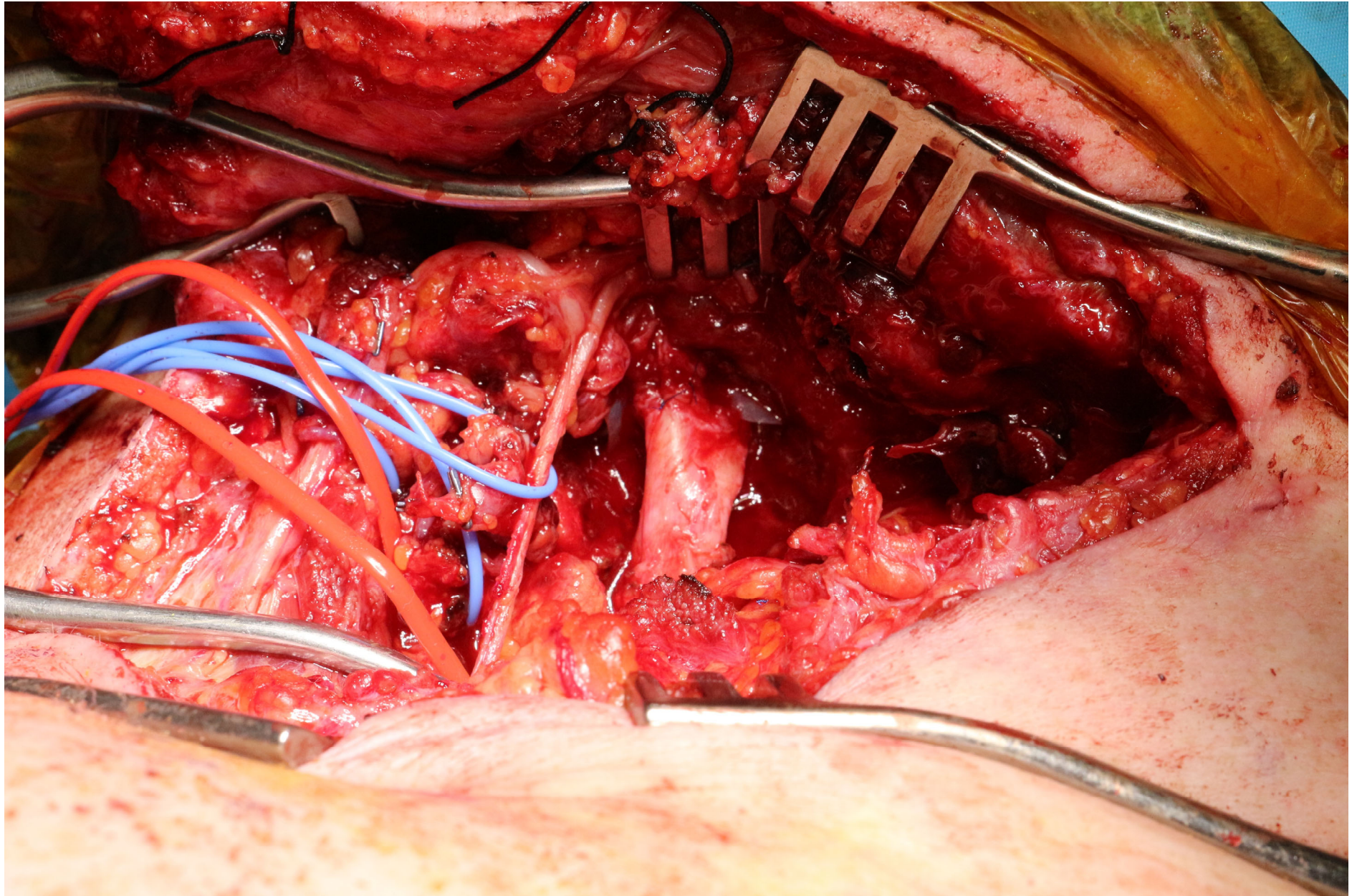
Case Example: Stab wound neck



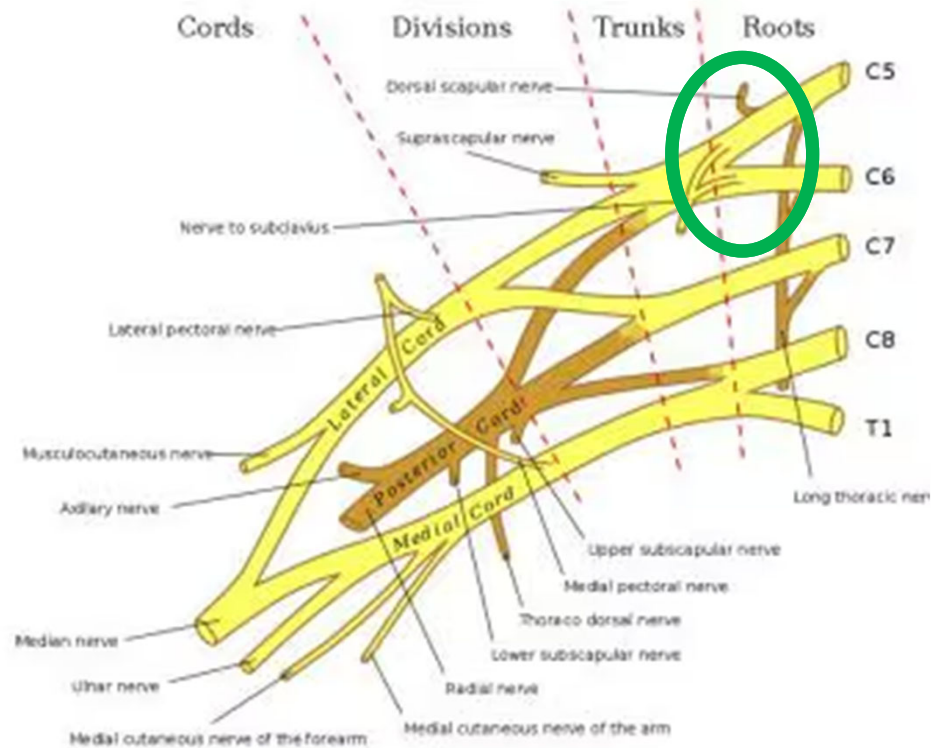
- No shoulder abduction or elbow flexion
- Exam Consistent with C5 and C6 root injuries



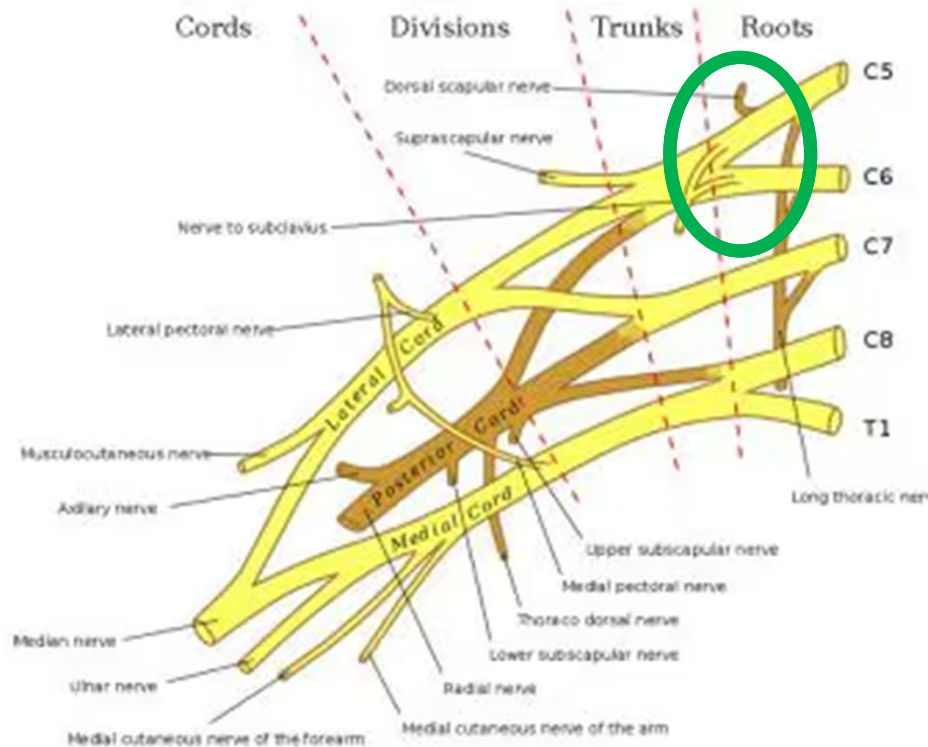




anything else to do ?



anything else to do ?

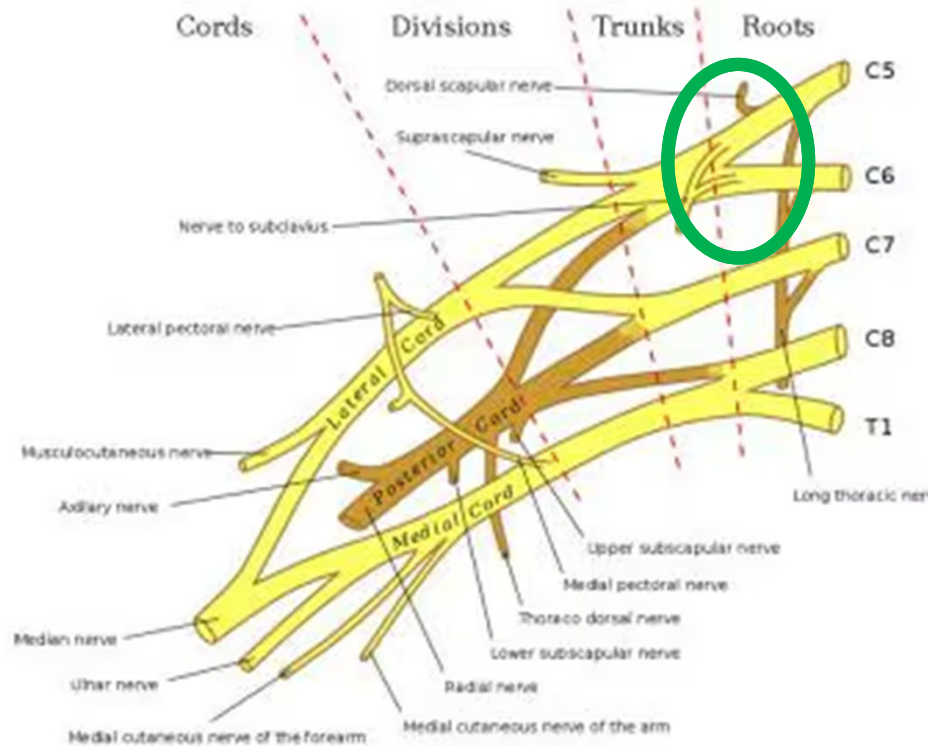


Ulnar to MSC (Oberlin) ?

Radial to Axillary (Leechavengvongs) ?

Spinal Accessory to Suprascapular ?

anything else to do ?



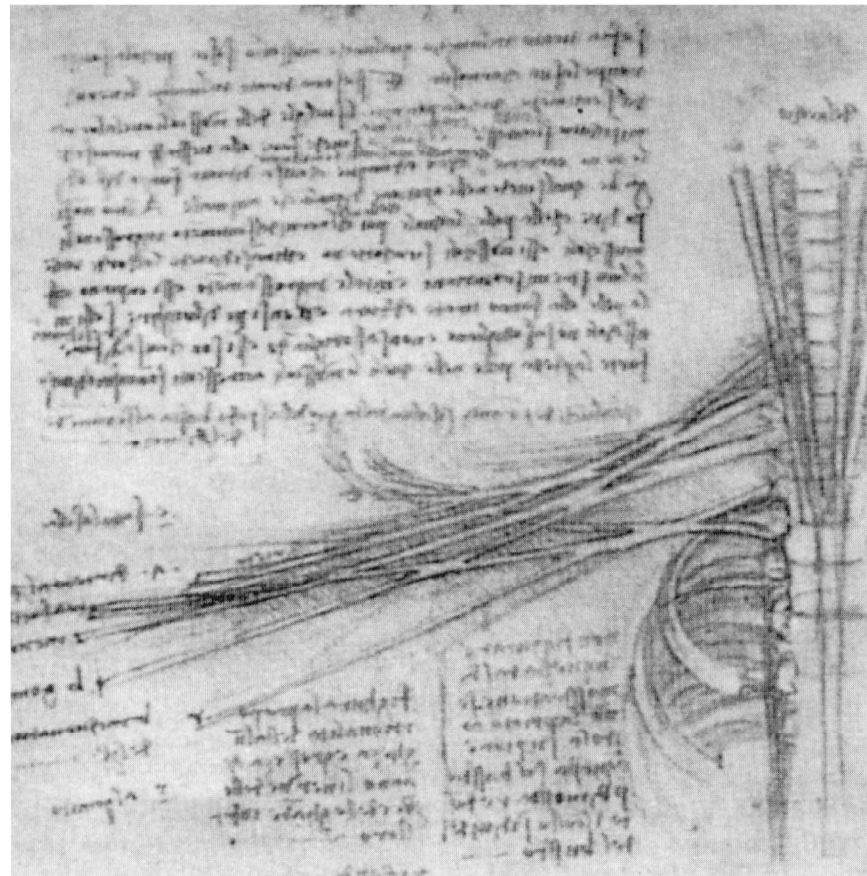
Ulnar to MSC (Oberlin) ?

Radial to Axillary (Leechavengvongs) ?

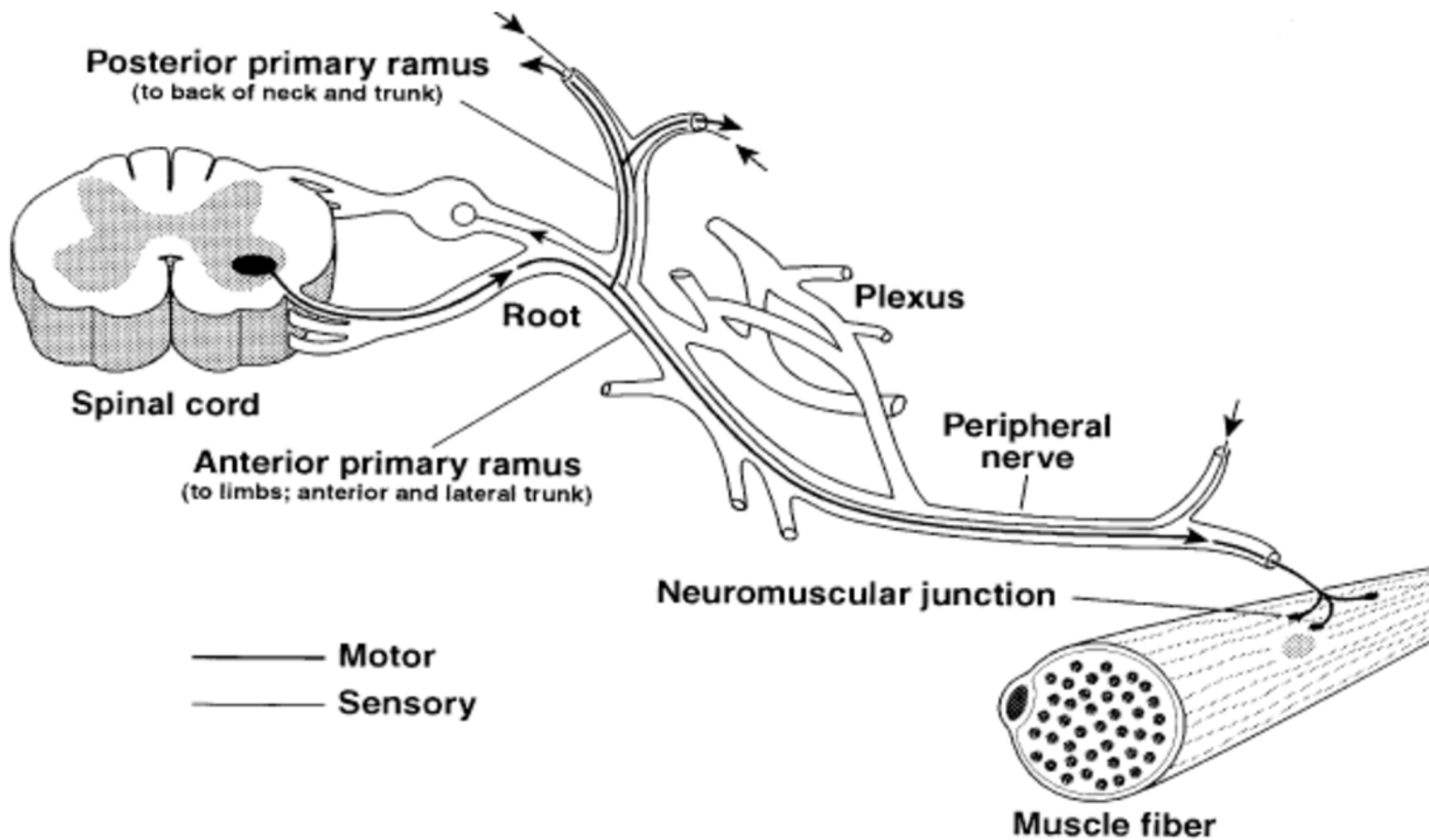
Spinal Accessory to Suprascapular ?

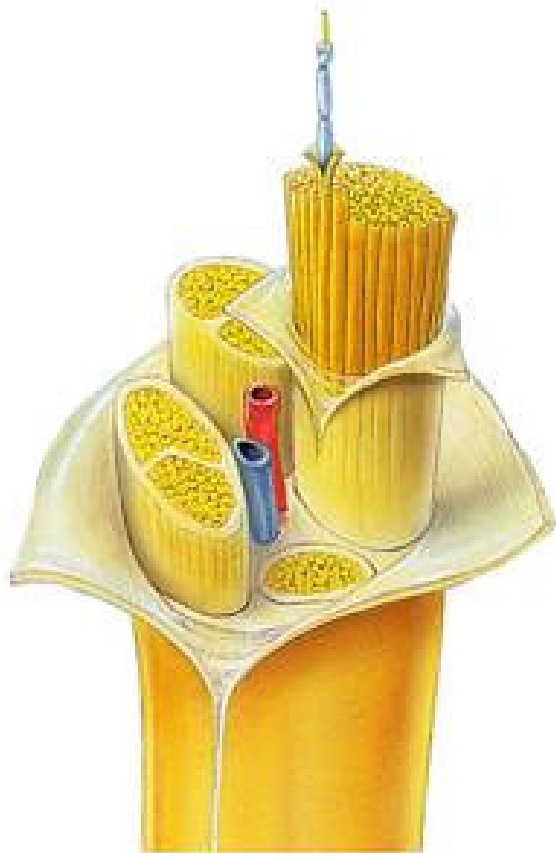
End to End ?

End to Side ?

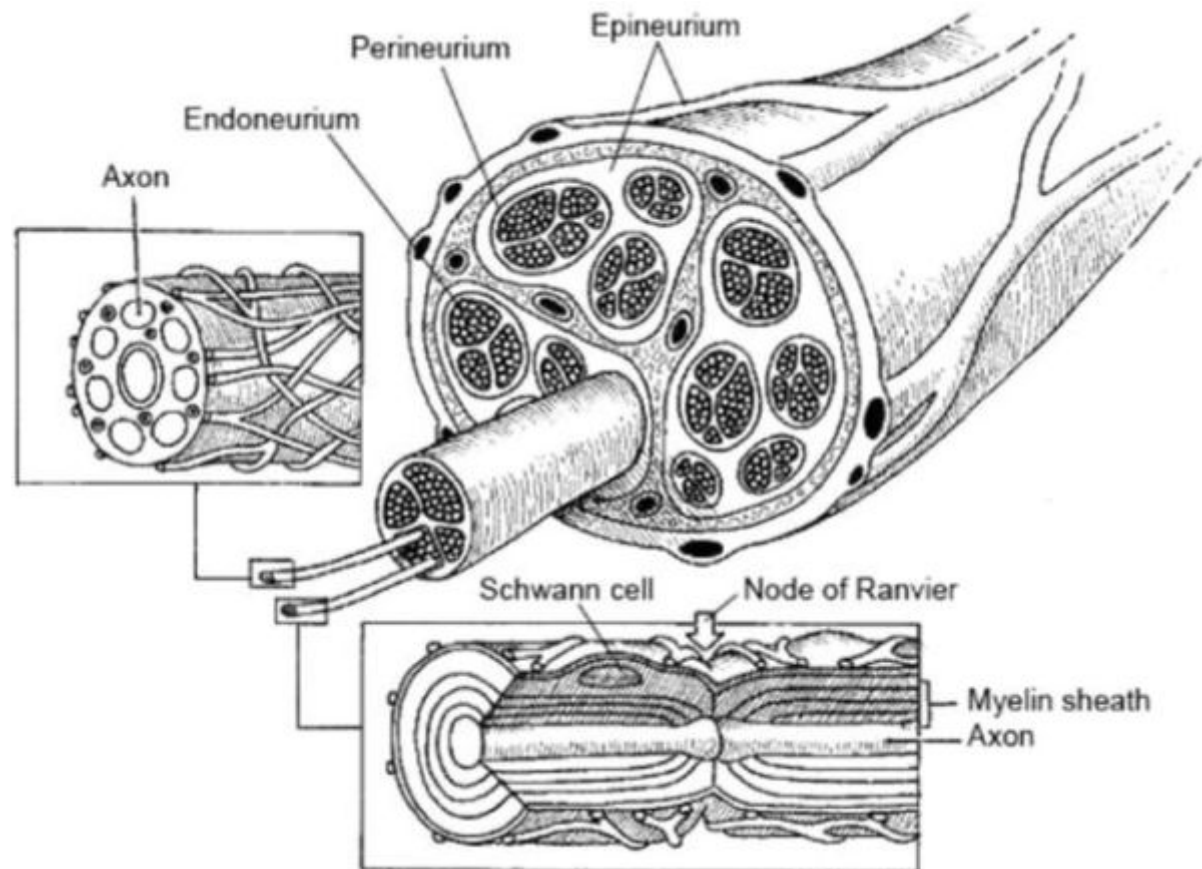


Leonardo da Vinci ~ 1500





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But Hector, in his shining helm, hit him with the barren stone at the shoulder, where the clavicle divides the neck from the chest, a most opportune point, and broke the nerve.

The arm fell numb to the wrist, he (Teucus) fell on his knees and lay still, the bow fallen from his hand.

Homer's Iliad
- 8th century BC

Classification

Tissue Damage

Recovery

Epineurium

Perineurium

Endoneurium

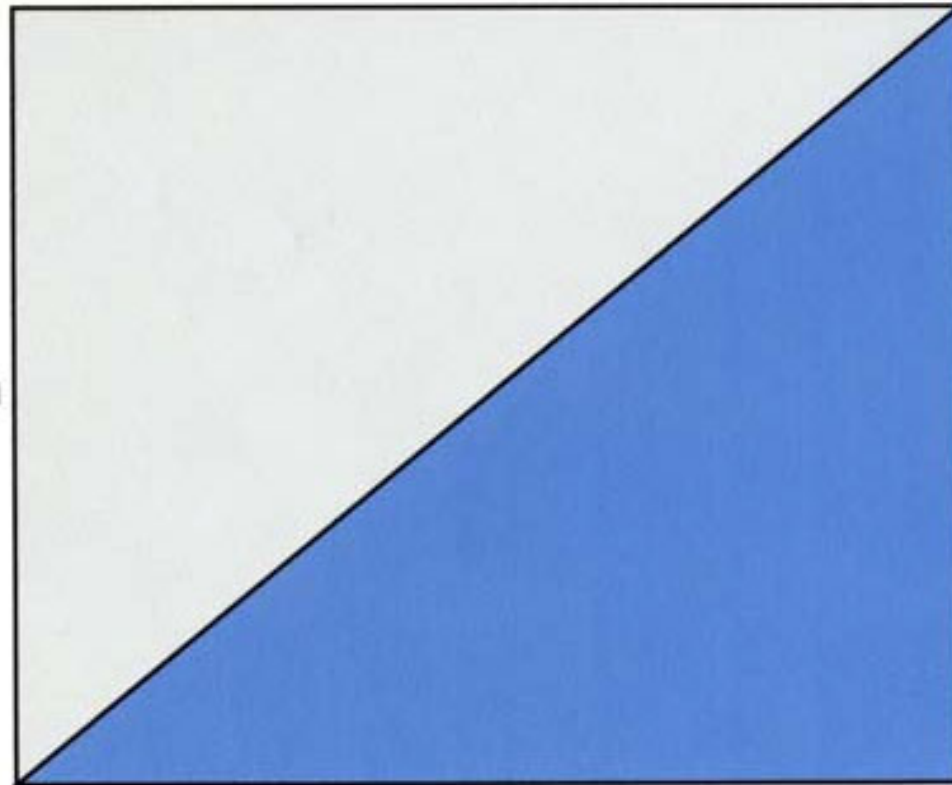
Axon

Myelin

None

Incomplete

Full



Seddon Grade

Neurapraxia

Axonotmesis

Neurotmesis

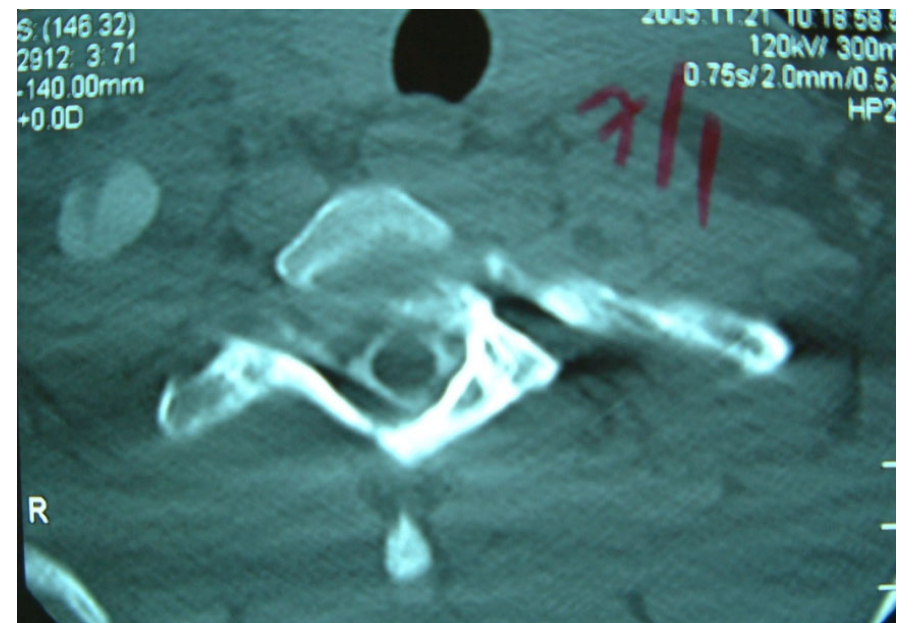
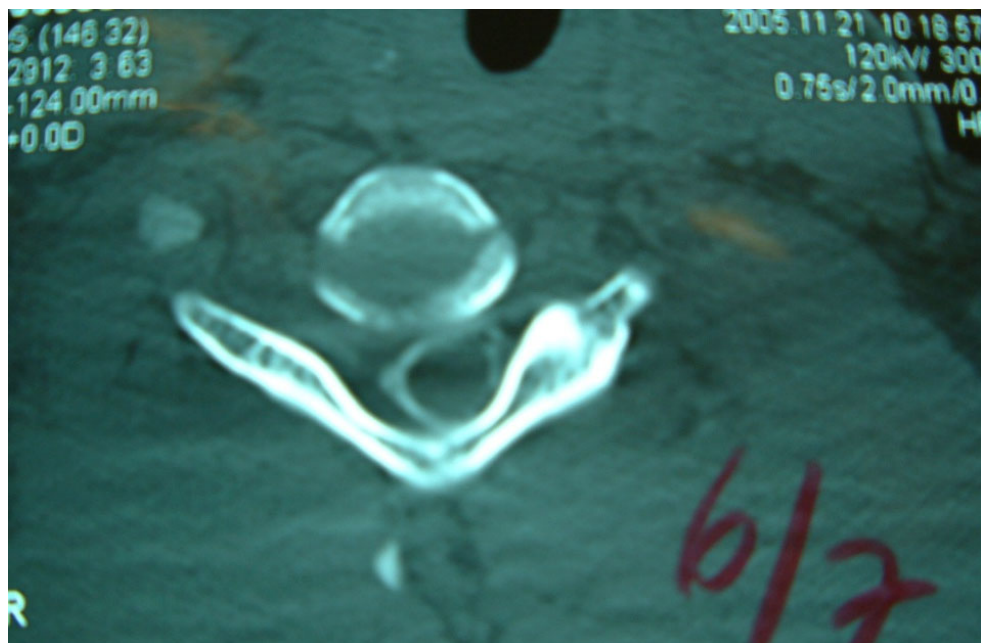
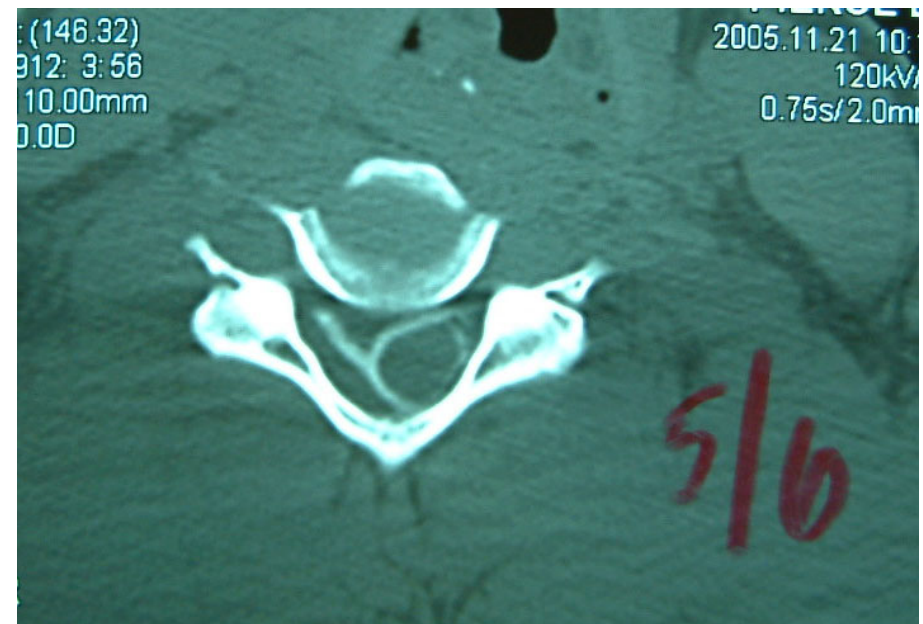
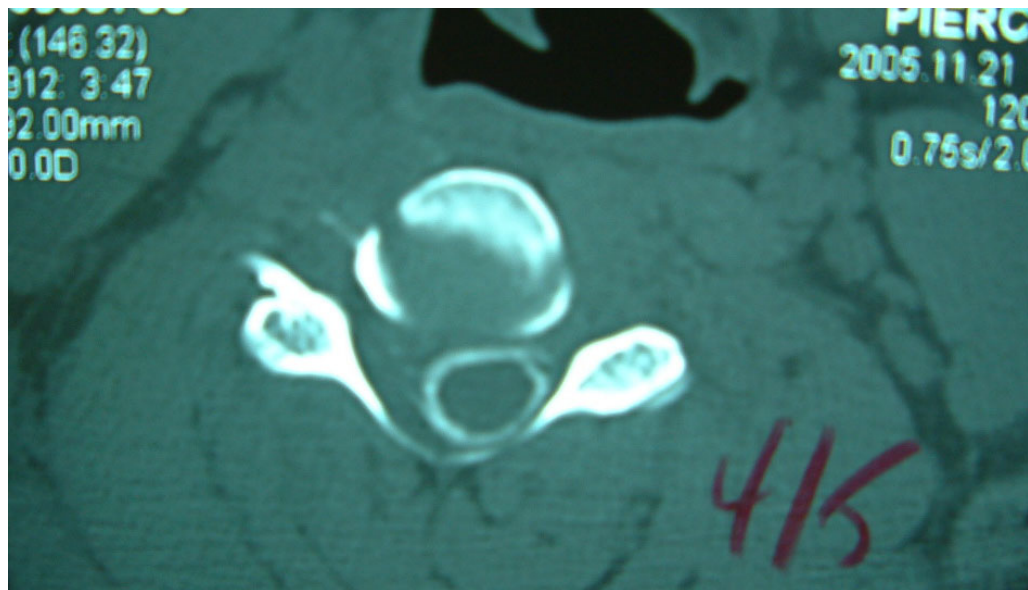


- Can remain a challenge
- Natural History vs Intervention
 - as interventions become more predictable, the intervention becomes more attractive
 - End to Side may have little morbidity and potential benefit

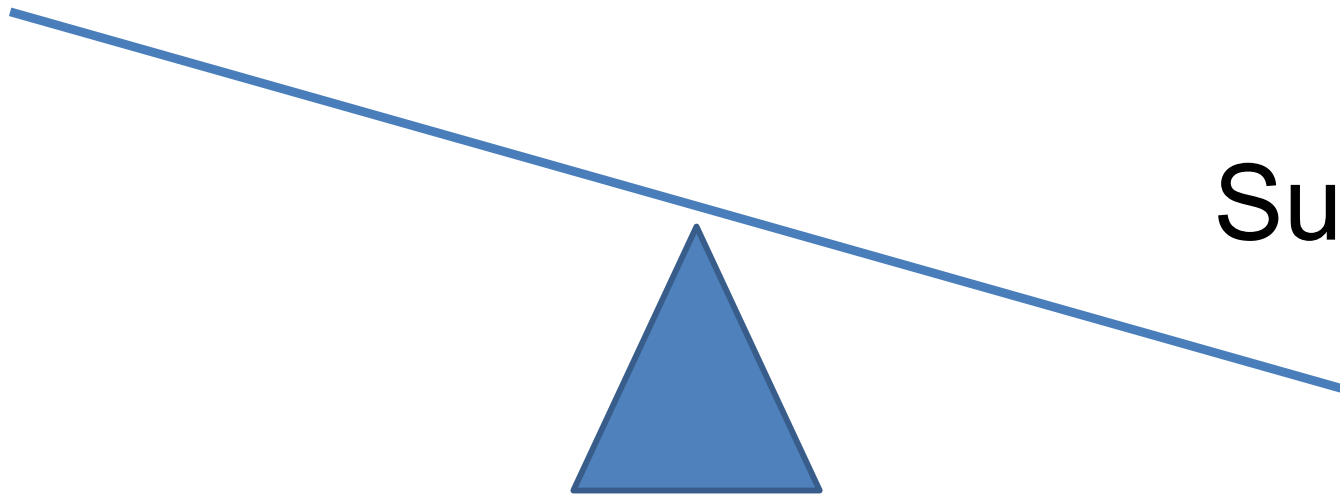
Most injuries closed, traction type



Studies: EMG, MRI, other ?



Natural History



Surgery

Targeted Muscle Reinnervation

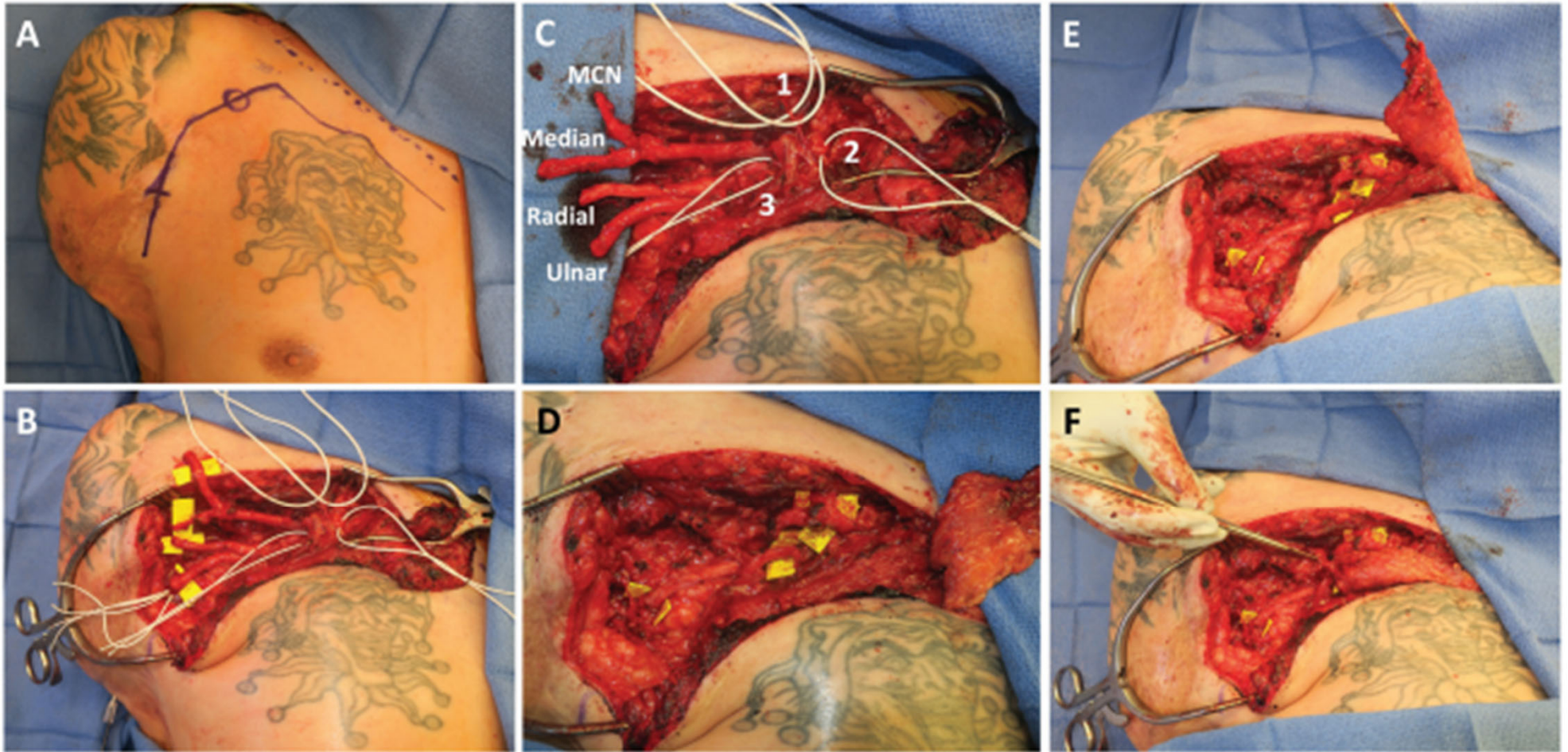
- TMR is a surgical technique **originally created to improve prosthetic function** following upper extremity amputations
- TMR has since been shown to be effective in the **prevention and treatment** of chronic postamputation phantom and **residual limb pain** for both UE and LE amputees

Targeted Muscle Reinnervation—an Up-to-Date Review: Evidence, Indications, and Technique

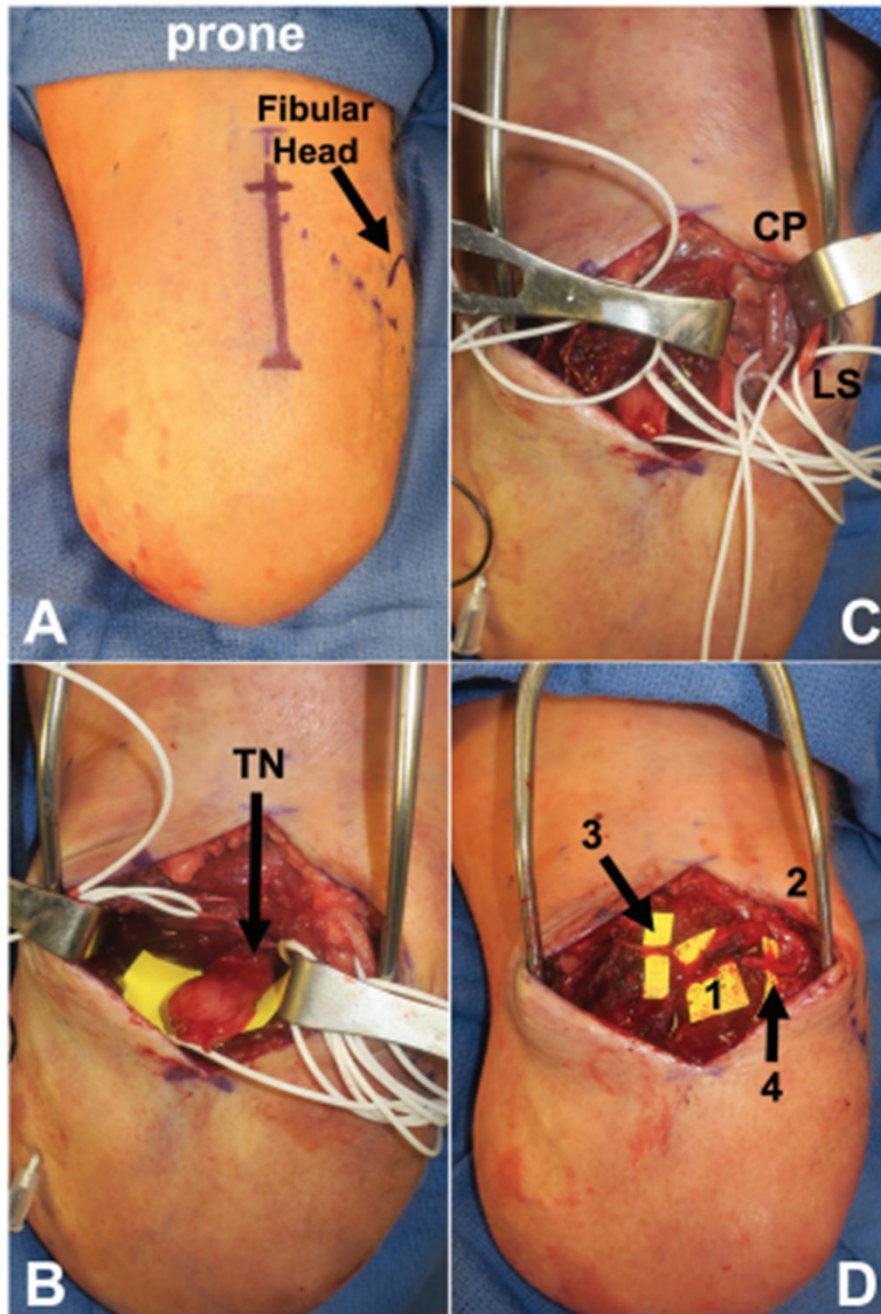
Ava G. Chappell, MD¹  Matthew D. Ramsey, MD¹  Seong Park, BA¹  Gregory A. Dumanian, MD¹ 
Jason H. Ko, MD, MBA¹ 

Arch Plast Surg 2025

TMR: Improving Prosthetic Function



TMR: Preventing and Treating Pain



TMR:

Tibial and Peroneal
Terminal Nerves
coapted into local
motor branches

RANDOMIZED CONTROLLED TRIAL

OPEN

Targeted Muscle Reinnervation Treats Neuroma and Phantom Pain in Major Limb Amputees

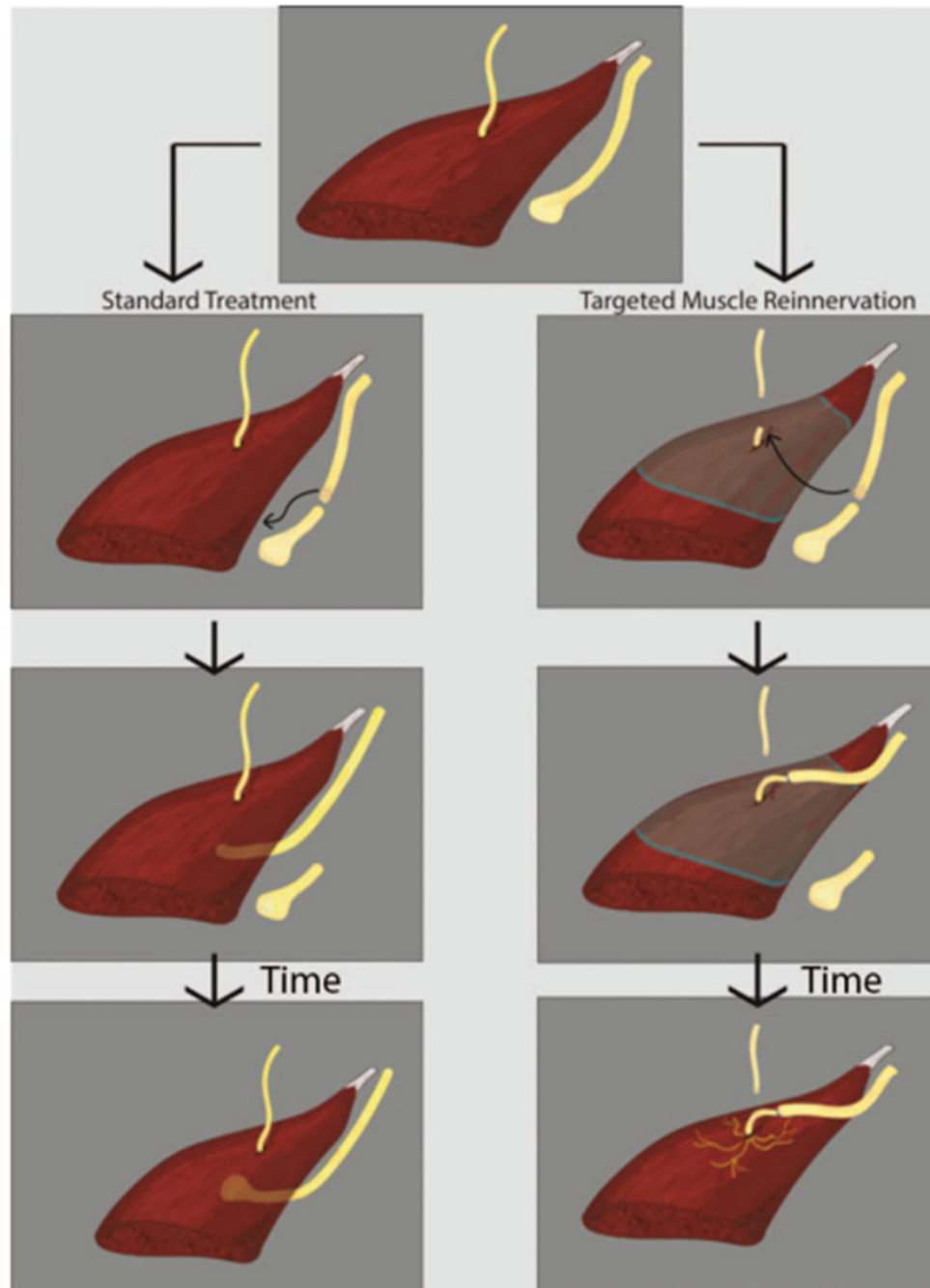
A Randomized Clinical Trial

Gregory A. Dumanian, MD,* Benjamin K. Potter, MD,† Lauren M. Mioton, MD,* Jason H. Ko, MD,*
Jennifer E. Cheesborough, MD,* Jason M. Souza, MD,† William J. Ertl, MD,‡ Scott M. Tintle, MD,†
George P. Nanos, MD,† Ian L. Valerio, MD,§ Todd A. Kuiken, MD, PhD,* A. Vania Apkarian, PhD,¶
Kyle Porter, MAS,|| and Sumanas W. Jordan, MD, PhD*§

Annals of Surgery, 2019

“...TMR improved phantom limb pain
and trended toward improved residual limb pain
compared with conventional neurectomy ...”

TMR and Phantom Limb Pain Improved



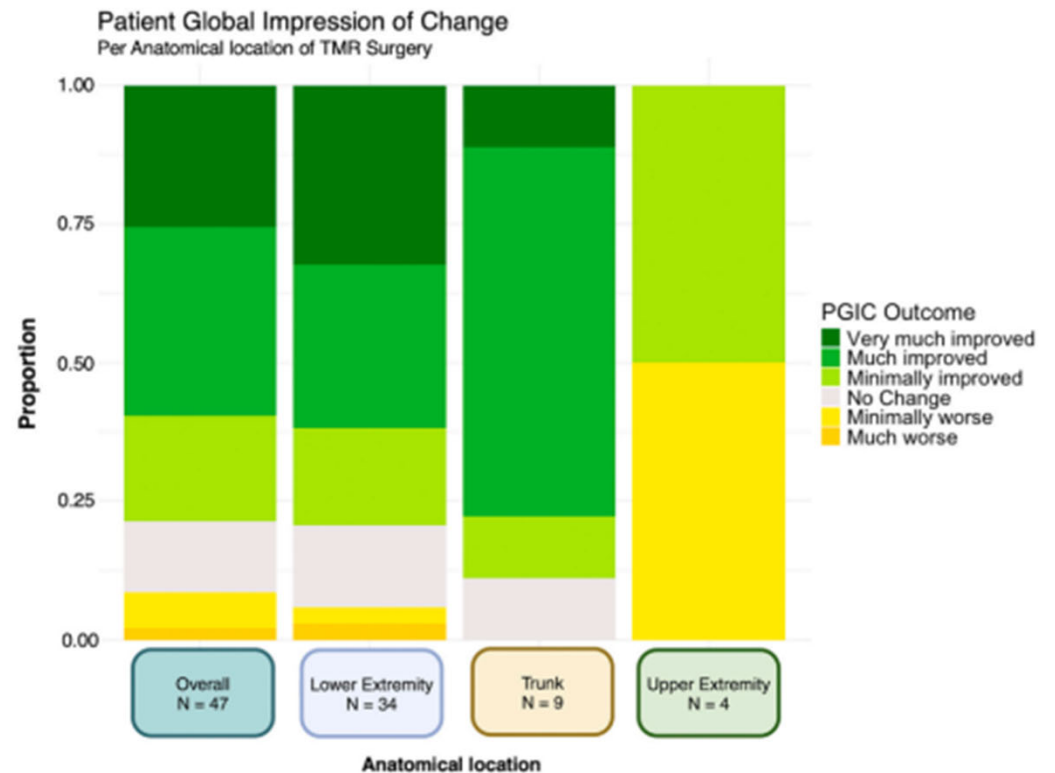
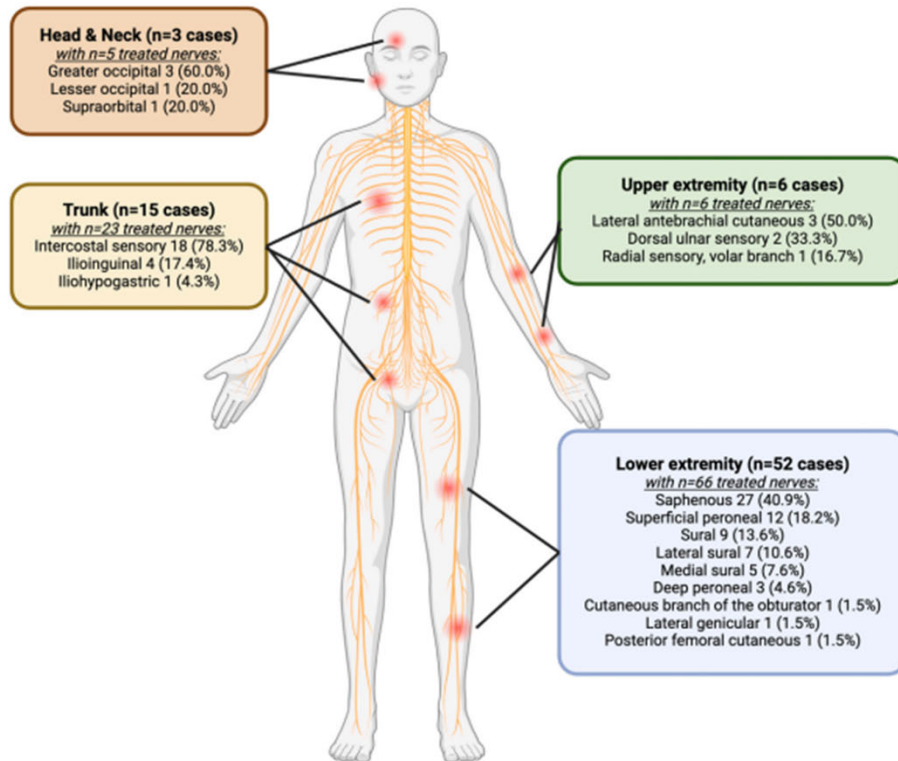
Targeted muscle reinnervation for treatment of neuropathic pain in non-amputees - Indications and outcomes

Floris V. Raasveld ^{a,b,c,1}, Otis C. van Varsseveld ^{b,d,1},
Caroline A. Hundepool ^c, J. Michiel Zuidam ^c,
Jonathan M. Winograd ^a, William G. Austen Jr. ^a,
Ian L. Valerio ^a, Kyle R. Eberlin ^{a,*}

J of Plastic, Recon & Aesthetic Surgery, 2025

Non-Amputees report improvement in neuropathic pain across various anatomic regions, suggesting the benefit of TMR beyond the amputee population

TMR Improves Pain for Non-Amputees



News Phillies

Zack Wheeler to undergo thoracic outlet decompression surgery, out for remainder of season

Benjamin Goldstein August 23, 2025 0



Venous Thoracic Outlet Syndrome

Aaron Cook (rockies)
Alex Cobb (giants)
Merrill Kelly (diamondbacks)
Clayton Richard (padres)
Chris Carpenter (cardinals)
Mike Foltynewicz (braves)
Stephen Strasburg (nationals)

News Phillies

Zack Wheeler to undergo thoracic outlet decompression surgery, out for remainder of season

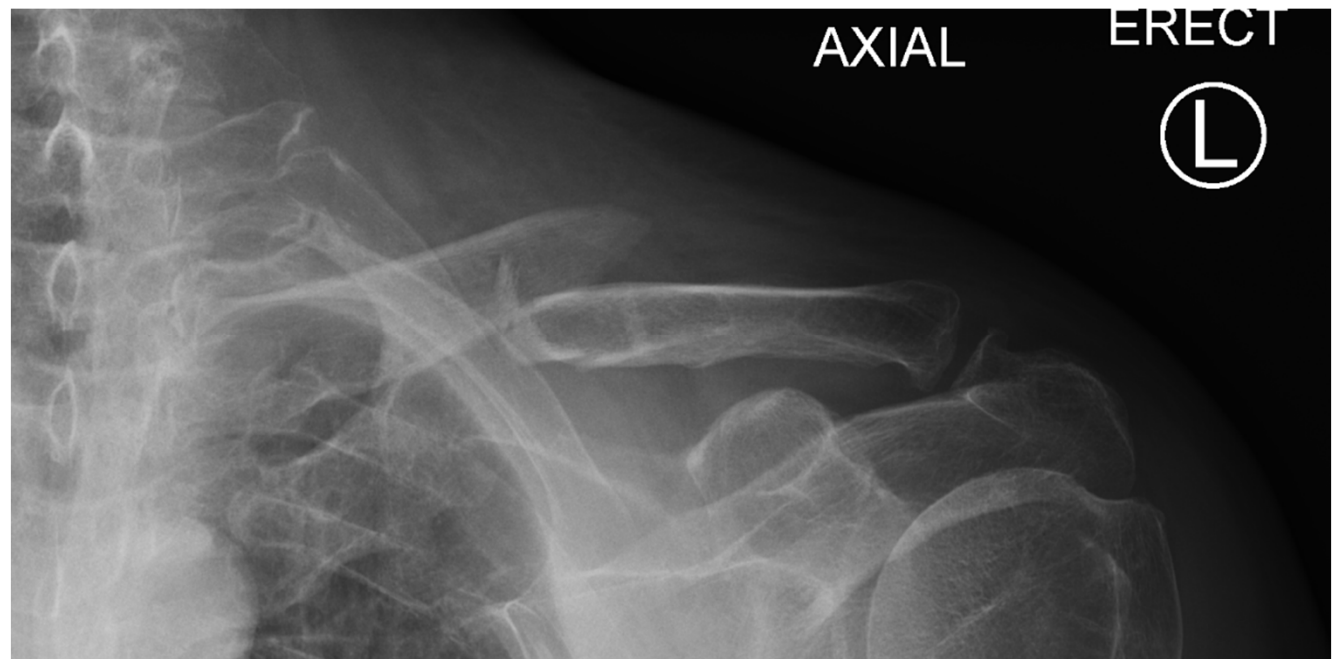
© Benjamin Goldstein August 23, 2025



- Remember History, Physical Exam and Differential Diagnosis
- Consider Structural Lesions amenable to Surgical Treatment

Case Example: TOS

- 72 yo male, slip and fall on ice at home
- 2/2019 clavicle and rib fractures
- History of ankylosing spondylitis



- 6/2019, 4 months post injury, exam worsening
- Arm becoming “**paralyzed**”, initially shoulder weakness but worsening hand

Left Hand/Wrist Exam

Muscle Strength

Wrist Extension: 4/5

Wrist Flexion: 4/5

Grip:

Comments: Weak grip strength

Finger strength 4/5

Left Shoulder Exam

Comments: Limited elbow motion with shoulder ER

Elbow flexion 5/5

Triceps strength 3/5

Shoulder abduction - 0/5

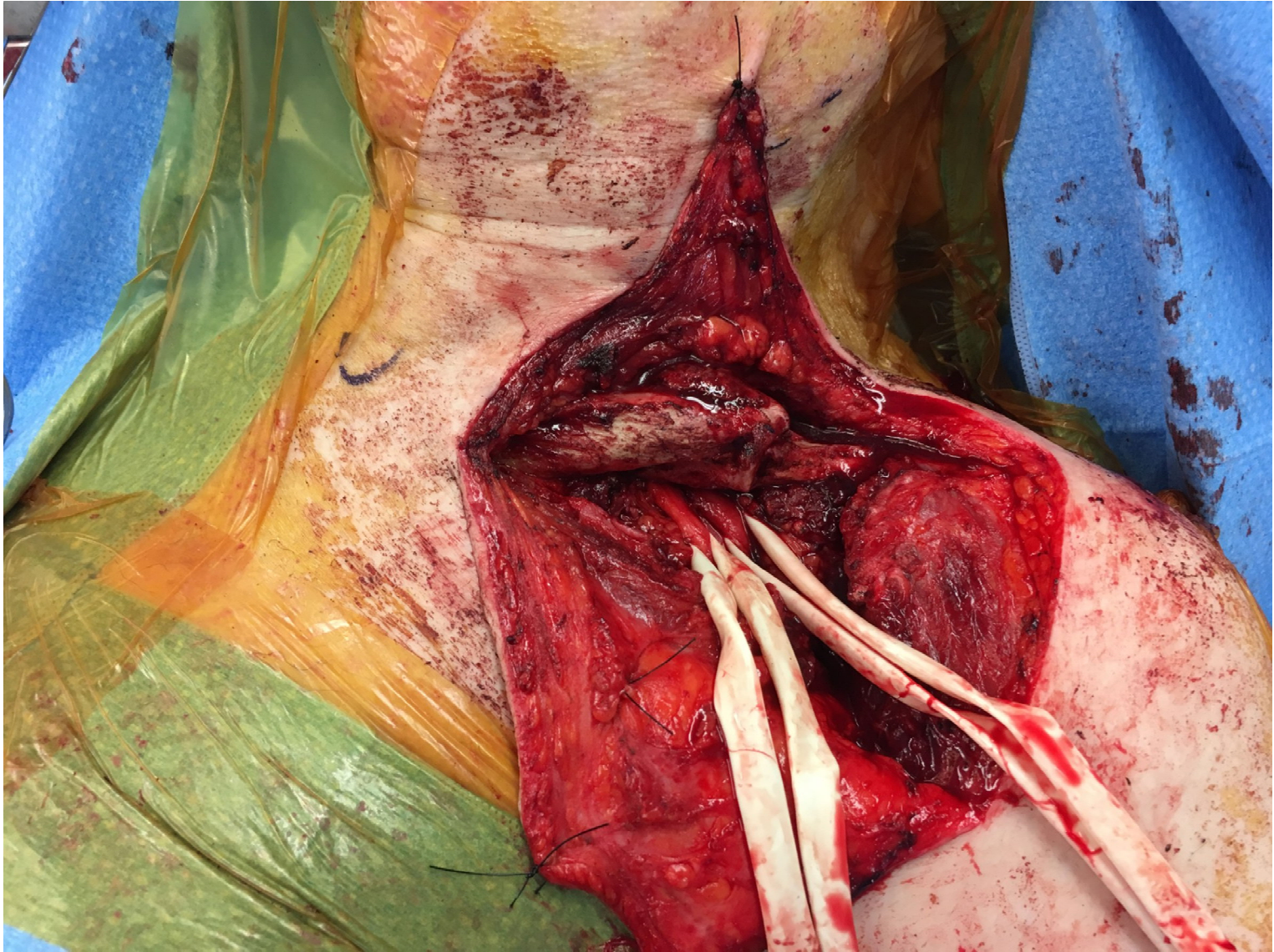
Shoulder er - 0/5

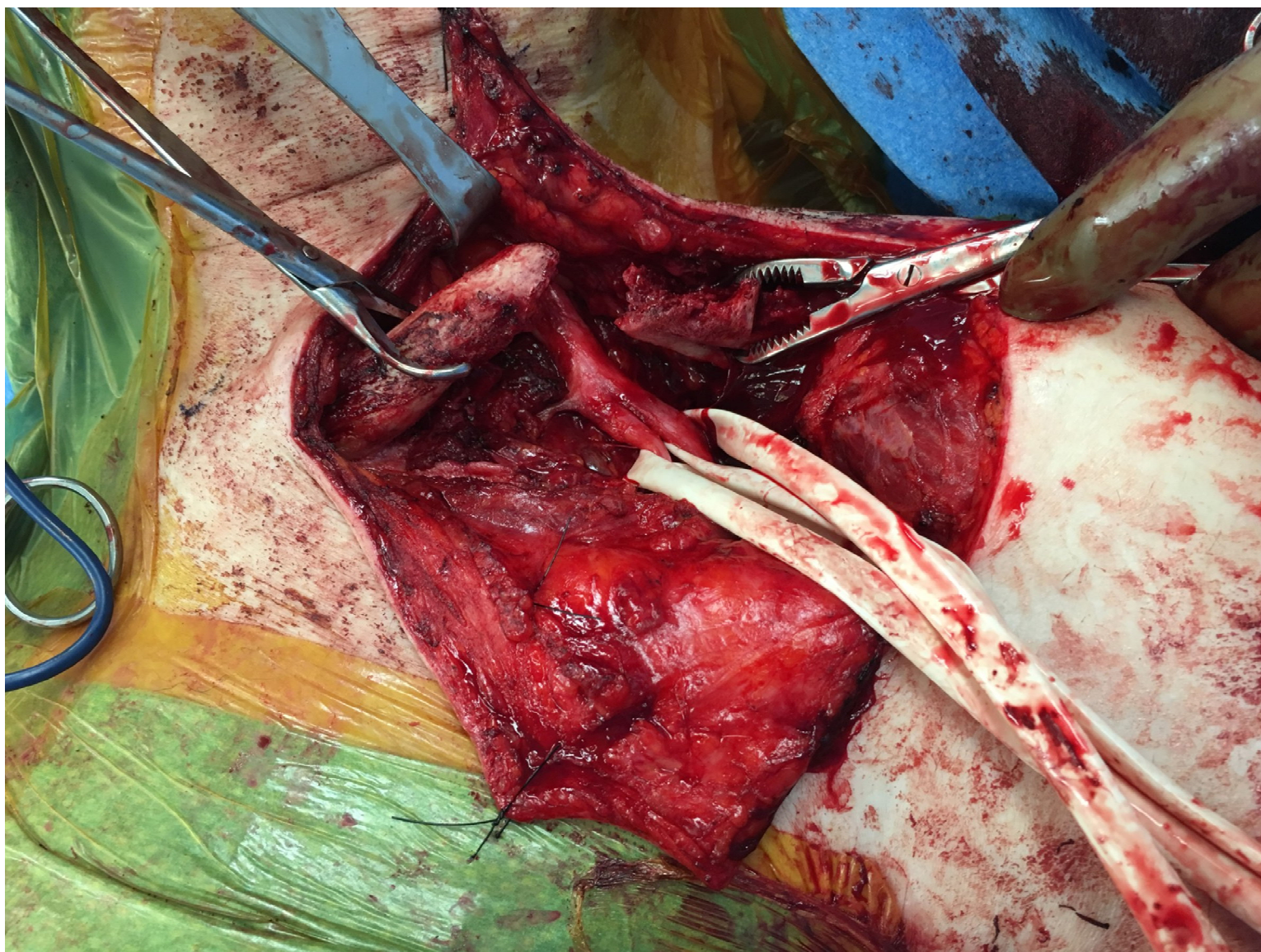
Wrist f/ex/ext - 4/5

- 6/2019 clavicle malunion



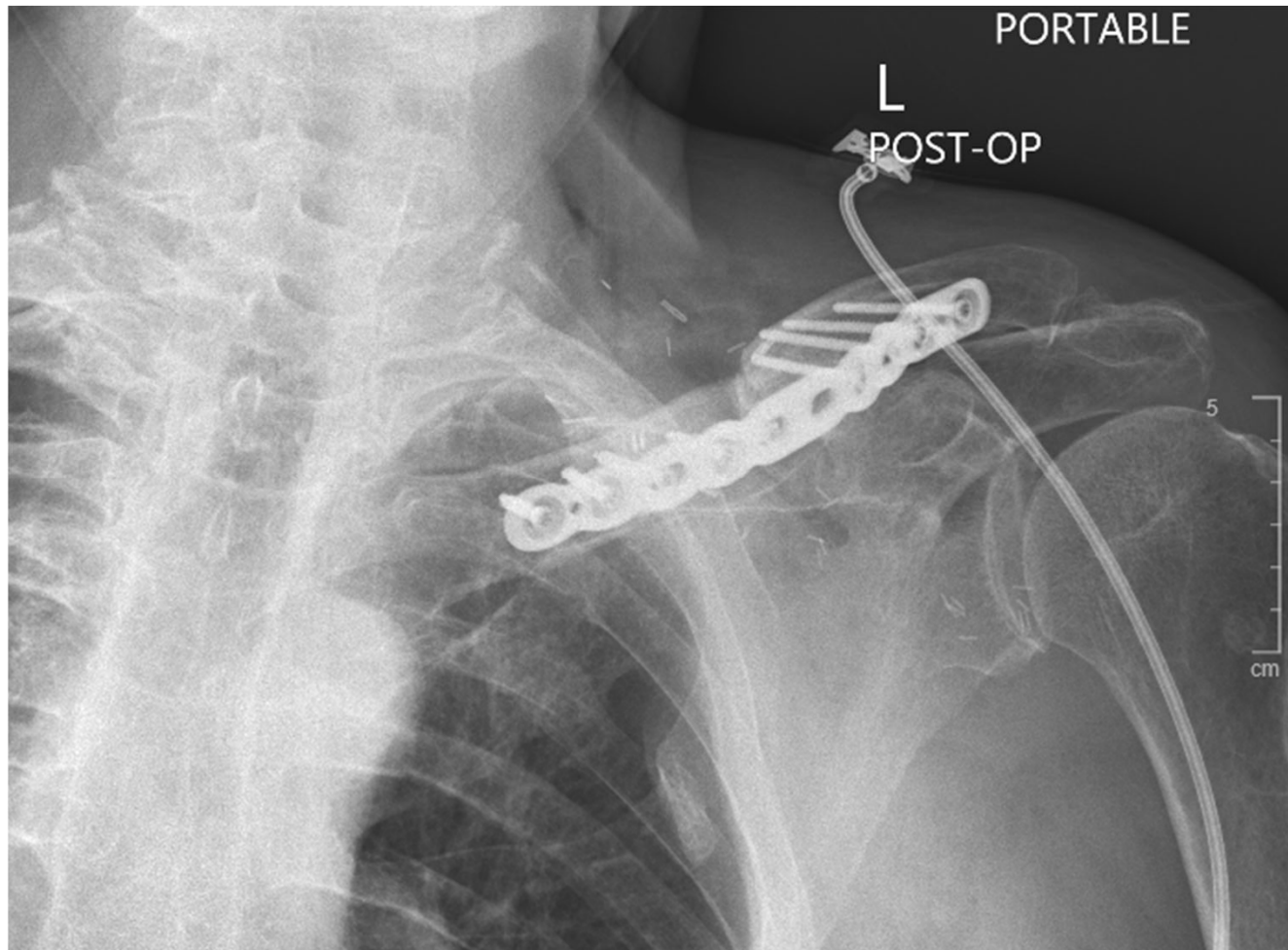
Severe shortening left clavicle



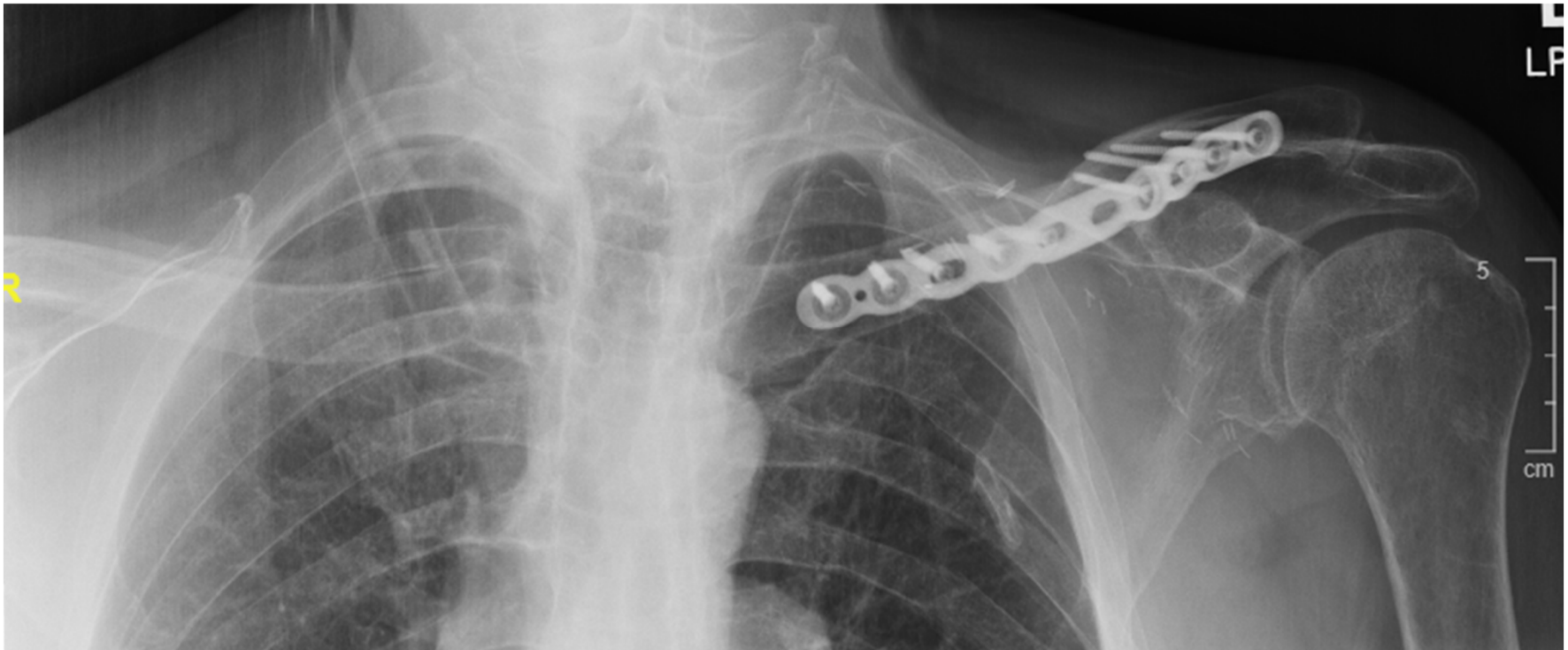




- 6/2019 Post Op Osteotomy

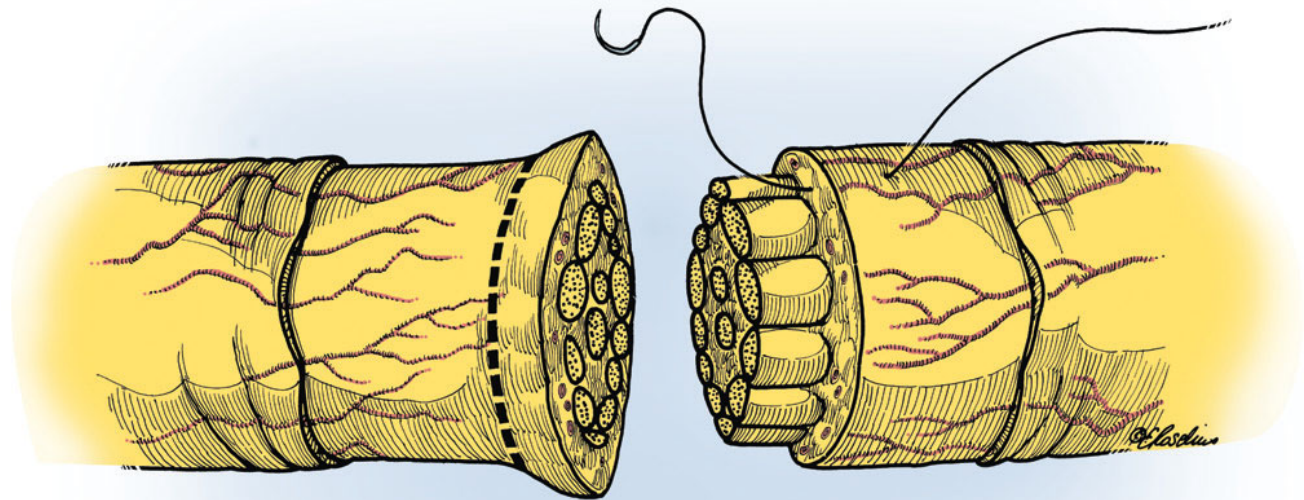


- 1/2020 healed osteotomy



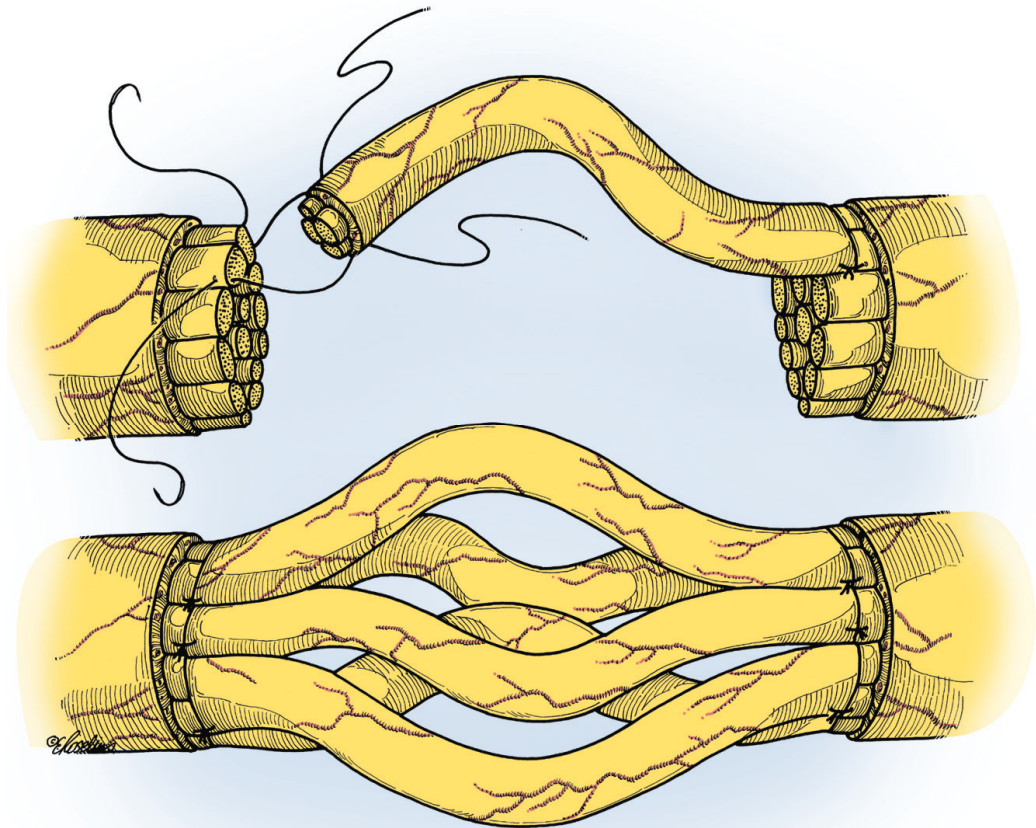
PE: full motion left shoulder, arm with dramatic strength improvement.
Only some mild residual weakness left hand. Full sensory recovery

- Nerve Coupling Techniques
- Nerve Grafts
- Nerve Conduits



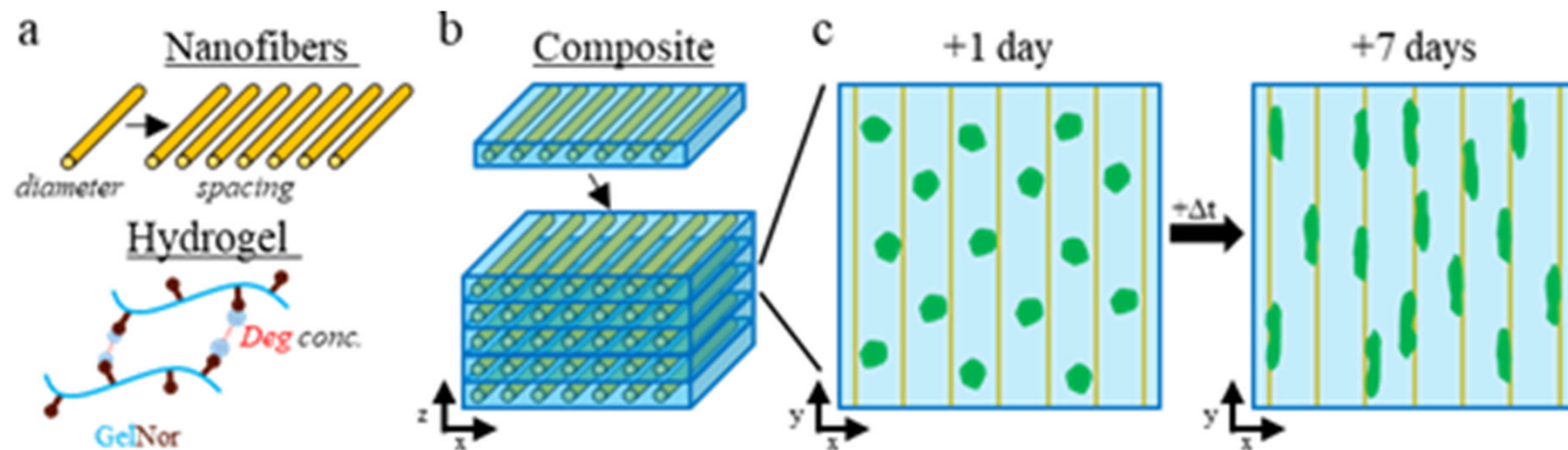
Nerve Repair

- Nerve Coupling Techniques
- Nerve Grafts
- Nerve Conduits



Synthetic Nerve Grafts

- Bioengineered Nerve Grafts



- Discuss Active Areas of Nerve Surgery with emphasis on Nerve Transfers
- Understand that Nerve Surgeries are expanding and may have an increasing role in practices
- Stimulate excitement about future growth areas

Thank you



Cooper Medical School
of Rowan University

Question 1:

Which of the following is true about a Nerve Transfer:

- 1) There are unlimited donor nerves
- 2) It is a surgical procedure that transfers a nonfunctioning donor nerve into a functioning recipient nerve
- 3) It is an experimental procedure
- 4) Despite improvements, the success rate of the procedure is less than 50%
- 5) Indications for the procedure are expanding beyond patients with brachial plexus injuries

Question 2:

For a successful nerve transfer, which of the following statements is accurate:

- 1) It needs to be performed in an End to Side Fashion
- 2) It needs to be performed in an End to End Fashion
- 3) It should be performed as far away from the target muscle as possible
- 4) It can be performed with coaptation of only a portion of a donor nerve
- 5) The donor and recipient functions should be antagonistic

Question 3:

A 26 year old female has sustained a C5-C6 root injury and now lacks elbow flexion 6 months after the injury. Elbow flexion can be restored thru nerve transfer. Successful restoration of elbow flexion has been performed by transfer of:

- 1) A portion of the ulnar nerve to the Musculocutaneous Nerve
- 2) The axillary nerve to the Musculocutaneous Nerve
- 3) A portion of the radial nerve to the Musculocutaneous Nerve
- 4) A portion of the radial nerve to the Axillary Nerve
- 5) Transfer of the AIN to the Ulnar nerve

Question 4:

For a 36 year male who a sustained radial nerve 2 years previously, what is true about reconstructive options for his radial nerve injury:

- 1) Tendon transfers are no longer indicated
- 2) Nerve reconstruction of the primary radial nerve injury should always be performed as a first step
- 3) Joint stiffness is not an issue for successful outcome after nerve transfer
- 4) Tendon transfers can effectively restore function
- 5) Nerve transfers can predictably improve his function

Question 5:

A 28 year male sustained a transtibial amputation after a motorcycle accident and is suffering from Neuroma pain in his terminal limb. Which if the following is true regarding targeted muscle reinnervation (TMR) as a treatment to reduce the pain for him:

- 1) TMR is only effective if done acutely
- 2) TMR is only effective in the upper extremity
- 3) TMR is effective for both upper and lower extremity amputees with neuroma pain
- 4) TMR is inferior to conventional neurectomy
- 5) TMR is more effective for an upper extremity amputation than a lower extremity amputation to reduce neuroma pain

Peripheral Nerve Repair: Can we Block Wallerian Degeneration with PEG

David A. Fuller, MD
Professor of Orthopaedic Surgery

Rocky Mountain University
2026 Annual Meeting
Provo, Utah
March 28, 2026

- 2 research grants
 - 1) American Foundation for Surgery of the Hand
 - 2) Camden Health Initiative
- None financial

Objectives

1. PEG history and development
2. PEG research updates
3. Update on PEG approval for human use
4. How this might impact practicing hand surgeons

Core Values

Learning-Centered

By challenging thinking, encouraging growth, and exploring possibilities within a supportive learning environment, we empower learners to rise to their highest potential.



**Rocky Mountain
University**
of Health Professions

Nerve Repair 1970s



Overcoming Wallerian Degeneration

Once a nerve is functionally cut, the distal portion undergoes a process of degeneration that we have accepted for centuries as inevitable

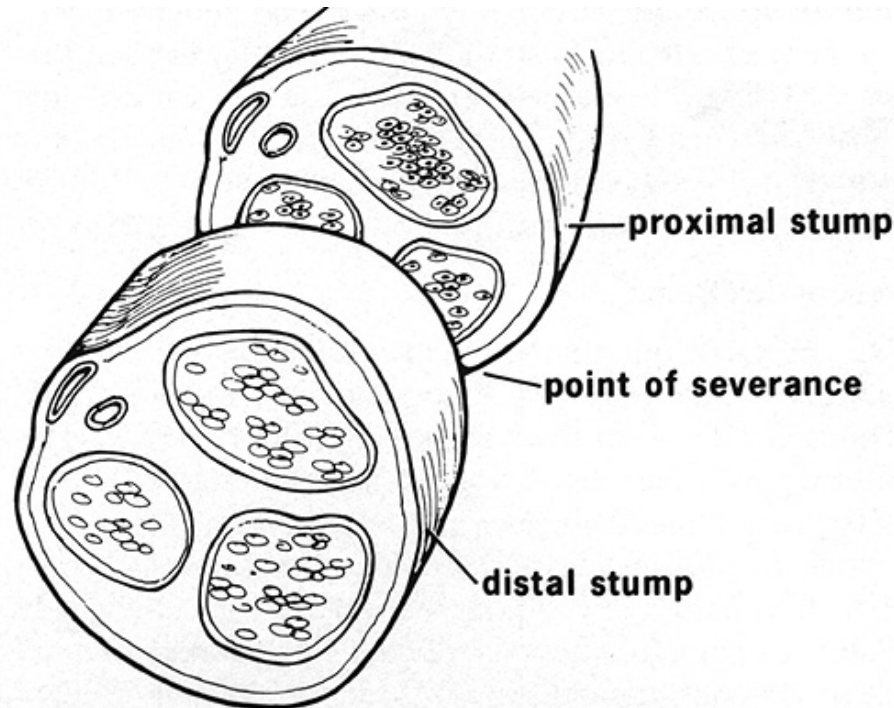


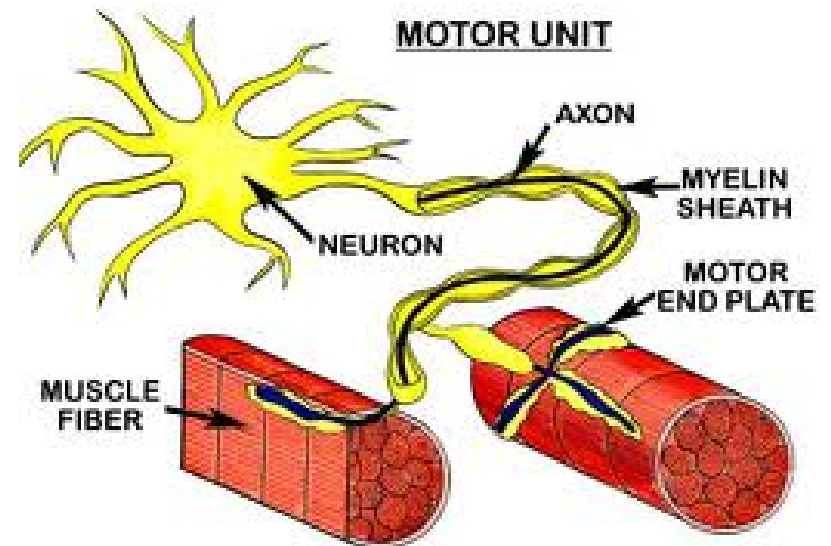
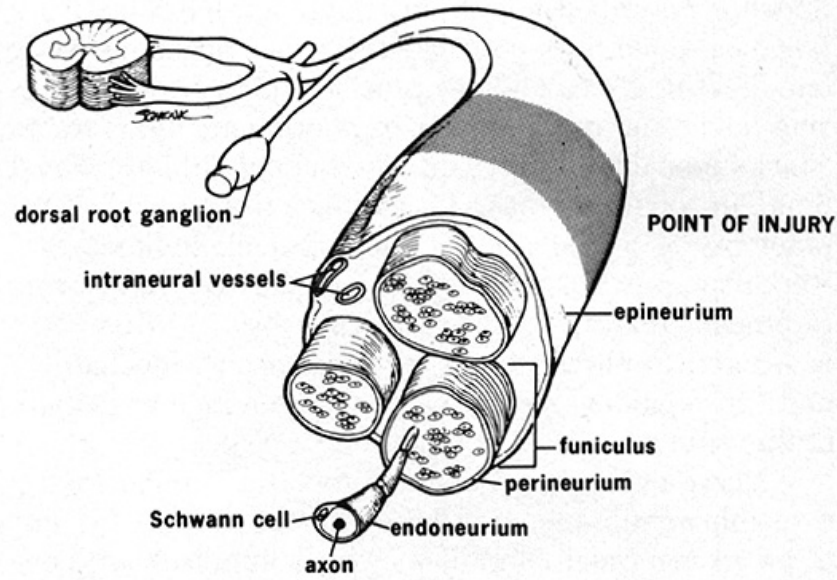
FIG. 65-7 Transection of entire nerve trunk results in neurotmesis. (Raffe MR: Peripheral nerve injuries in the dog, Part II. Compendium on Continuing Education for the Small Animal Practitioner 1:269–276, 1979)

Augustus Waller, 1816-1870



British Physician and Neurophysiologist
described degeneration of severed nerve fibers (1851)

Downstream Activity after Neurotmesis:



Nerve and Muscle

Microarray and qPCR Analyses of Wallerian Degeneration in Rat Sciatic Nerves

Sheng Yi[†], Xin Tang[†], Jun Yu, Jie Liu, Fei Ding^{*} and Xiaosong Gu^{*}

Key Laboratory of Neuroregeneration of Jiangsu and Ministry of Education, Co-Innovation Center of Neuroregeneration, Nantong University, Nantong, China

Wallerian degeneration occurs immediately following injury to mammal peripheral nerves. To better understand the molecular events occurring during Wallerian degeneration, a rat model of sciatic nerve transection was used to assess differentially expressed genes at 0.5, 1, 6, 12, 24 h, 4 days, 1, 2, 3, and 4 weeks post nerve injury (PNI). Hierarchical clustering, Euclidean distance matrix, and principal component analysis (PCA) collectively suggested three distinct phases within the post-injury period of 4 weeks. Gene ontology (GO) analysis suggested that phase I (0–6 h PNI), phase II (6–24 h PNI), and phase III (4 days to 4 weeks) were associated with acute response to injury, preformation of Wallerian degeneration, and complete execution of Wallerian degeneration, respectively. Critical signaling pathways and transcriptional factor networks responsible for the regulation of Wallerian degeneration were further identified and integrated using Kyoto Enrichment of Genes and Genomes (KEGG) pathway analysis and Ingenuity Pathway Analysis (IPA), respectively. Our results may help to elucidate some molecular mechanisms of gene regulation associated with Wallerian degeneration that occurs after traumatic injury to peripheral nerve axons in mammals.

Keywords: Wallerian degeneration, peripheral nerve injury, microarray, gene expression, bioinformatic analysis

The Whole Transcriptome Involved in Denervated Muscle Atrophy Following Peripheral Nerve Injury

Jian Weng, Peixun Zhang, Xiaofeng Yin* and Baoguo Jiang*

Department of Orthopedics and Trauma, Peking University People's Hospital, Beijing, China

Peripheral nerve injury (PNI) usually leads to progressive muscle atrophy and poor functional recovery. Previous studies have demonstrated that non-coding ribonucleic acid (ncRNA) is a key regulator of muscle atrophy and beneficial for the treatment of PNI. We aimed to analyze the whole transcriptome involved in denervated muscle atrophy after PNI. Animal models of sciatic nerve injury were assessed at 0 (control group), 1, 2, 4, and 8 weeks after injury. The expression patterns in the whole transcriptome in the gastrocnemius muscle were profiled using RNA sequencing at each time point and compared to that obtained in the control group. Six-hundred and sixty-four long non-coding RNAs, 671 microRNAs, 236 circular RNAs, and 12,768 messenger RNAs (mRNAs) were differentially expressed (DE) after injury. Changes in some of the DE ncRNAs and mRNAs were validated using quantitative polymerase chain reaction. Gene Ontology and Kyoto Encyclopedia of Genes and Genomes analysis revealed the potential functions of and relationships among the DE ncRNAs and mRNAs. To our knowledge, this is the first study to expound the whole transcriptome involved in denervated muscle atrophy, and provides a theoretical basis for further research targeting ncRNAs.

Keywords: denervated muscle atrophy, sequencing, whole transcriptome, ncRNAs, peripheral nerve injury

Brain Research, 367 (1986) 351–355
Elsevier

351

BRE 21403

Reconnection of severed nerve axons with polyethylene glycol

GEORGE D. BITTNER, MARTIS L. BALLINGER and MARY A. RAYMOND

Department of Zoology, University of Texas, Austin, TX 78712 (U.S.A.)

(Accepted October 29th, 1985)

Key words: axonal severance — reconnection — polyethylene glycol — crayfish

Severed medial giant axons in crayfish can be rejoined in vitro with polyethylene glycol (PEG) to produce axoplasmic continuity and through transmission of action potentials. Severed axon-like processes of a mammalian neuroblastoma/glioma cell line seem to be re-joined to the cell body using PEG in tissue culture. Our data suggest that PEG might be used to rejoin severed axons in vivo in various organisms.

The curious ability of PEG-fusion technologies to restore lost behaviors after nerve severance

G.D. Bittner¹, D.R. Sengelaub², R.C. Trevino³, J.D. Peduzzi⁴, M. Mikesch¹, C.L. Ghergherehchi¹, T. Schallert⁵, and W.P. Thayer⁶

¹Department of Neuroscience, University of Texas at Austin, Austin, TX 78712

Wallerian Degeneration Greatly Reduced !

Abstract

Traumatic injuries to PNS and CNS axons are not uncommon. Restoration of lost behaviors following severance of mammalian peripheral nerve axons (PNAs) relies on regeneration by slow outgrowths and is typically poor or nonexistent if after ablation or injuries close to the soma. Behavioral recovery after severing spinal tract axons (STAs) is poor because STAs do not naturally regenerate. Current techniques to enhance PNA and/or STA regeneration have had limited success and do not prevent the onset of Wallerian degeneration of severed distal segments. This review describes the use of a recently-developed polyethylene glycol (PEG)-fusion technology combining concepts in biochemical engineering, cell biology and clinical microsurgery. Within minutes after micro-suturing carefully-trimmed cut ends and applying a well-specified sequence of solutions, PEG-fused axons exhibit morphological continuity (assessed by intra-axonal dye diffusion) and electrophysiological continuity (assessed by conduction of action potentials) across the lesion site.

Wallerian degeneration of PEG-fused PNAs is greatly reduced as measured by counts of sensory and/or motor axons, and maintenance of axonal diameters and neuromuscular synapses. After

PEG-fusion repair, cut- or crush-severed or ablated PNAs or crush-severed STAs rapidly (within days to weeks), more completely, and permanently restore PNA- or STA-mediated behaviors compared to non-treated or conventionally-treated animals. PEG-fusion success is enhanced or decreased by applying anti-oxidants or oxidants, trimming cut ends or stretching axons, exposure to Ca^{2+} -free or -containing solutions, respectively. PEG-fusion technology employs surgical

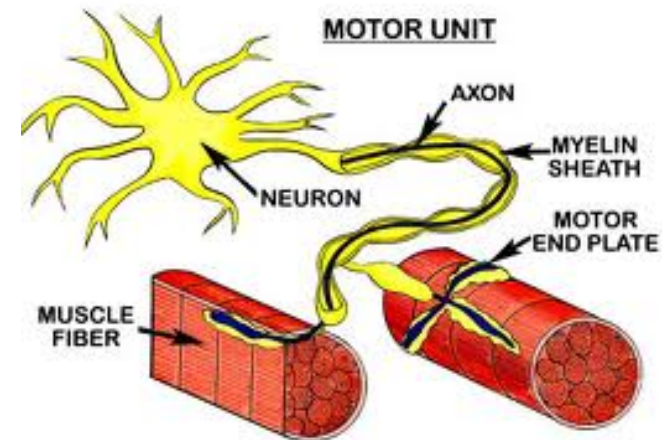
Why aren't we using this for our patients yet ?

A novel therapy to promote axonal fusion in human digital nerves

Ravinder Bamba, MD, Thanapong Waitayawinyu, MD, Ratnam Nookala, MBBS, David Colton Riley, Richard B. Boyer, PhD, Kevin W. Sexton, MD, Chinnakart Boonyasirikool, MD, Sunyarn Niempoog, MD, Nathaniel D. Kelm, MS, Mark D. Does, PhD, Richard D. Dortch, PhD, Robert Bruce Shack, MD, and Wesley P. Thayer, MD, PhD, Nashville, Tennessee

BACKGROUND:	Peripheral nerve injury can have a devastating impact on our military and veteran population. Current strategies for peripheral nerve repair include techniques such as nerve tubes, nerve grafts, tissue matrices, and nerve growth guides to enhance the number of regenerating axons. Even with such advanced techniques, it takes months to regain function. In animal models, polyethylene glycol (PEG) therapy has shown to improve both physiologic and behavioral outcomes after nerve transection by fusion of a portion of the proximal axons to the distal axon stumps. The objective of this study was to show the efficacy of PEG fusion in humans and to retrospectively compare PEG fusion to standard nerve repair.
METHODS:	Patients with traumatic lacerations involving digital nerves were treated with PEG after standard microsurgical neurotaphy. Sensory assessment after injury was performed at 1 week, 2 weeks, 1 month, and 2 months using static two-point discrimination and Semmes-Weinstein monofilament testing. The Medical Research Council Classification (MRCC) for Sensory Recovery Scale was used to evaluate the level of injury. The PEG fusion group was compared to patient-matched controls whose data were retrospectively collected.
RESULTS:	Four PEG fusions were performed on four nerve transections in two patients. Polyethylene glycol therapy improves functional outcomes and speed of nerve recovery in clinical setting assessed by average MRCC score in week 1 (2.8 vs 1.0, $p = 0.03$). At 4 weeks, MRCC remained superior in the PEG fusion group (3.8 vs 1.3, $p = 0.01$). At 8 weeks, there was improvement in both groups with the PEG fusion cohort remaining statistically better (4.0 vs 1.7, $p = 0.01$).
CONCLUSION:	Polyethylene glycol fusion is a novel therapy for peripheral nerve repair with proven effectiveness in animal models. Clinical studies are still in early stages but have had encouraging results. Polyethylene glycol fusion is a potential revolutionary therapy in peripheral nerve repair but needs further investigation. (<i>J Trauma Acute Care Surg.</i> 2016;81: S177–S183. Copyright © 2016 Wolters Kluwer Health, Inc. All rights reserved.)
LEVEL OF EVIDENCE:	Therapeutic study, level IV.
KEY WORDS:	Peripheral nerve injury; polyethylene glycol; axonal fusion; nerve transection; traumatic neuropathy.

If PEG can restore immediate function, then an immediate downstream impact on Nerve and Muscle physiology and pathophysiology must exist after peripheral nerve injury



RESEARCH ARTICLE

An analysis of differential gene expression in peripheral nerve and muscle utilizing RNA sequencing after polyethylene glycol nerve fusion in a rat sciatic nerve injury model

Samantha N. Weiss¹, Joseph M. Legato², Yichuan Liu³, Courtney N. Vaccaro³, Renata Pellegrino Da Silva³, Sandra Miskiel², Grace V. Gilbert², Hakon Hakonarson³, David A. Fuller^{2†}, Russell J. Buono^{1,3†*} 

PLOS ONE

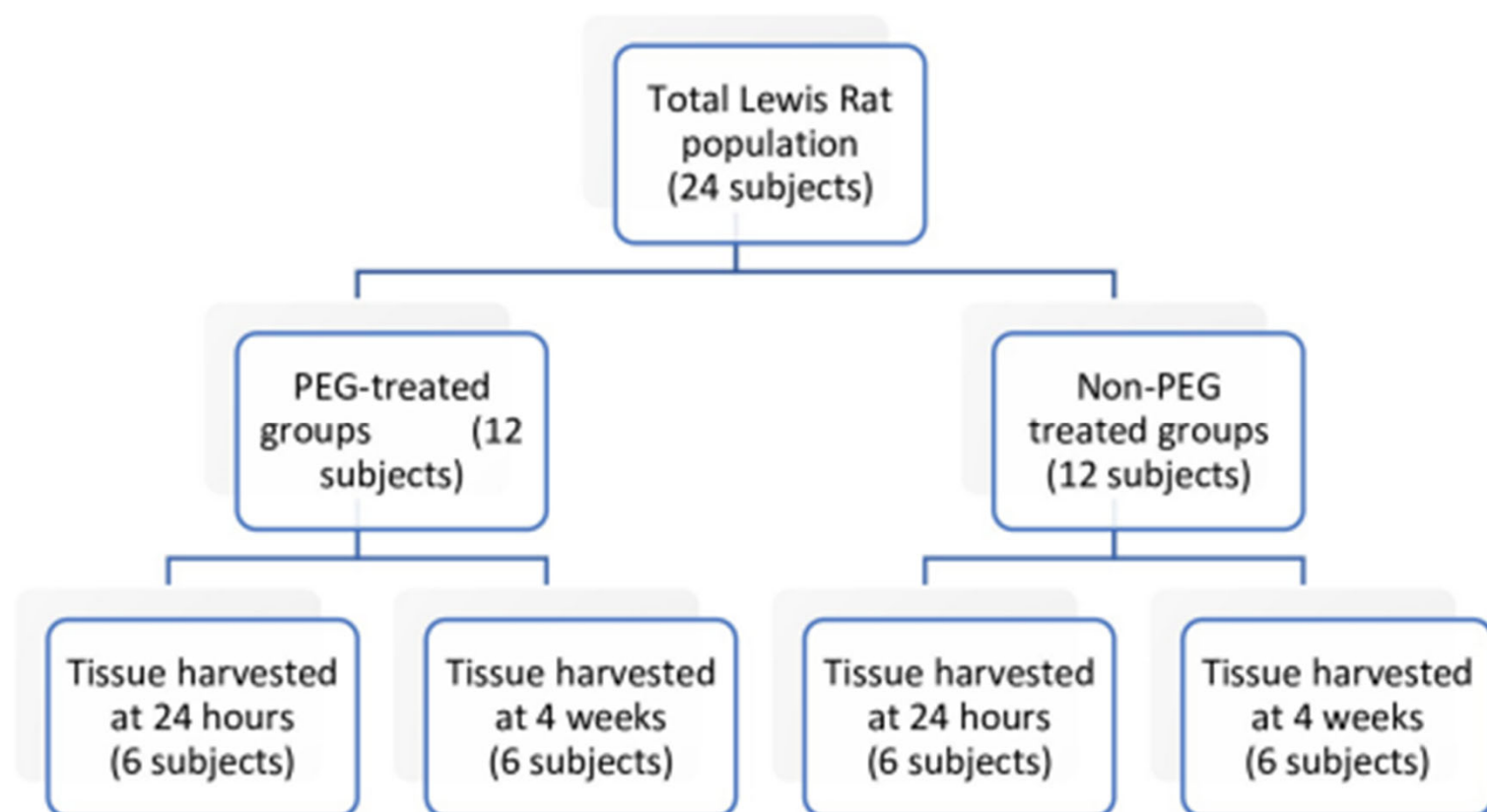


Table 1. PEG induced differential gene expression in sciatic nerve and tibialis muscle.

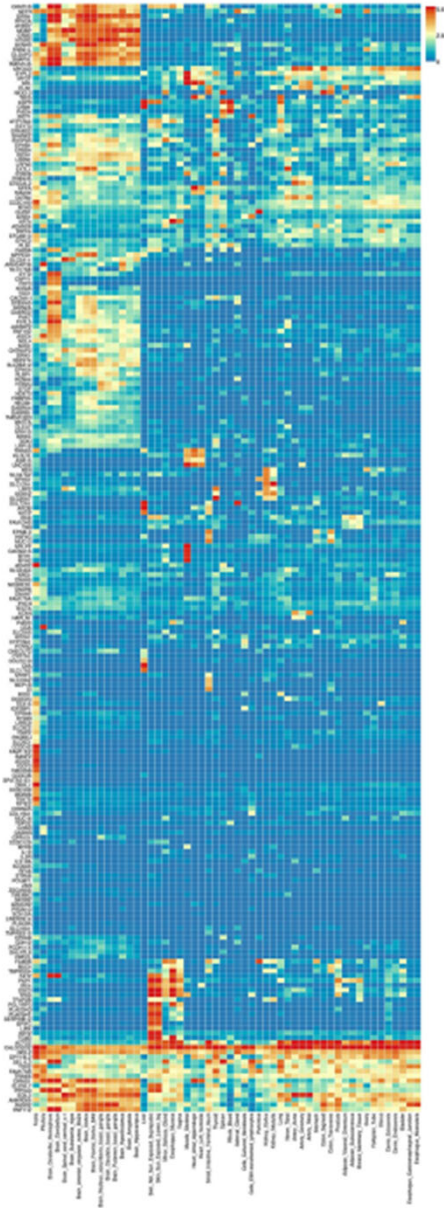
	Number of genes differentially expressed in Sciatic Nerve	Number of genes differentially expressed in Tibialis Muscle
24 Hours	0	1480
4 Weeks	918	3

Nerve Pathways impacted by PEG: 4 weeks

Gene Ontology Pathway	P Value	Corrected P Value
System Development	3.39E-13	9.11E-10
Multi-Cellular Organism Development	5.47E-12	9.79E-09
Anatomical Structure Development	4.83E-10	3.71E-07
Nervous System Development	9.06E-08	5.40E-05
Tissue Development	9.45E-07	3.62E-04
Peripheral Nervous System Development	1.66E-06	5.57E-04
Cell Differentiation	1.66E-06	5.57E-04
Lipid Metabolic Process	2.56E-06	8.09E-04
Sterol Biosynthetic Process	1.10E-05	3.70E-03
Cellular Response to Growth Factor Stimulus	2.03E-05	4.49E-03
Neurogenesis	5.60E-05	8.02E-03
Steroid Biosynthetic Process	7.76E-05	9.91E-03
Neuron Projection Morphogenesis	1.03E-04	1.15E-02
Myelination	1.06E-04	1.16E-02
<u>Ensheatment</u> of Neurons	1.27E-04	1.33E-02
Cell Morphogenesis Involved in Neuron Differentiation	2.52E-04	2.07E-02

Table 2. Differentially Regulated GO Pathways in PEG-Treated Sciatic Nerve Tissue at 4 Weeks

Nerve at 4 weeks



Heatmap of Nerve DEGs at 4 weeks
(Red – Increased, Blue – Decreased)

CNS clusters are located in upper
and lower left corners

Fig 3. Heatmap of rat sciatic nerve DEGs upregulated by 1.5-fold or more at 4-weeks after PEG treatment. Shows individual genes and their intensity of expression across 54 adult human tissues on the GTEX version 8 database. Red indicates high expression and blue indicates low expression for any given gene in different tissues. Intensity measures are only valid in the horizontal direction for a single gene across different tissues and are not valid vertically such that expression of many genes cannot be compared in one tissue.

Muscle Pathways impacted by PEG: 24 hours

Gene Ontology Pathway	P Value	Corrected P Value
Cell Adhesion	4.61E-11	1.19E-07
Inflammatory Response	1.53E-10	2.21E-07
Cytokine-Mediated Signaling Pathway	3.64E-09	3.95E-06
Integrin-Mediated Signaling Pathway	4.65E-09	4.03E-06
Angiogenesis	2.73E-08	1.98E-05
Neutrophil Chemotaxis	9.11E-08	5.65E-05
Positive Regulation of Cell Migration	1.34E-07	6.44E-05
Oxygen Transport	5.72E-07	2.26E-04
Collagen Fibril Organization	7.98E-07	2.89E-04
Innate Immune Response	2.46E-06	6.27E-04
Extracellular Matrix Organization	3.43E-06	8.27E-04
Cartilage Development	5.41E-06	1.17E-03
Actin Filament Bundle Assembly	8.17E-06	1.61E-03
Negative Regulation of Apoptotic Process	8.73E-06	1.65E-03
Wound Healing	1.77E-04	1.98E-02
Endodermal Cell Differentiation	3.00E-04	3.03E-02

Table 3. Differentially Regulated GO Pathways in PEG-Treated Tibialis Anterior Tissue at 24 Hours

Muscle at 24 hours

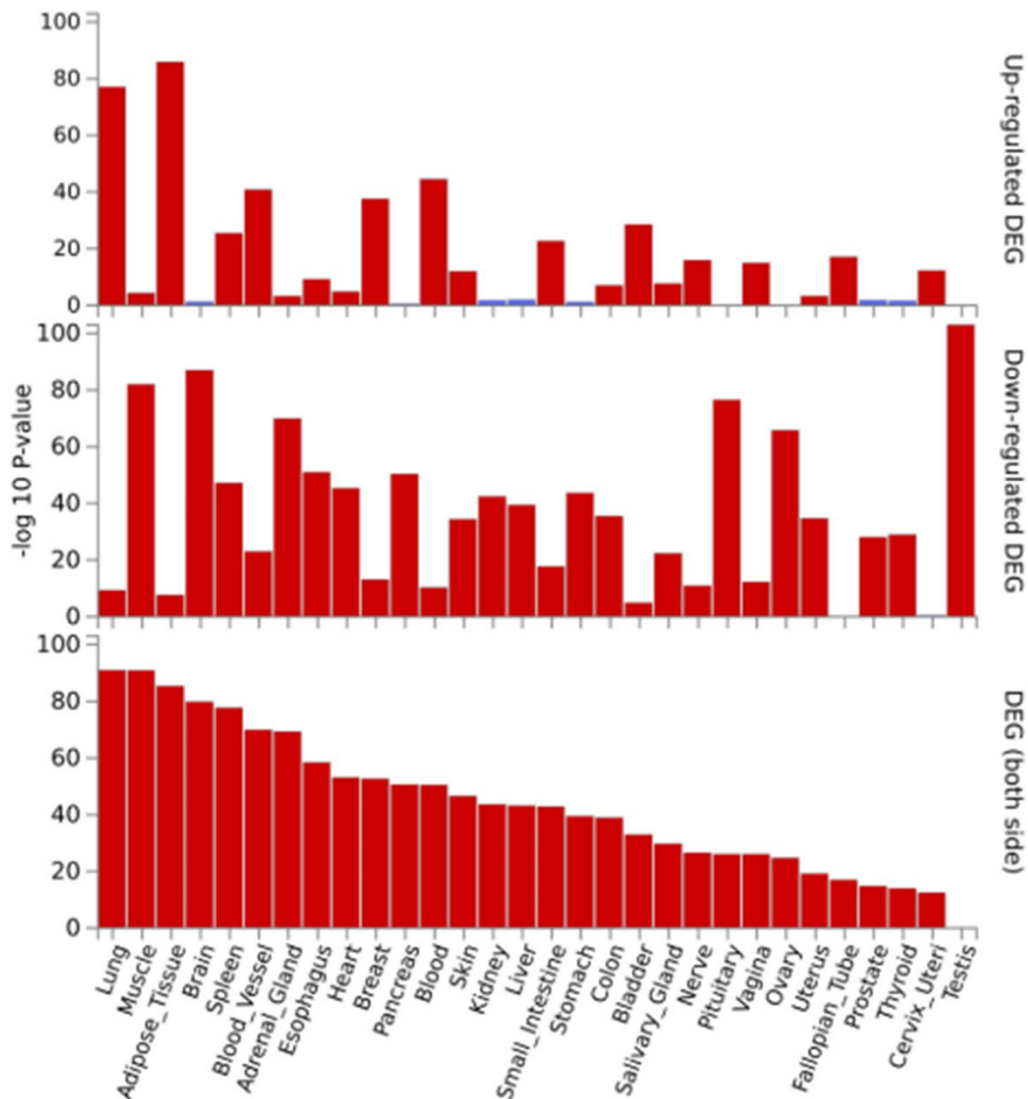


Fig 4. FUMA analysis of 1480 DEGs identified in tibialis muscle at 24 hours after PEG treatment and their qualitative expression in 30 different tissues in GTEx V8. Red bars indicate where input DEG gene sets show significant expression and overlap with GTEx V8 RNAseq data collected from 30 different tissues. Blue (and no) bars are not significant. The FUMA output shows upregulation and downregulation of all genes comparing each genes expression against all other genes and using t-statistics from RNA seq data analysis to determine direction of regulation.

Majority of Muscle DEGs are **down regulated**, suggesting that PEG reduces the proinflammatory pathways induced by the denervation injury

“PEG application during primary neurorrhaphy leads to significant differential gene regulation in the neural and muscular environment that is associated with improved functional outcome compared to non-PEG controls”

RESEARCH ARTICLE

An analysis of differential gene expression in peripheral nerve and muscle utilizing RNA sequencing after polyethylene glycol nerve fusion in a rat sciatic nerve injury model

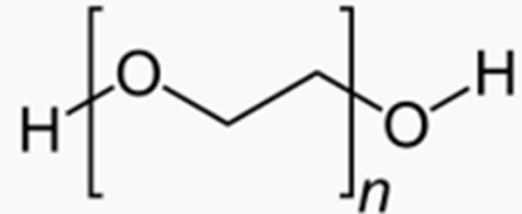
Samantha N. Weiss¹, Joseph M. Legato², Yichuan Liu³, Courtney N. Vaccaro³, Renata Pellegrino Da Silva³, Sandra Miskiel⁴, Grace V. Gilbert², Hakon Hakonarson³, David A. Fuller^{2,4}, Russell J. Buono^{1,2,4} 

- PEG induced differential gene regulation in both distal nerve and muscle contributes to the inhibition of Wallerian degeneration and functional preservation
- Opens window to molecular landscape that could be used to develop specific treatment strategies for PNI

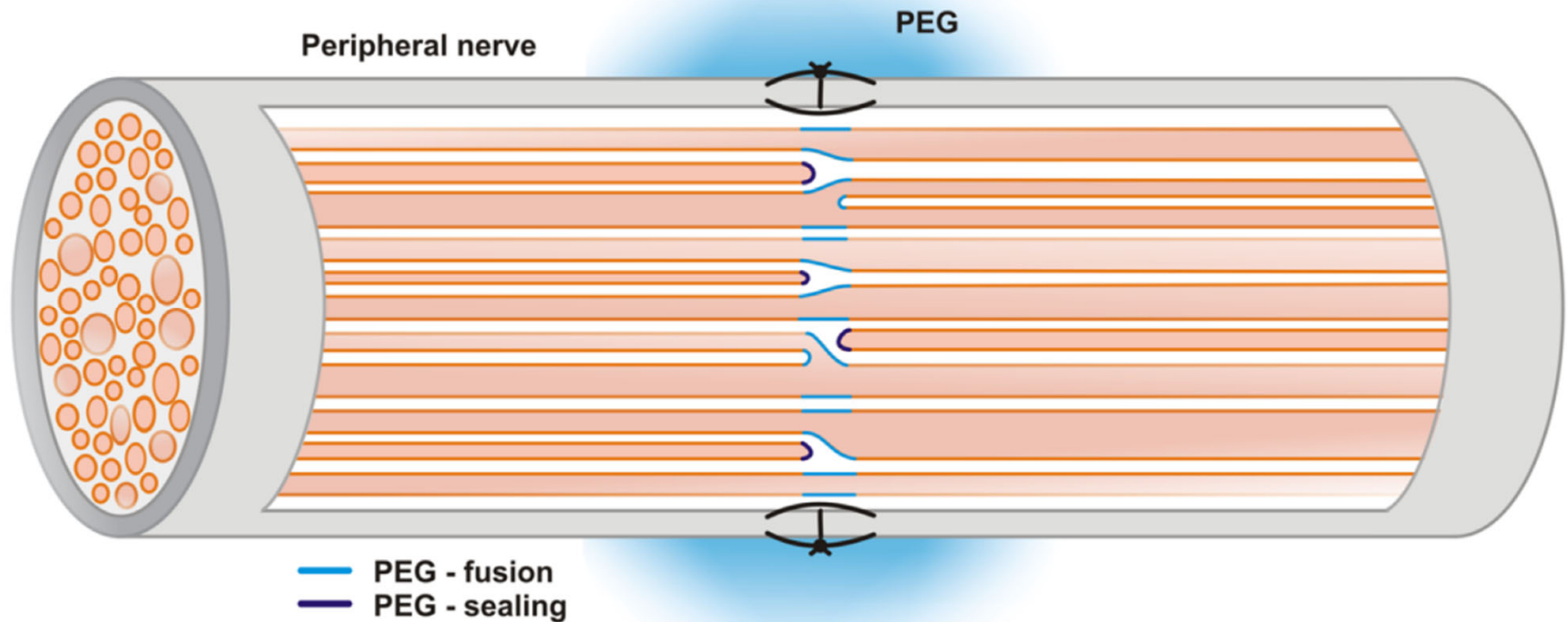
Polyethylene Glycol

- Basis for laxatives
 - *MiraLax*, Bowel Preps
- Used as an excipient in many medical products
 - Oral, topical, parenteral forms

Polyethylene glycol



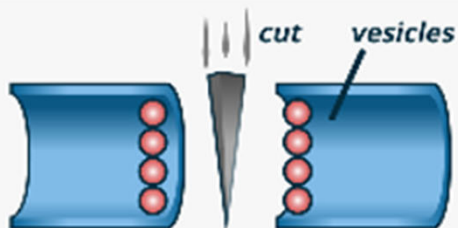
- Steps of Nerve Fusion
- Suturing
- Timing



Technique

Condition

After an axon is severed, in-rushing extracellular Ca^{2+} induces vesicles to plug the ends.



Solution 1: Primer

Ca^{2+} -free, hypotonic saline causes axon swelling to eject the vesicle plugs, thus opening the ends



Solution 2 - Antioxidant

Methylene blue inhibits reformation of vesicles thus keeping the ends open



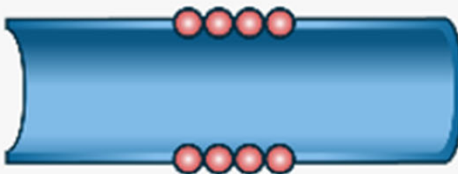
Solution 3 - Fusogen

High-concentration, low-MW PEG in H_2O induces membrane fusion across the severance of closely apposed, open axon ends



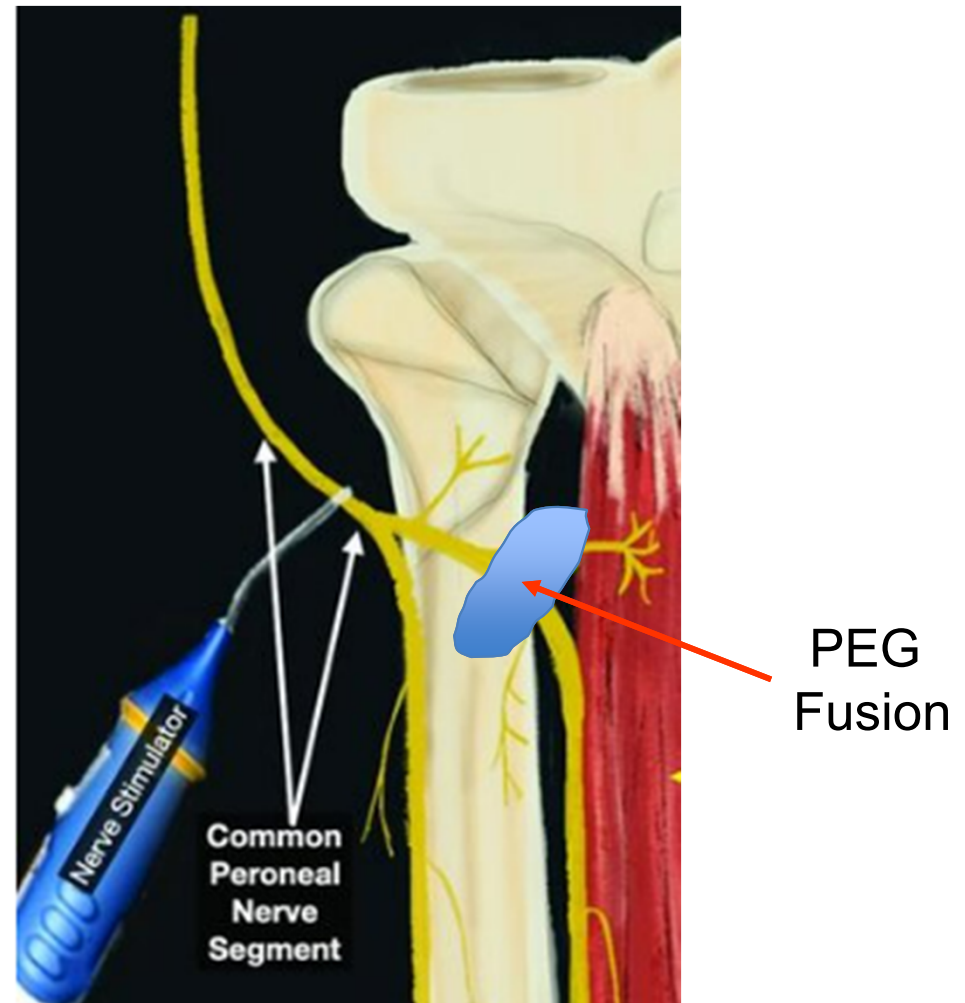
Solution 4 - Sealing

Normal Ca^{2+} -containing saline rinses away residual PEG & induces vesicles to seal any remaining holes in the axonal membrane

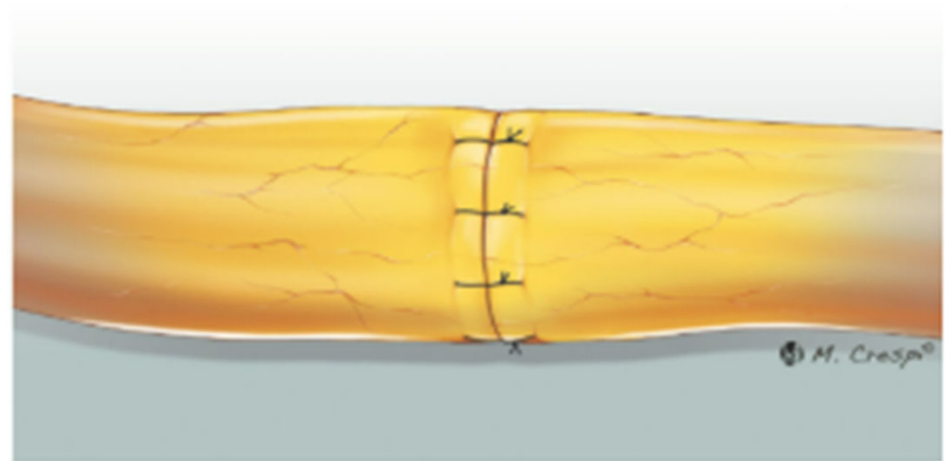
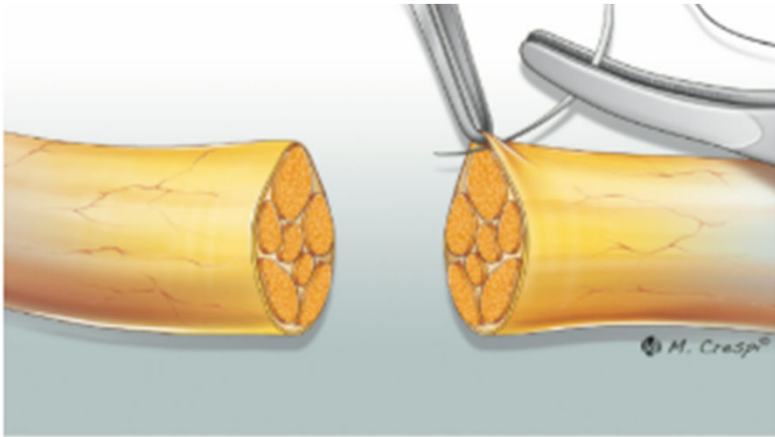


Confirmation of Successful Nerve Fusion

- After PEG nerve fusion complete, confirmation achieved thru intraoperative nerve stimulation with observe muscle twitch distally



Technique



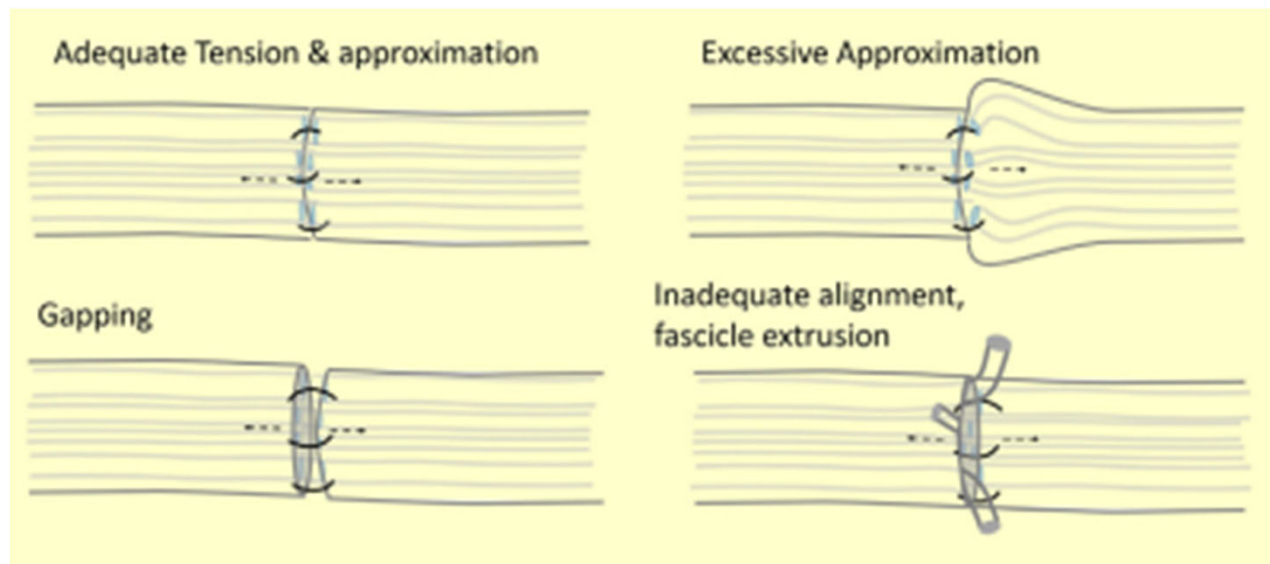
Suturing Done before
PEG application

Avoid overtightening

Need to have nerve
ends opposed
(touching)

Does the Number of Epineural Sutures Matter ?

As we were studying PEG technique, we began to take an interest in the circumferential closure of the epineurium and its contribution to successful PEG nerve fusion



Original Article

A comparison of two versus five epineural sutures to achieve successful polyethylene glycol (PEG) nerve fusion in a rat sciatic nerve repair model

Andrew Simon¹, Grace Victoria Gilbert¹, Alec Hayes Fisher² , Parker Hunt Johnsen¹ , Brandon Herb³, Sebastián L. Vega^{1,3} , Elliot Bodofsky⁴, David A. Fuller¹ 

www.surgicalneurologyint.com



Surgical Neurology International

Editor-in-Chief: Nancy E. Epstein, MD, Professor of Clinical Neurosurgery, School of Medicine, State U. of NY at Stony Brook.

No Differences Detected

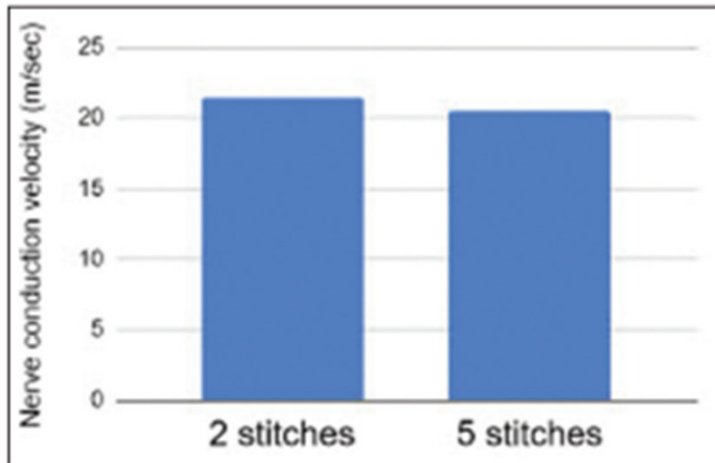


Figure 1: Nerve conduction for two and five suture repairs. No statistically significant difference in nerve conduction velocity between two suture repairs with polyethylene glycol (PEG) fusion and five suture repairs with PEG fusion ($P = 0.444$, Wilcoxon signed-rank test).

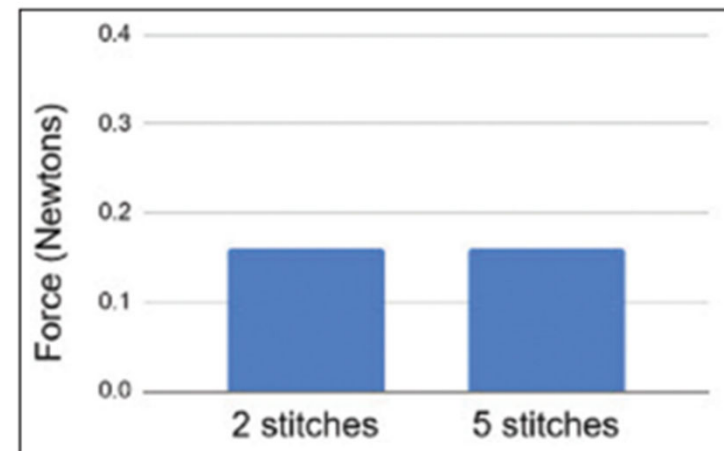


Figure 2: Anterior tibialis force generation for two and five suture repairs. No statistically significant difference in isometric tetanic tibialis anterior muscle force between the two groups ($P = 0.820$, Wilcoxon signed-rank test).

Clinical Implication: # of epineural sutures used remains at discretion of surgeon

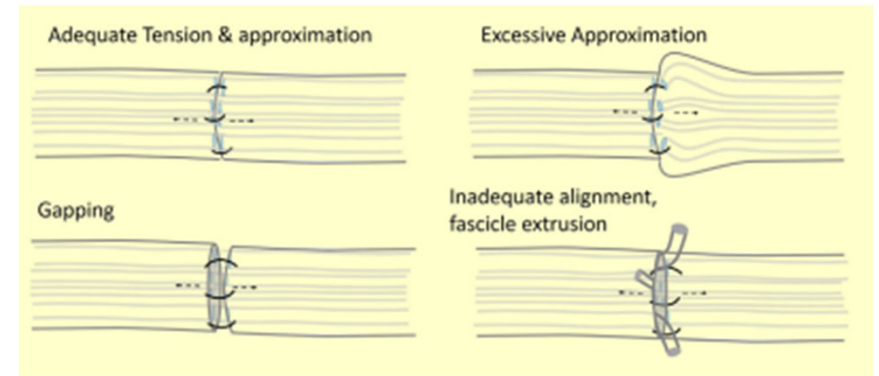
A comparison of two versus five epineural sutures to achieve successful polyethylene glycol (PEG) nerve fusion in a rat sciatic nerve repair model

Andrew Simon¹, Grace Victoria Gilbert¹, Alec Hayes Fisher², Parker Hunt Johnson³, Brandon Herb¹, Sebastián L. Vega¹, Elliot Bodofsky⁴, David A. Fuller⁵

Microhook Device vs Suture

Nerve Suturing Challenges:

Tension at coaptation site
Strength of repair
Alignment of fascicles
Gapping
Fascicle extrusion

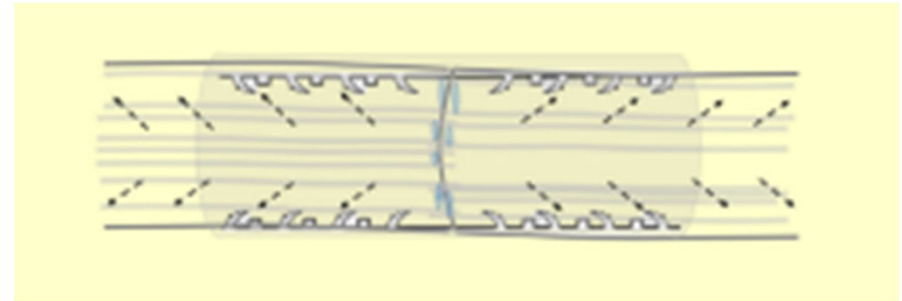


EDITOR'S CHOICE

Microhook Nerve Connector-Assisted Polyethylene Glycol Fusion for Immediate Restoration of Axon Continuity in a Rodent Model

Geetanjali S. Bendale, PhD,* Andrew Azdell, MD,* Jonathan Miller, MD,* Jonathan E. Isaacs, MD*

Microhook Device vs Suture with PEG



Outcomes:

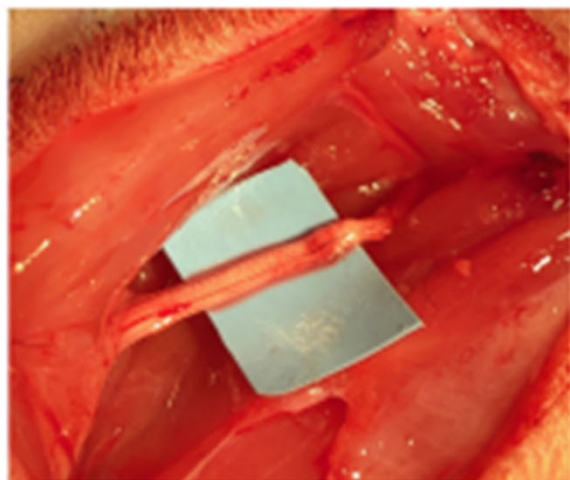
1. Sciatic Functional Index
2. Motor Strength Testing
3. Muscle Mass
4. Retrograde Labeling

EDITOR'S CHOICE

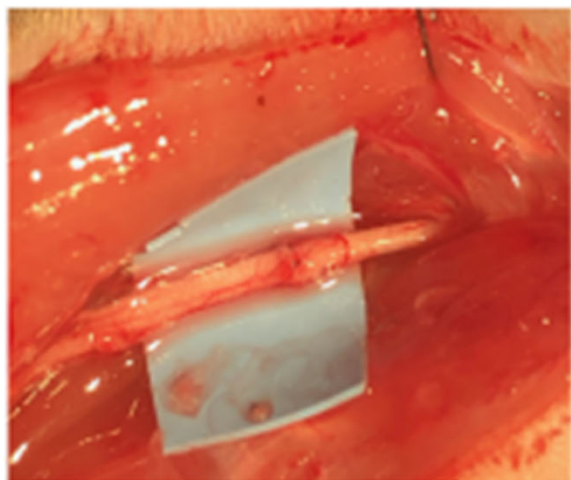
**Microhook Nerve Connector-Assisted
Polyethylene Glycol Fusion for Immediate
Restoration of Axon Continuity in a
Rodent Model**

Geetanjali S. Bendale, PhD,* Andrew Azdell, MD,* Jonathan Miller, MD,* Jonathan E. Isaacs, MD*

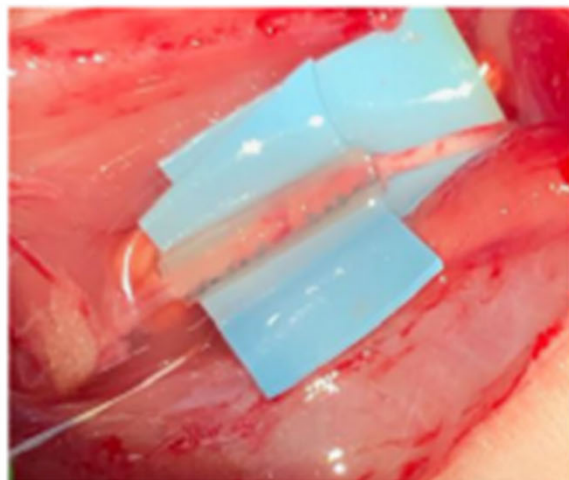
Microsuture

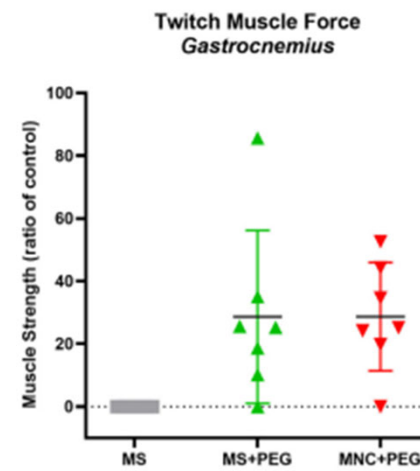
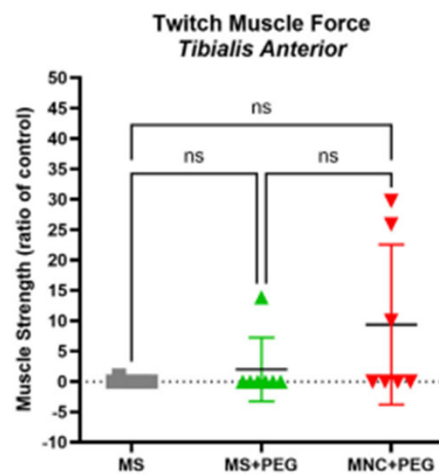
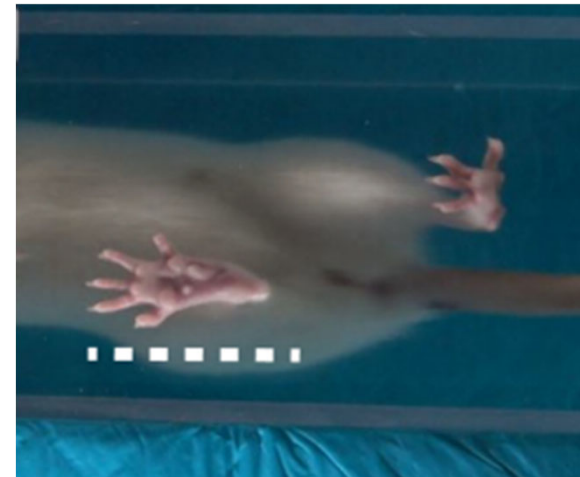
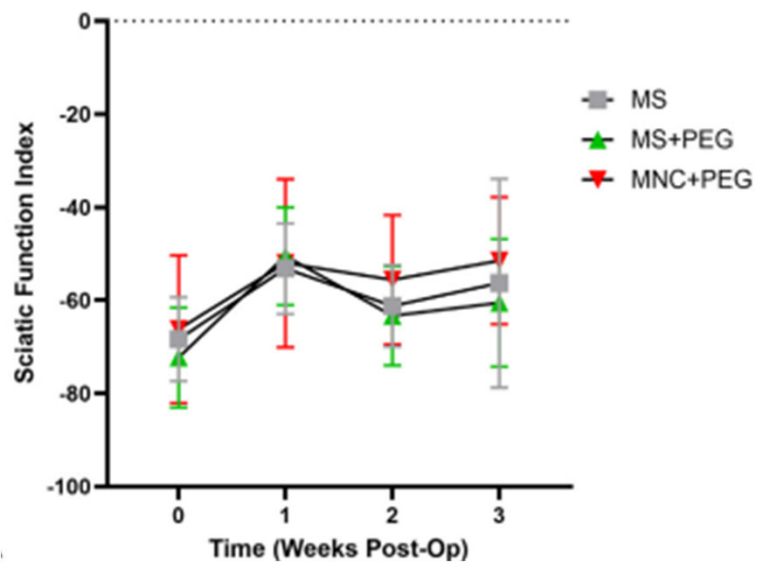


Microsuture+PEG



MNC+PEG





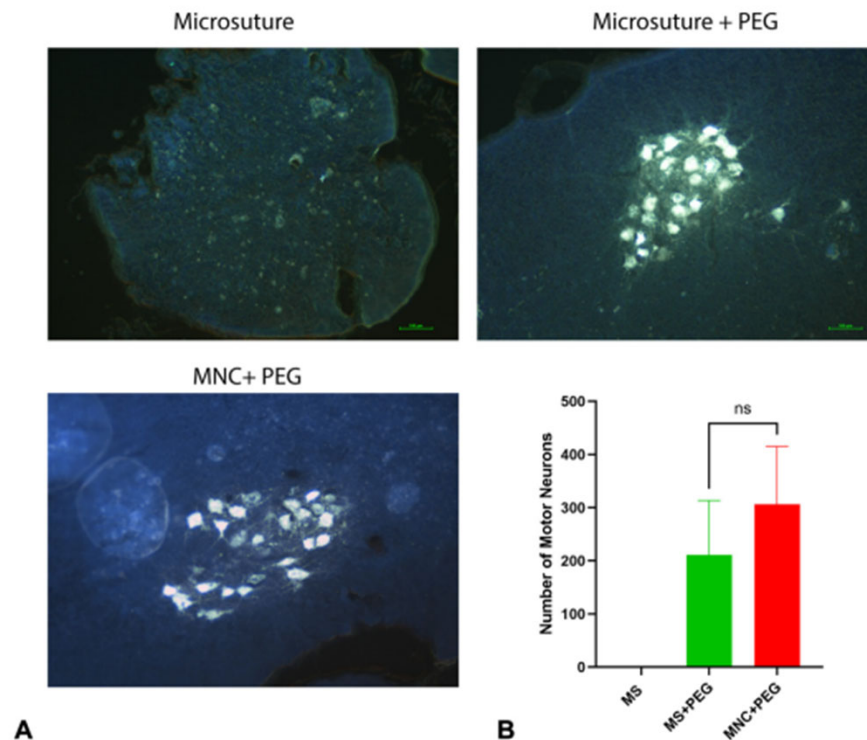
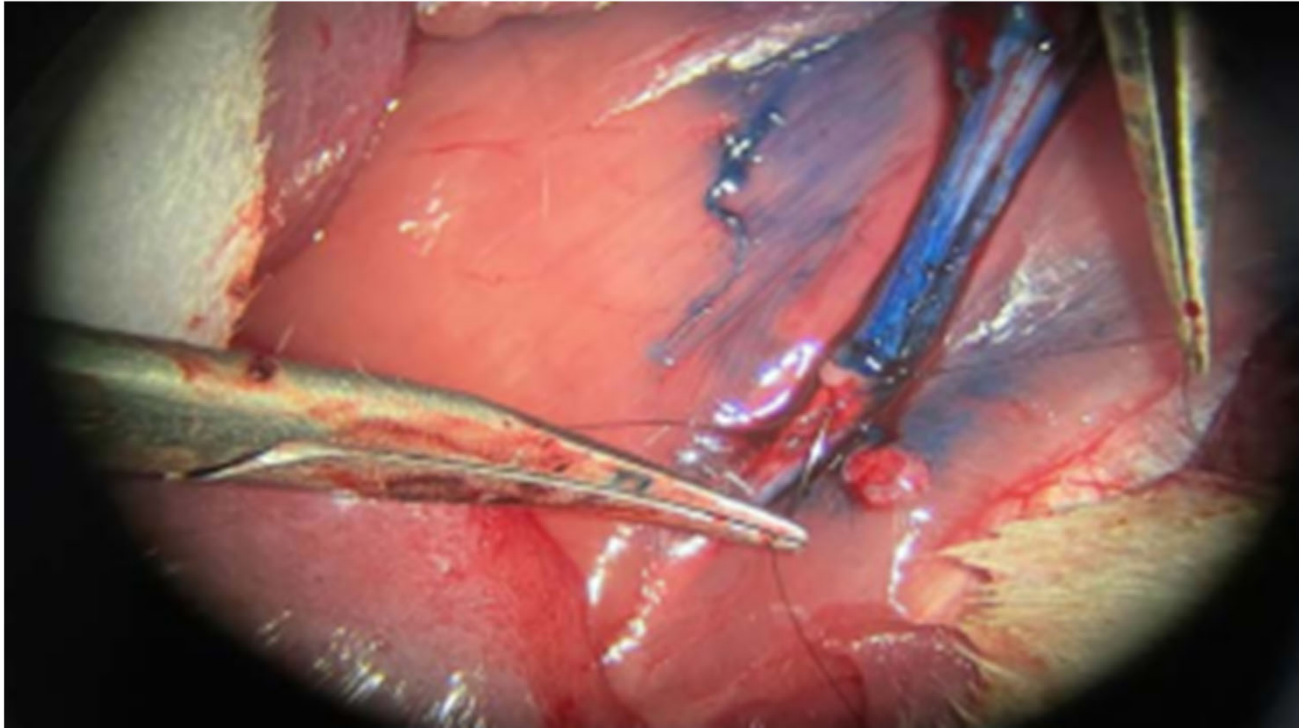


FIGURE 5: Retrograde labeling data. **A** Example images of motor neurons from the ventral horn that were labeled for FluoroGold tracer. **B** Quantification of the total number of motor neurons for each group.

Conclusions:

No advantage seen of microhook nerve connector over suture with PEG
Retrograde labeling confirms fusion of at least some axons in both PEG groups
Muscle contraction preserved in 21/24 PEG vs 0/12 non-PEG

How about fibrin glue at coaptation site ?



OPEN



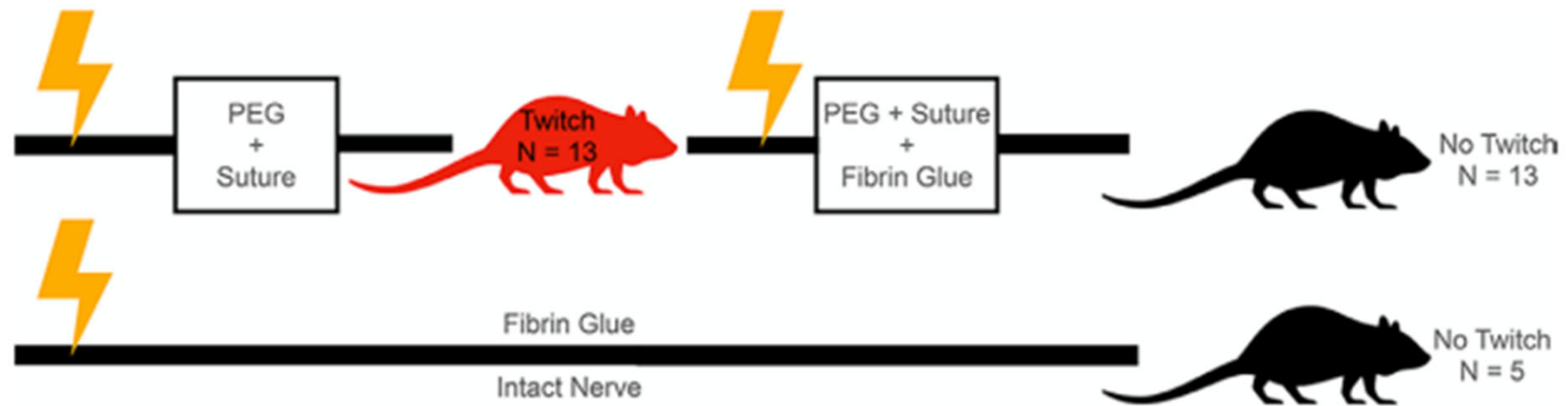
ORIGINAL ARTICLE
Peripheral Nerve

Fibrin Glue Acutely Blocks Distal Muscle Contraction
after Confirmed Polyethylene Glycol Nerve Fusion:
An Animal Study

2024

Alec H. Fisher, MD*
Parker H. Johnsen, MD†
Andrew Simon, MD†
Cameron J. Burns, BS‡
Vineeth Romiyo, BS§
Elliot B. Bodofsky, MD†
Sebastián L. Vega, PHD§
David A. Fuller, MD†§

How about fibrin glue at coaptation site ?



Clinical Implication: Fibrin glue acutely blocks nerve conduction and distal twitch for nerves with or without PEG fusion

*Timing of PEG Application *

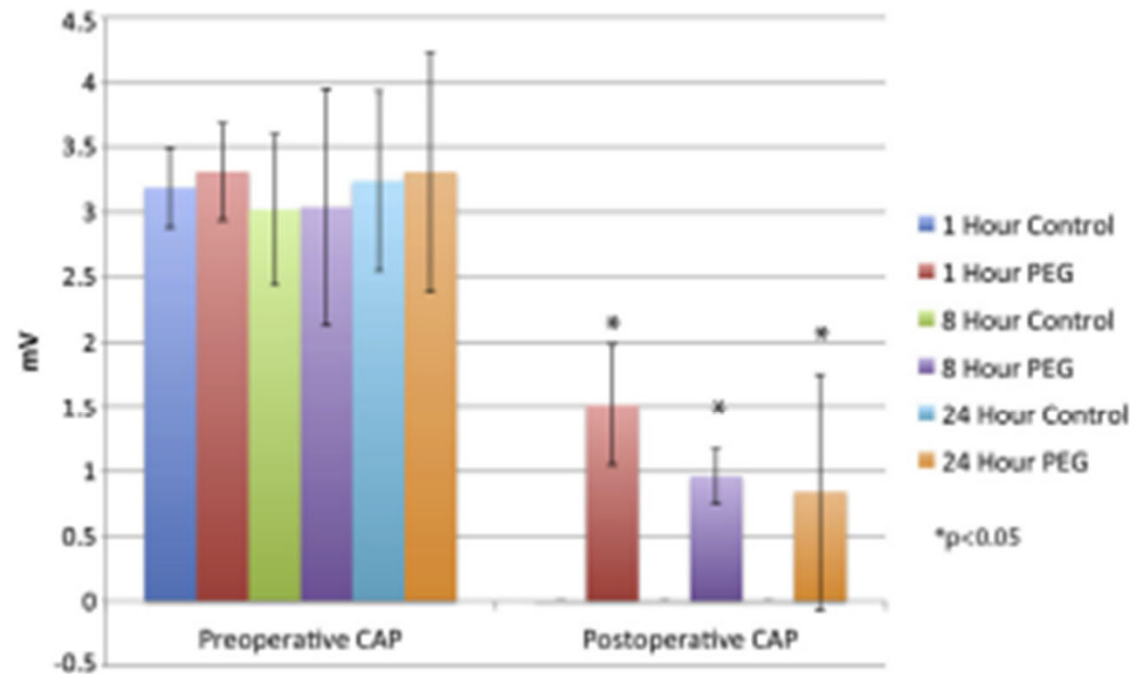


FIGURE 1: Compound action potential of all nerves before and after surgery.

Evaluation of a Nerve Fusion Technique
With Polyethylene Glycol in a Delayed
Setting After Nerve Injury

Ravinder Bamba, MD,*† David Colton Riley, BS,*† Justine S. Kim, BS,* Nancy L. Cardwell, BS,*
Alonda C. Pollins, MLI,* R. Bruce Shack, MD,* Wesley P. Thayer, MD, PhD*

Timing of PEG Application

- Improved Compound Action Potentials for all 3 timepoints
- Sciatic Function index higher at 3 and 7 days for all 3 timepoints
- Distal Motor and Sensory Axon counts higher

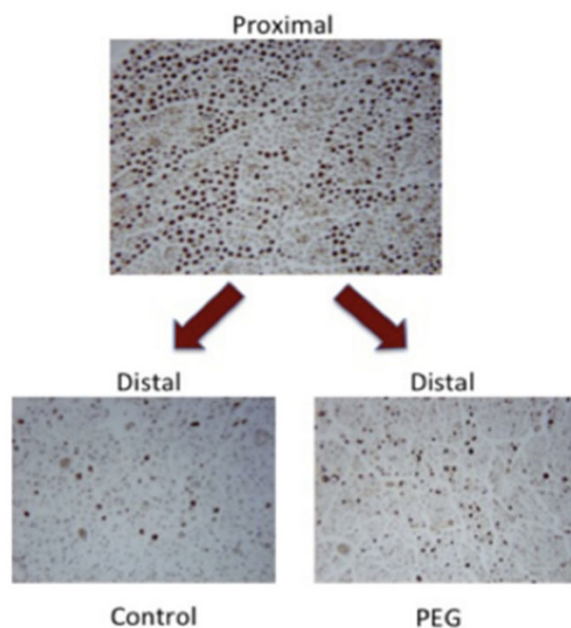


FIGURE 2: Representative photomicrographs of motor axons stained with choline acetyltransferase (magnification $\times 20$).

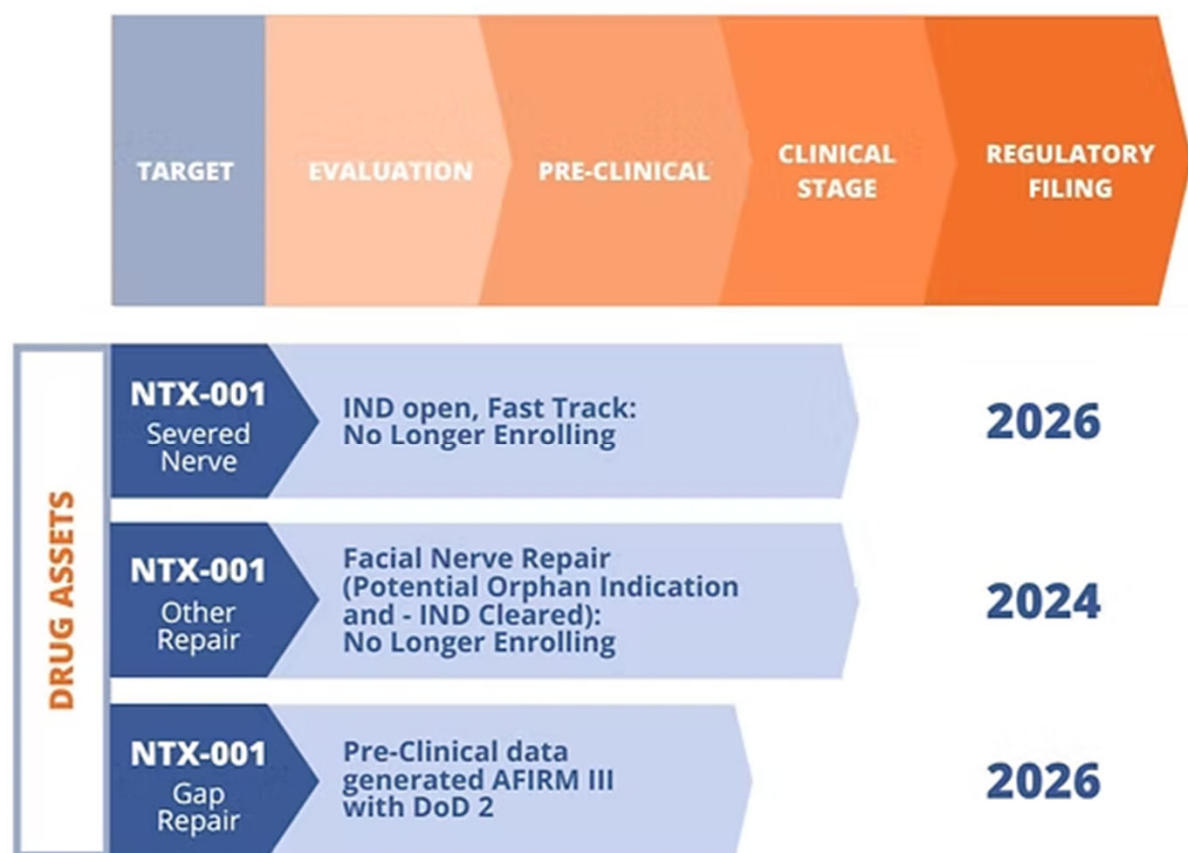
Clinical Implication: OK to use PEG up to 24 hours after nerve injury

**Evaluation of a Nerve Fusion Technique
With Polyethylene Glycol in a Delayed
Setting After Nerve Injury**

Ravinder Bamba, MD,*† David Colton Riley, BS,*† Justine S. Kim, BS,* Nancy L. Cardwell, BS,*
Alonda C. Pollins, MLI,* R. Bruce Shack, MD,* Wesley P. Thayer, MD, PhD*

Product Pipeline

PEG-FUSION in combination with standard neurorrhaphy is superior in improving functional outcomes following peripheral nerve injury compared to standard neurorrhaphy





Completed ⓘ

Safety and Efficacy of NTX-001 Compared to SOC in Acute Single Peripheral Nerve Injuries

ClinicalTrials.gov ID ⓘ NCT04572906

Sponsor ⓘ Neuraptive Therapeutics Inc.

Information provided by ⓘ Neuraptive Therapeutics Inc. (Responsible Party)

Last Update Posted ⓘ 2024-11-20

Sep 19, 2024 12:31 PM Eastern Daylight Time

Neuraptive Therapeutics Announces Presentation of Phase 2 Topline Interim Results for NTX-001 at the 2024 American Society for Surgery of the Hand (ASSH) Annual Meeting

Key interim findings at 24 weeks include:

- NTX-001 demonstrated a significant improvement in MHQ total scores at 8- and 24 weeks post-operation compared to standard of care alone
- There were no safety findings associated with the use of NTX-001
- At all-time points, patients treated with NTX-001 demonstrated significantly reduced post-operative pain compared with SOC alone.

 > **Breaking News**

REGULATORY

Neuraptive Therapeutics' Nerve Repair Surgical Kit Wins FDA Breakthrough Designation

Company is preparing to launch a Phase 3 pivotal program for NTX-001 early next year.

October 3, 2024



Recruiting ⓘ

This Study Will Evaluate the Effectiveness of NTX-001, a Surgical Nerve Repair Product When Used in People With Upper Extremity Nerve Lacerations.

ClinicalTrials.gov ID ⓘ NCT06616025

Sponsor ⓘ Neuraptive Therapeutics Inc.

Information provided by ⓘ Neuraptive Therapeutics Inc. (Responsible Party)

Last Update Posted ⓘ 2025-03-18



[Home](#) > [Search Results](#) > Study Record

Recruiting ⓘ

Evaluating the Safety and Efficacy of Polyethylene Glycol (PEG) Mediated Fusion (PEG Fusion)

ClinicalTrials.gov ID ⓘ NCT04789044

Sponsor ⓘ Major Extremity Trauma Research Consortium

Information provided by ⓘ Major Extremity Trauma Research Consortium (Responsible Party)

Last Update Posted ⓘ 2024-12-20

- PEG nerve fusions likely headed to clinical practice
 - 40 years of basic science
 - animal studies complete, human trials
- Peripheral nerve repairs may become time sensitive
 - < 24 (*maybe 48*) hours

Can we block Wallerian Degeneration ?

- YES
 - Animal models definitive
 - Technique dependent
 - Ongoing work to improve outcomes
- Human trials ongoing
- Why has it taken so long to reach human use ?

Thank you



Question 1:

Peripheral nerve injuries currently

- 1) Are all repaired emergently (<24 hours)
- 2) Can be repaired by plastic, orthopaedic, neuro or general surgeons with training
- 3) Are treated only at Level 1 trauma centers
- 4) Must be repaired with microscopic sutures
- 5) All require surgery

Question 2:

A 27 year old male sustains a traumatic laceration in his volar, distal forearm when struck with a knife. He complains of immediate numbness in his thumb, index and long fingers. He has weakness in his thumb. His median nerve will likely:

- 1) be contused and recovery with observation
- 2) undergo Wallerian degeneration unless repaired surgically
- 3) will recover in 2-3 months with surgical repair
- 4) will always require a nerve graft to repair it
- 5) will undergo Wallerian degeneration even with surgical repair

Question 3:

Polyethylene Glycol (PEG)

- 1) is not currently FDA approved for any medical usage
- 2) is FDA approved for nerve repair
- 3) has shown promising results in nerve fusion to protect nerve function
- 4) is expensive
- 5) has not been studied in human nerve repair

Question 4:

Animal studies (primarily rodent) using PEG at the time of a nerve repair have shown that:

- 1) Immediate, normal nerve function can occur with PEG application
- 2) PEG can be used on its own to repair the ends together of injured nerves
- 3) Signals can be conducted immediately across a nerve repair that has been performed with PEG
- 4) Application of PEG can only be performed successfully 2 or more days after a nerve injury
- 5) Application of PEG to a nerve repair will have no impact on mRNA expression in the distal muscles

Question 5:

Areas under study to define the role of PEG in for human benefit with nerve repair include:

- 1) Only digital sensory nerves
- 2) Only larger mixed motor/sensory nerves
- 3) Both digital and mixed motor/sensory nerves
- 4) Nerve transfers only
- 5) Children only

I just had surgery on my arm and my nerves aren't working, What do I do now ?

David A. Fuller, MD
Professor of Orthopaedic Surgery

Rocky Mountain University
2026 Annual Meeting
Provo, Utah

March 28, 2026

- No financial disclosures relevant

Objectives

- Discuss Incidence of Intraoperative Nerve Injuries with Upper Extremity Surgeries
- Discuss Diagnosis and Prevention Strategies
- Discuss Treatment Strategies for Such Nerve Issues

Core Values

Authentic

We recognize our imperfections and welcome growth stemming from both success and failure. We believe in our unlimited capacities to find solutions. We model gratitude. We seek to understand, to empathize, and to forgive.



**Rocky Mountain
University**
of Health Professions

Nerve Injury

- IntraOperative
(iatrogenic)
- PostOperative
- Underlying
(pre-existing)



Nerve Injury

- IntraOperative
(iatrogenic)
- PostOperative
- Underlying
(pre-existing)

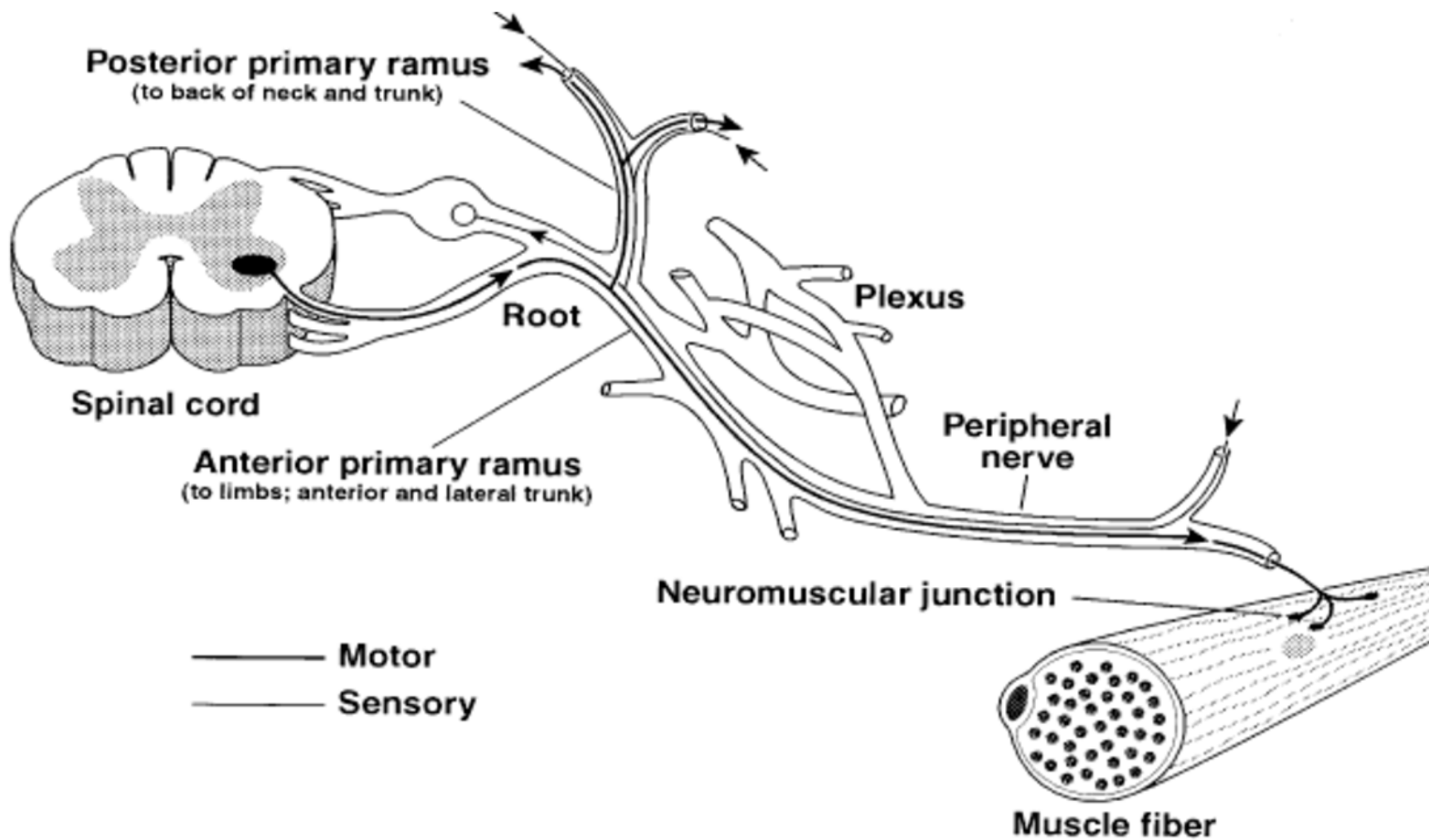


- Direct Trauma

- Retractors
- Knives, Scissors, Sutures, Drills
- Thermal

- Indirect Trauma

- Positioning
- Tourniquets
- Swelling
- Stretch
- Ischemia
- Other metabolic



Classification

Tissue Damage

Recovery

Epineurium

Perineurium

Endoneurium

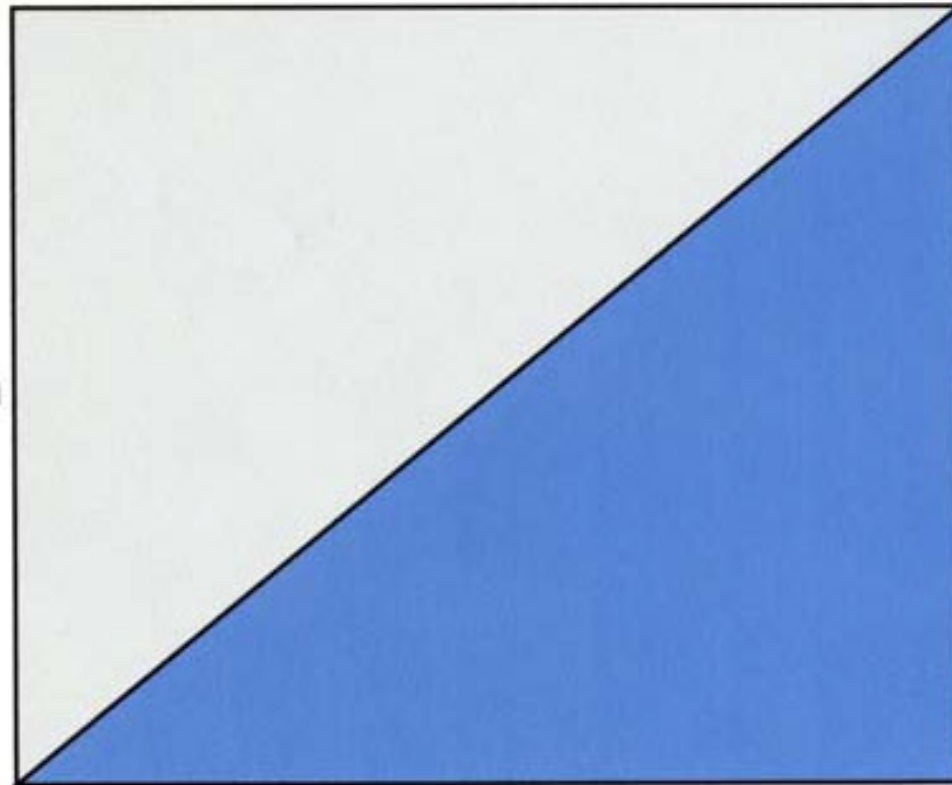
Axon

Myelin

None

Incomplete

Full



Seddon Grade

Neurapraxia

Axonotmesis

Neurotmesis



- Colleague (surgeon), 68 yo male
- Right hand numbness – median innervated distribution
- He had already had EMG/NCS done:

EMG/NCS evaluation

Sensory Summary Table

Stim Site	NR	Onset (ms)	Peak (ms)	Norm Peak (ms)	O-P Amp (µV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Median D2 Sensory (2nd Digit) 26.8 °C												
Wrist		3.6	4.5	<3.8	11.6	>10.0	Wrist	2nd Digit	3.6	14.0	39	>50.0
Right Ulnar Sensory (5th Digit)												
Wrist		2.6	3.1	<3.2	7.9	>5.0	Wrist	5th Digit	2.6	14.0	54	>50.0
Left Median D2 Sensory (2nd Digit)												
Wrist		3.0	3.8	<3.8	24.5	>10.0	Wrist	2nd Digit	3.0	14.0	47	>50.0
Left Ulnar Sensory (5th Digit)												
Wrist		2.4	3.0	<3.2	10.7	>5.0	Wrist	5th Digit	2.4	14.0	58	>50.0

Motor Summary Table

Stim Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Neg Dur (ms)	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Median Motor (Abd Poll Brev)												
Wrist		4.5	<4.0	5.4	>5.0	4.84	Wrist	Abd Poll Brev	4.5	7.0		
Elbow		9.1		5.2	>5.0	5.31	Elbow	Wrist	4.6	25.5	55	>50.0
Right Ulnar Seg Motor (Abd Dig Minimi)												
Wrist		2.3	<3.1	7.7	>7.0	5.47	Wrist	Abd Dig Minimi	2.3	7.0		
B Elbow		6.1		5.5	>7.0	5.94	B Elbow	Wrist	3.8	25.0	66	>50.0
A Elbow		9.2		4.2	>7.0	6.41	A Elbow	B Elbow	3.1	11.5	37	>50.0
Axilla		10.5		4.5	>7.0	6.72	Axilla	A Elbow	1.3	10.5	81	>50.0
MarGru	NR				>7.0		Erb's Pt	Axilla		0.0		>50.0

EMG

Side	Muscle	Root	Ins Act	Fibs	Psw	Fascic	Recruit	Rate	Dur	Amp	Poly	Other	Comment
Right	Deltoid	C5-6	Nml	None	None	None	Nml	Nml	Nml	Nml	Nml	None	
Right	PronatorTeres	C6-7	Nml	None	None	None	Nml	Nml	Nml	Nml	SI	None	
Right	Triceps	C6-7-8	Nml	None	None	None	Nml	Nml	Nml	Nml	SI	FWU	
Right	Abd Poll Brev	C8-T1	Nml	None	None	None	Nml	Nml	SI	Nml	Nml	None	
Right	1stDorInt	C8-T1	Nml	None	None	None	Dec	Nml	SI	Nml	Nml	None	
Right	FlexCarpiUln	C8,T1	Nml	None	None	None	Dec	Nml	SI	Nml	Nml	None	

Carpal Tunnel: Post Surgical Nerve Deficits

- Colleague (surgeon), 68 yo male
- Right hand numbness – median innervated distribution
- Surgical (vs NonSurgical)
 - Right Carpal and Cubital tunnel releases (mini-open)
 - Local with Sedation
- PostOperative Plan
 - Trip to Florida
 - RTW 7-10 days



- Phone Call:
“my fingers are still numb”

“my fingers are still numb”

- My feelings:



- Observe
- Study
 - U/S, MRI, EMG/NCS
- Explore

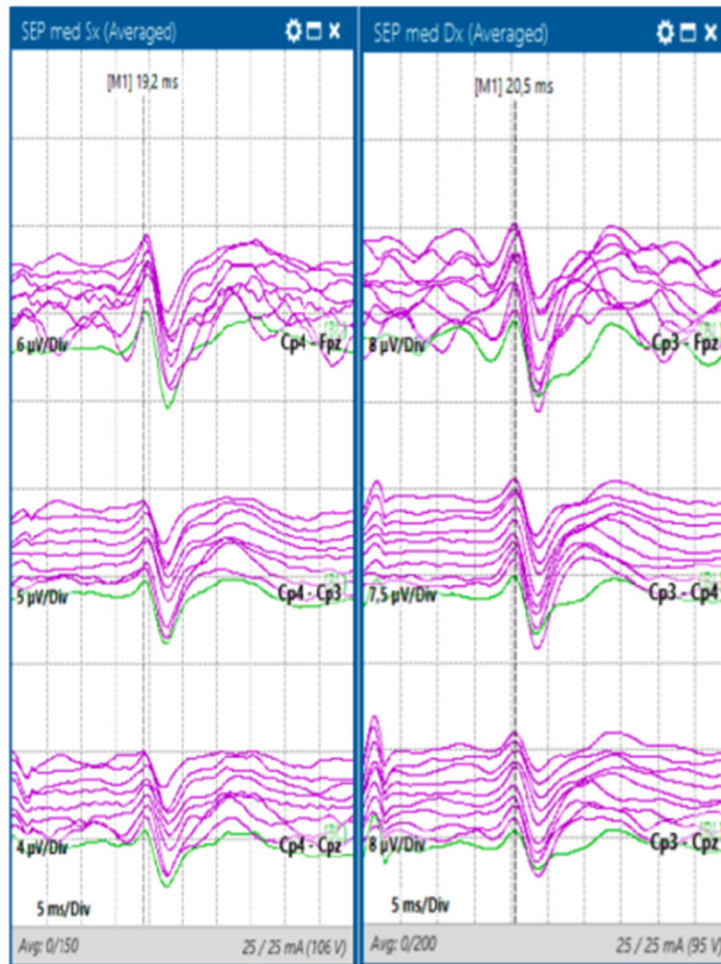
HISTORY and PHYSICAL EXAMINATION ESSENTIAL

Feeling Returns 2-3 days !

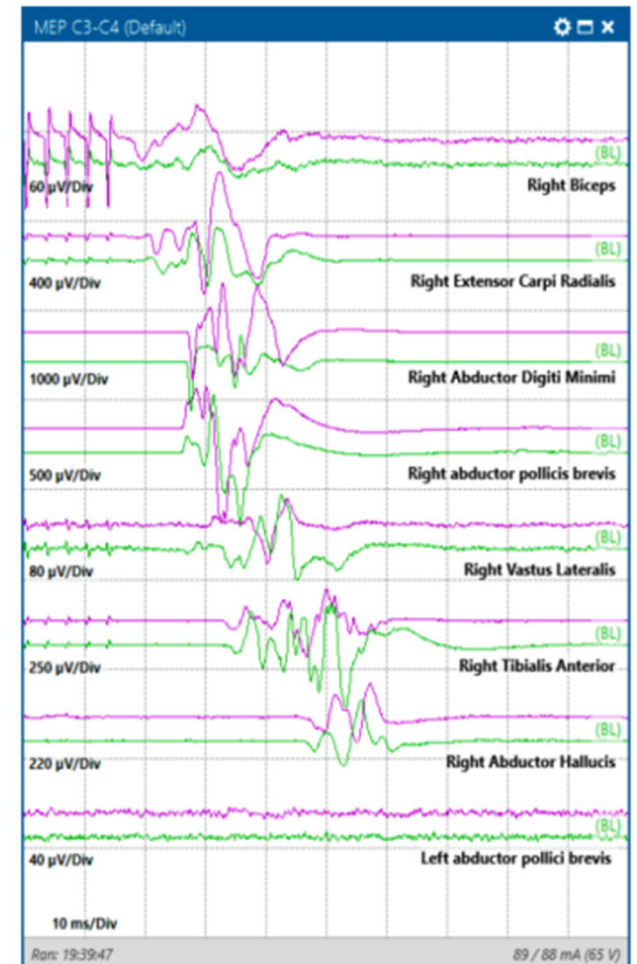


Intraoperative Neuromonitoring (IONM)

- EMG
- SSEP
- MEP



SSEPs



MEPs

- IONM thought to enhance procedural safety and precision
- Adoption of multimodal IONM (EMG, SSEP, MEP) is frequently deemed necessary for spine surgery
- Several recent studies however have yielded inconclusive evidence regarding improved outcomes when using IONM
- Lack of consensus

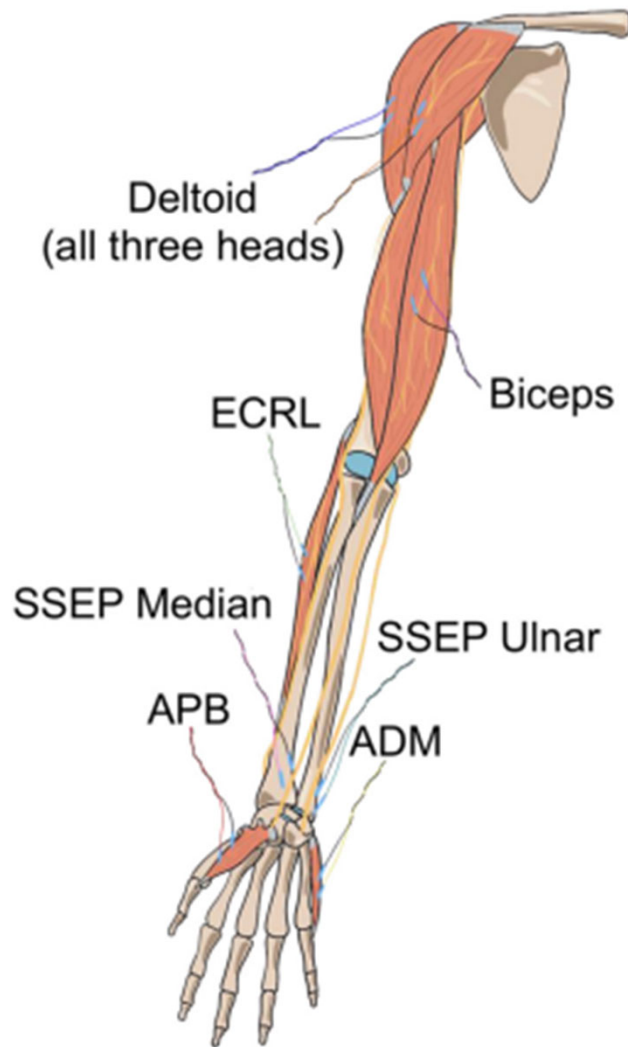
- IONM used for consecutive series (44) of rTSA
- EMG, SSEP, MEP
- 0% major/minor nerve injury postoperatively
- 4 (9%) had distal peripheral neuropathy postoperatively
- Retrospective, no control group
- *“difficult to assess the role that IONM plays in preventing nerve injuries”*

Incidence of peripheral nerve injury in revision total shoulder arthroplasty: an intraoperative nerve monitoring study

Manan S. Patel, BA^{a,*}, W. Bryan Wilent, PhD^b, Michael J. Gutman, BA^a, Joseph A. Abboud, MD^a



IONM: Shoulder



Lead Placements:

- 1) 1 each for all major UE nerves:
Axillary, Musculocutaneous,
Radial, Median and Ulnar
- 2) 2 superficially for SSEP median
and ulnar

Incidence, Risk Factors, Prevention, and Management of Peripheral Nerve Injuries Following Shoulder Arthroplasty



Manan S. Patel, MD^a, Mohammad Daher, BSc^b,
David A. Fuller, MD^c, Joseph A. Abboud, MD^{d,*}

Ortho Clinic N Amer 2022

Intra-Operative Nerve “Injuries”: Shoulder

CURRENT CONCEPTS REVIEW

Prevention and Treatment of Nerve Injuries in Shoulder Arthroplasty

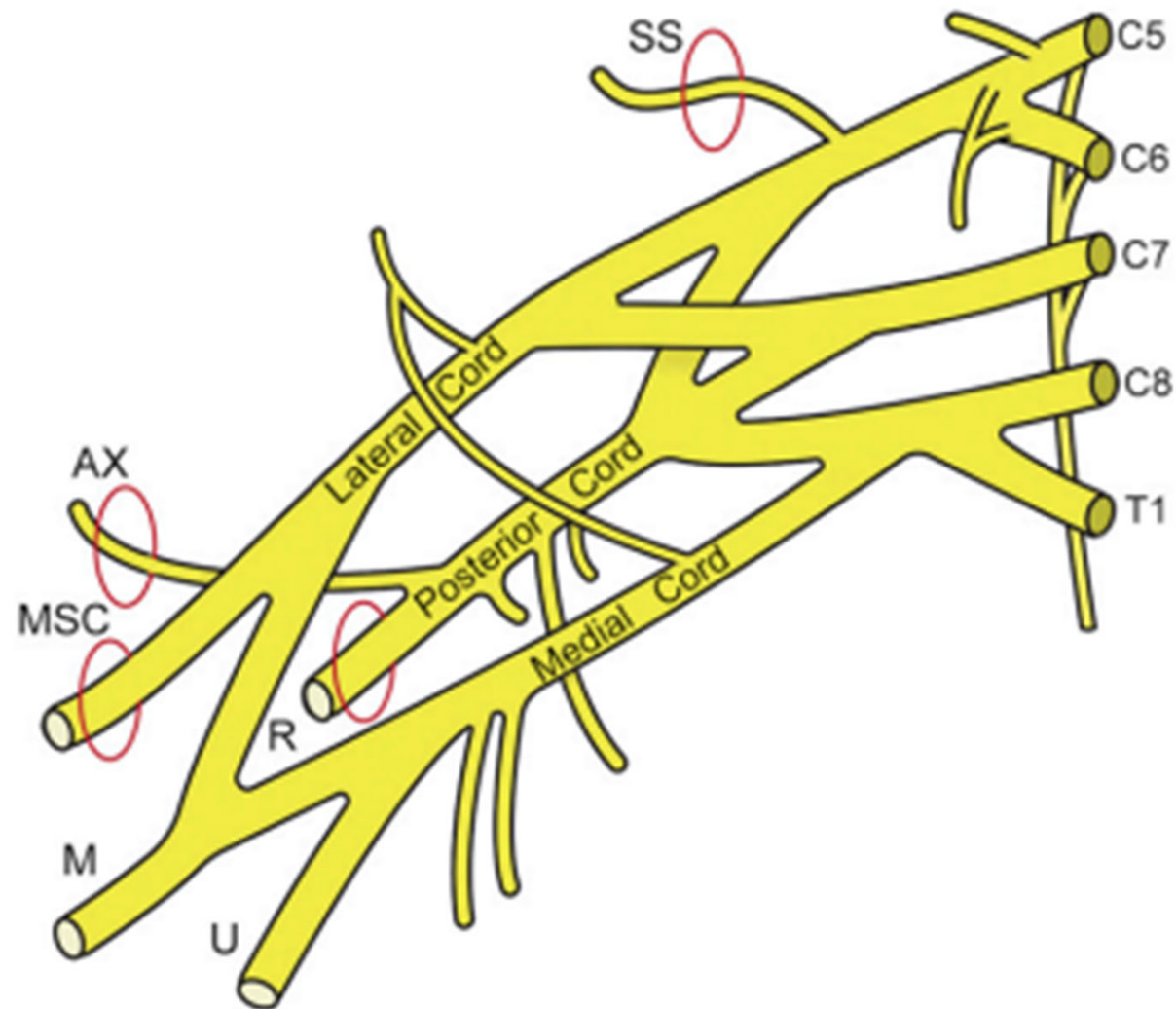
- Nerve injuries during shoulder arthroplasty have traditionally been considered rare events, but recent electrodiagnostic studies have shown that intraoperative nerve trauma is relatively common.
- The brachial plexus and axillary and suprascapular nerves are the most commonly injured neurologic structures, with the radial and musculocutaneous nerves being less common sites of injury.

Matthew Florczynski, MD, MSc, Ryan Paul, MD, FRCSC, Timothy Leroux, MD, MEd, FRCSC, and
Heather Baltzer, MD, MSc, FRCSC, FACS

- Clinically Apparent Nerve Injuries
 - 0.63% TSA
 - 1.2 % Rev TSA
 - (higher than other arthroplasties)
- Electrodiagnostic Studies
 - 31.5 % to 97 %
- Ranging from Neuropraxia to Neurotmesis

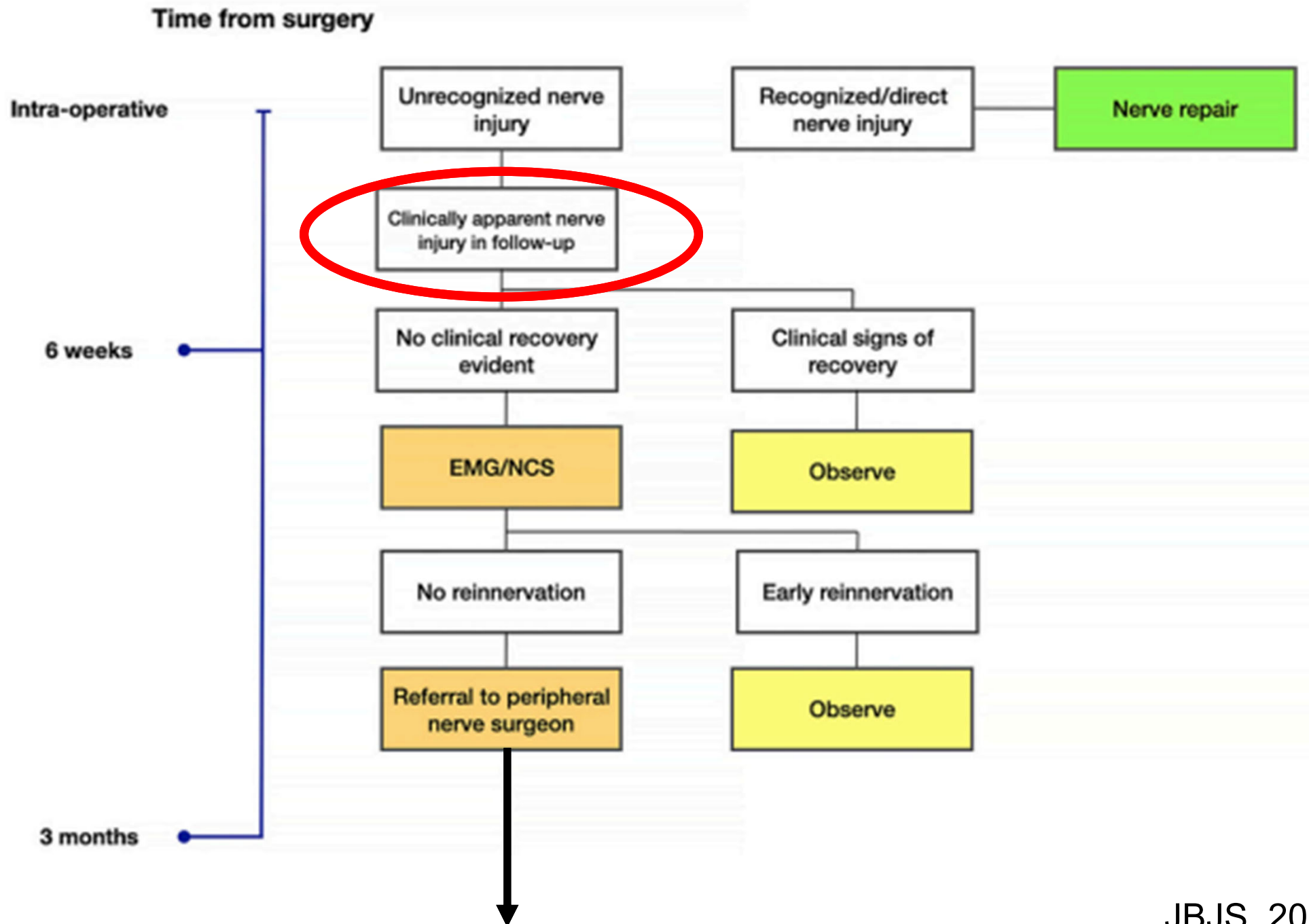
*Decreased Preoperative shoulder motion linked to increased risk
JBJS, 2021

“most susceptible” nerves to injury during TSA

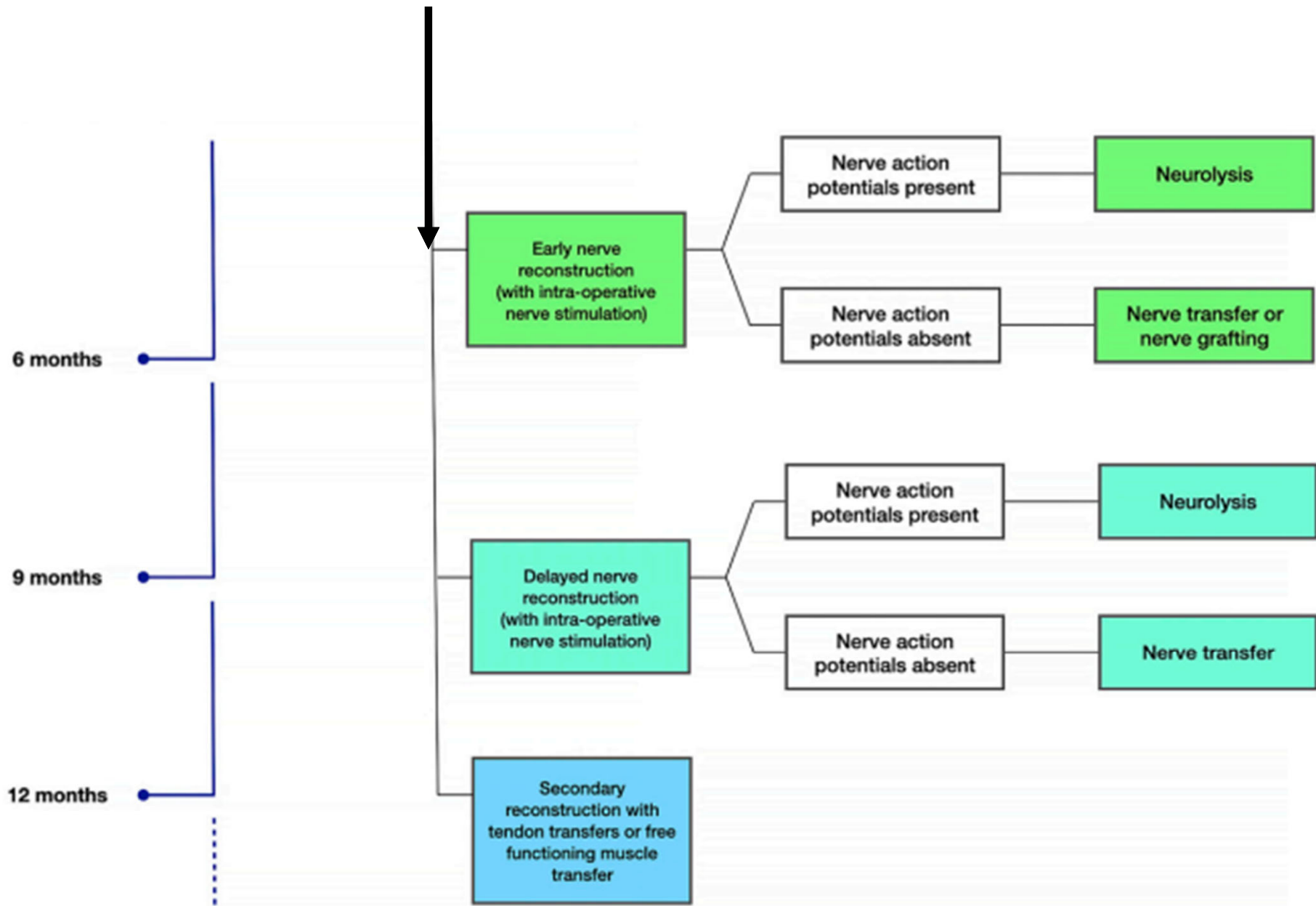


- Direct Trauma
 - Surgical Approach, Dissection, Exposure
- Indirect Trauma
 - Intra-Operative Arm Positioning
 - Lengthening
- Treatment Algorithm Proposed:

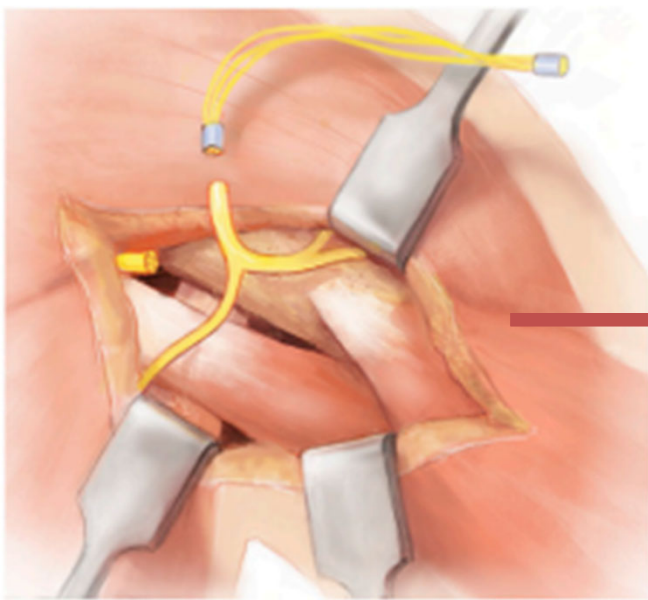
Recommendations: 1st 3 months post injury



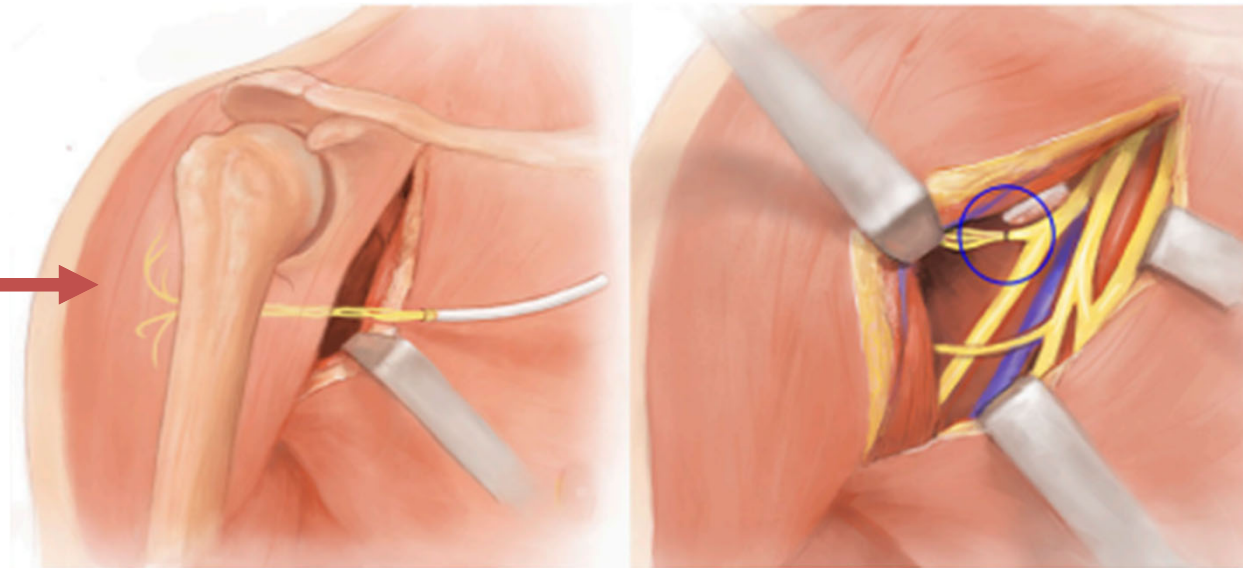
Recommendations: After 3 months



Axillary Nerve Reconstruction with Graft

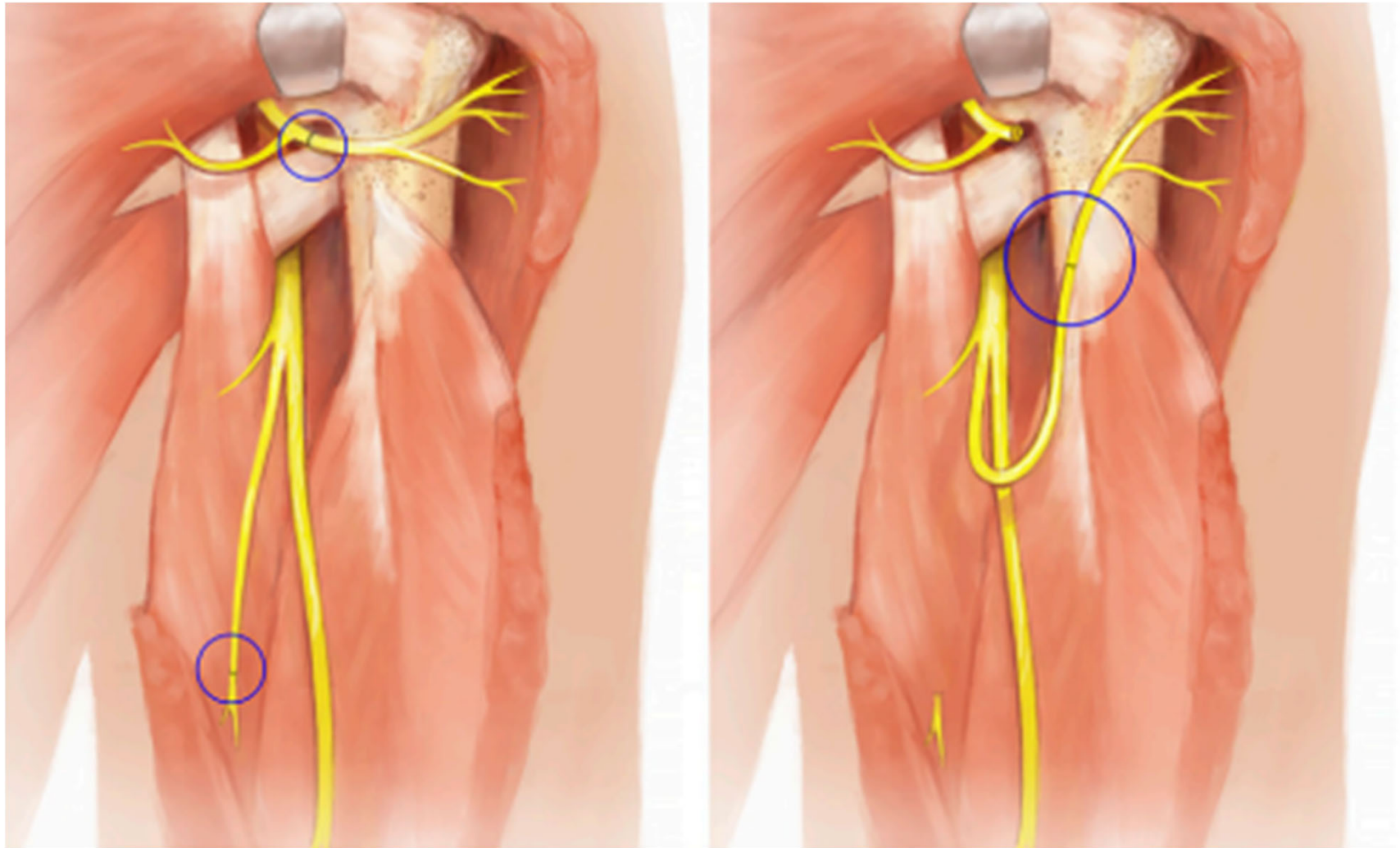


1. Posterior View
(attaching cable grafts to
distal axillary nerve stump)



2. Anterior View
(tunnelling cable grafts and attaching to proximal axillary nerve stump)

Axillary Nerve Reconstruction with Nerve Transfer



Case Example:

- 56 yo female, nurse
- Left shoulder Open Rotator Cuff Repair 4/2025
- Fall a few weeks after surgery, unable to abduct arm
- Repeat MRI, repeat left shoulder surgery 9/2025
(Revision rotator cuff repair, Acromioplasty)
- Still unable to abduct shoulder
 - Limited A/P ROM
 - Abduction strength 3/5

57 yo, 2 rotator cuff surgeries, limited shoulder Abd, EMG:

Side	Muscle	Ins Act	Fibs	Psw	Amp	Dur	Poly	Recrt	Discharges	Comment
Left	1stDorInt	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	ExtIndicis	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	ExtDigCom	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	Triceps	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	Biceps	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	PronatorTeres	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	Deltoid	Nml	Nml	Nml	1+lrg	1+lrg	0	Nml	0	
Left	Cervical Parasp Up	Nml	Nml	Nml					0	
Left	Infraspinatus	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	Supraspinatus	Nml	Nml	Nml	Nml	Nml	0	Nml	0	

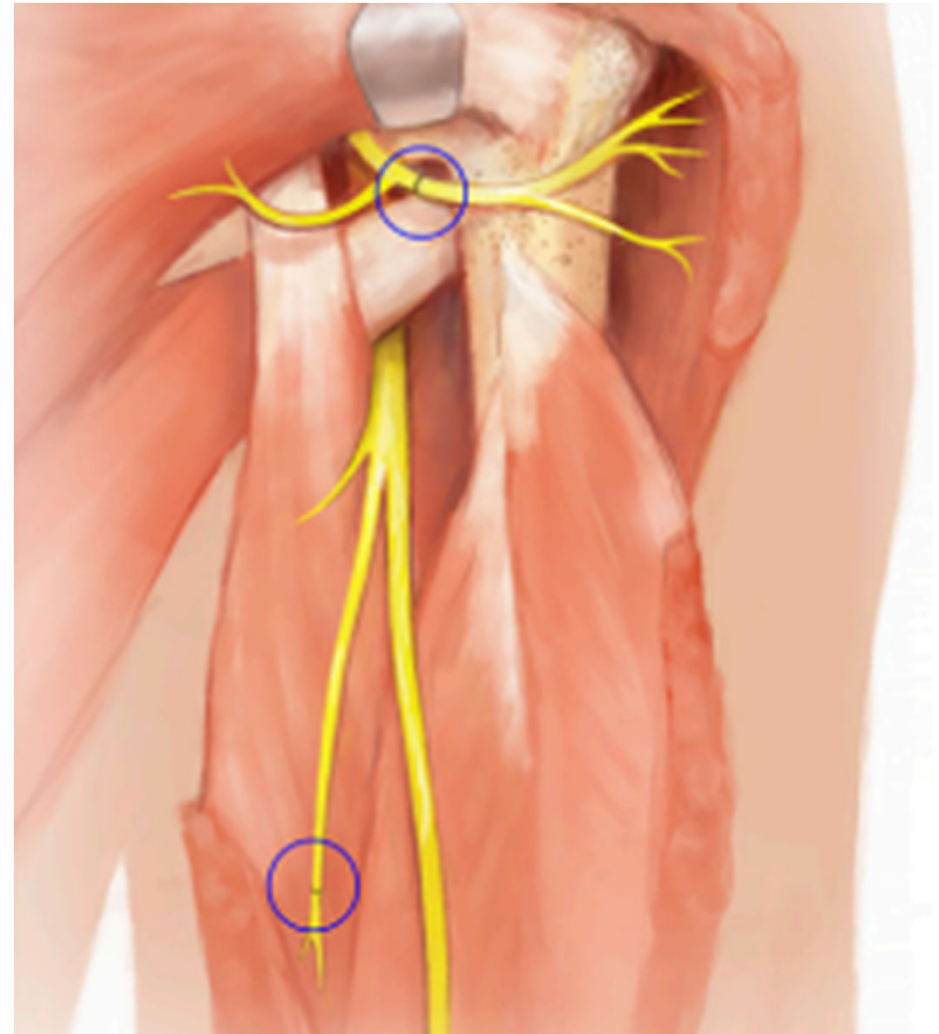
There is electrophysiologic evidence that favors a left axillary neuropathy. These findings are chronic and may also be seen with prior trauma to the deltoid muscle. There was no electrodiagnostic evidence of a median neuropathy at the left wrist, ulnar neuropathy at the left elbow, left cervical radiculopathy or left brachial plexopathy.

57 yo, 2 rotator cuff surgeries, limited shoulder Abd, EMG:

Side	Muscle	Ins Act	Fibs	Psw	Amp	Dur	Poly	Recrt	Discharges	Comment
Left	1stDorInt	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	ExtIndicis	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	ExtDigCom	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	Triceps	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	Biceps	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	PronatorTeres	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	Deltoid	Nml	Nml	Nml	1+Irg	1+Irg	0	Nml	0	
Left	Cervical Parasp Up	Nml	Nml	Nml					0	
Left	Infraspinatus	Nml	Nml	Nml	Nml	Nml	0	Nml	0	
Left	Supraspinatus	Nml	Nml	Nml	Nml	Nml	0	Nml	0	

Treatment Options ??

May Favor End To Side Nerve Transfer:

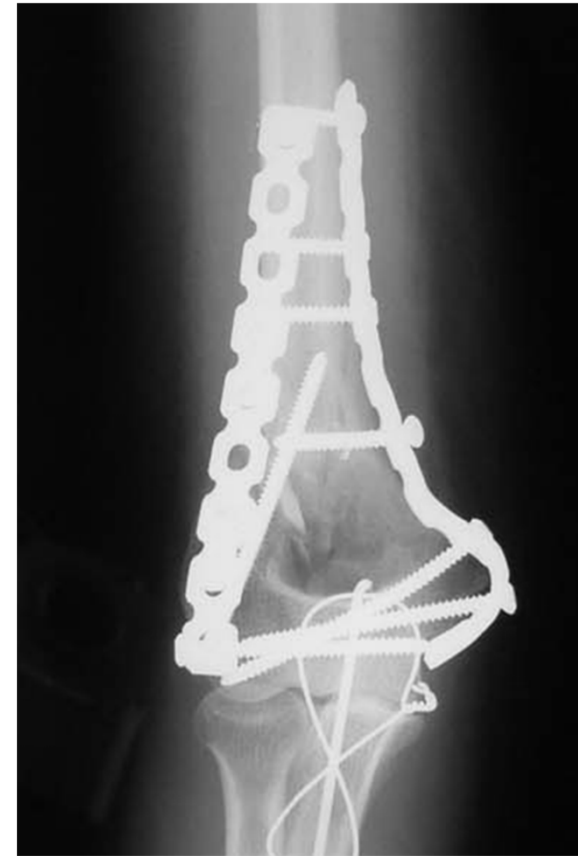


ORIGINAL ARTICLE

The Bovill Award Paper
2016 OTA Annual Meeting

In Situ Placement Versus Anterior Transposition of the Ulnar Nerve for Distal Humerus Fractures Treated With Plate Fixation: A Multicenter Randomized Controlled Trial

*Niloofar Dehghan, MD,^{a,b} Aaron Nauth, MD,^c Jeremy Hall, MD,^c
Milena Vicente, RN,^c Michael D. McKee, MD,^b and Emil H. Schemitsch, MD^d
On behalf of the Canadian Orthopaedic Trauma Society*



- 58 randomized patients: In situ vs Transposition
(45 patients had EMG testing at 6 weeks)
- Conclusions:
 - No significant difference in outcomes (Ulnar Nerve)
 - Either strategy acceptable
 - *Ulnar Nerve Symptoms are present in a large number of patients postinjury and postoperatively*
 - The majority improve by 1 year

- ***62 % of patients had abnormal EMG findings at 6 weeks***

55 % in situ (12/22)

70 % transposition (16/23)

(p = 0.30)

EMG Testing: COTS

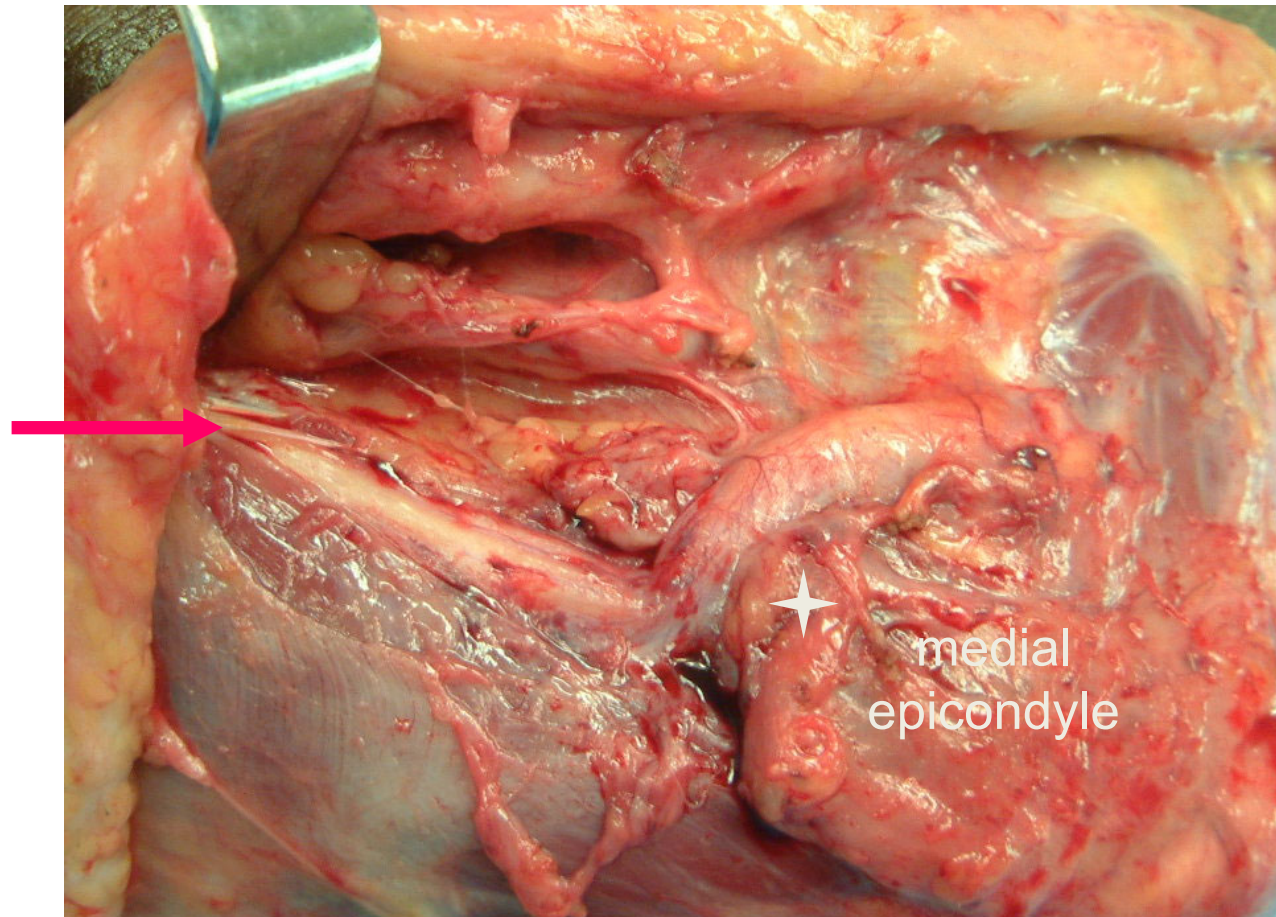
- 62 % of patients had abnormal EMG findings at 6 weeks*

TABLE 2. Results of Nerve Conduction Testing

Nerve Conduction Test Results	All Patients (N = 45)		In Situ Placement (N = 22)		Anterior Transposition (N = 23)		<i>P</i>
	N	%	N	%	N	%	
Abnormal results	28	62	12	55	16	70	0.30
Minor abnormality	15	33	6	27	9	39	0.40
Severe abnormality	12	27	6	27	6	26	0.93
Sensory only	10	22	3	14	7	30	0.17
Sensory + motor	16	36	8	36	8	35	0.91

Complications: Ulnar Nerve Injury

Ulnar Nerve
after release from
constricting band



- Ulnar neuropathy 20 % after ORIF elbow
(Range 0 % - 51%)
- Despite advances in management of elbow fractures, complications such as ulnar neuropathy continue to pose a challenge

Ulnar Nerve Management with Distal Humerus Fracture Fixation: A Meta-Analysis



Jonathan W. Shearin, MD^a, Talia R. Chapman, MD^{b,*},
Andrew Miller, MD^b, Asif M. Ilyas, MD^c

Hand Clin 34 (2018) !

- In Situ Management vs Transposition
- Meta-analysis reveals that in situ management results in less postoperative ulnar neuropathy than transposition
- A Medial Colum Plate is a risk factor for postoperative ulnar neuropathy

**Ulnar Nerve Management
with Distal Humerus
Fracture Fixation: A Meta-Analysis** 

Jonathan W. Shearin, MD^a, Talia R. Chapman, MD^{b,*},
Andrew Miller, MD^b, Asif M. Ilyas, MD^c

Hand Clin 34 (2018) !

- In Situ vs Transposition



TYPE Systematic Review
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[†]These authors share first authorship

Efficacy and safety of anterior transposition of the ulnar nerve for distal humerus fractures: A systematic review and meta-analysis

Ting Li^{1,2†}, Jingxin Yan^{3,4†}, Qiuyu Ren^{1,2†}, Jiang Hu¹, Fei Wang¹,
Chengwei Xiao^{1*} and Xilin Liu^{1*}

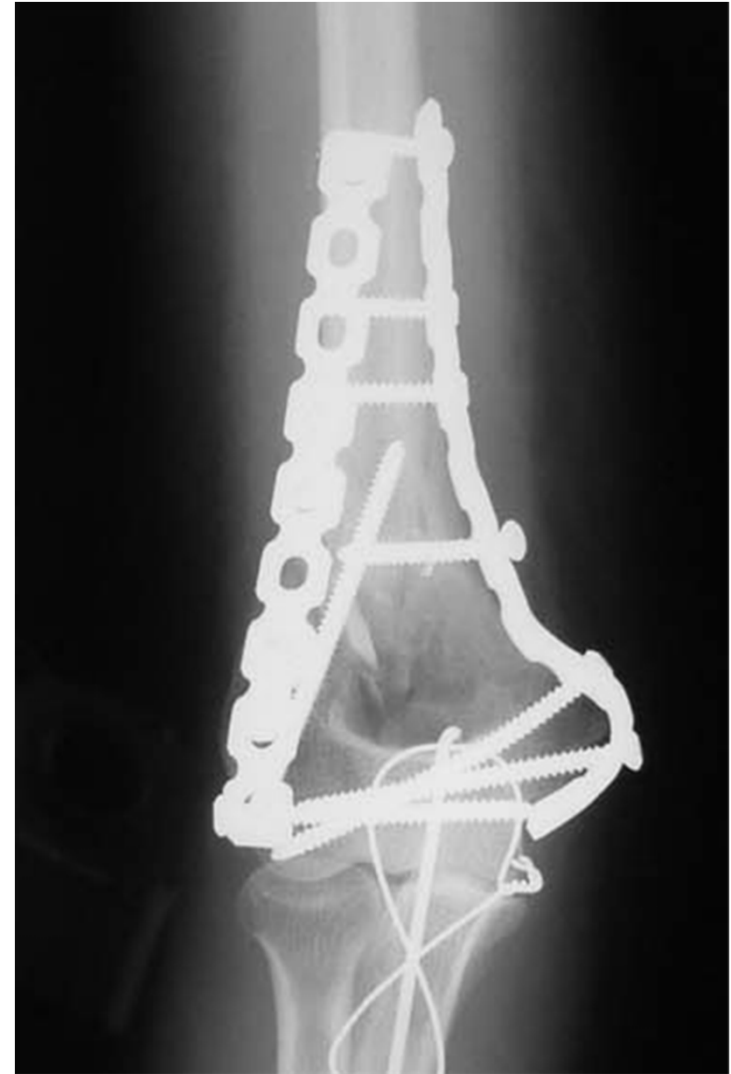
¹Department of Orthopedics, Sichuan Provincial People's Hospital, Chengdu, China, ²Department of Postgraduate, Chengdu Medical College, Chengdu, China, ³Department of Interventional Therapy, Affiliated Hospital of Qinghai University, Xining, China, ⁴Department of Postgraduate, Qinghai University, Xining, China

- Conclusions

- Anterior transposition is NOT superior to nontransposition
- *Non transposition group might decrease the incidence of postoperative ulnar neuritis after ORIF*

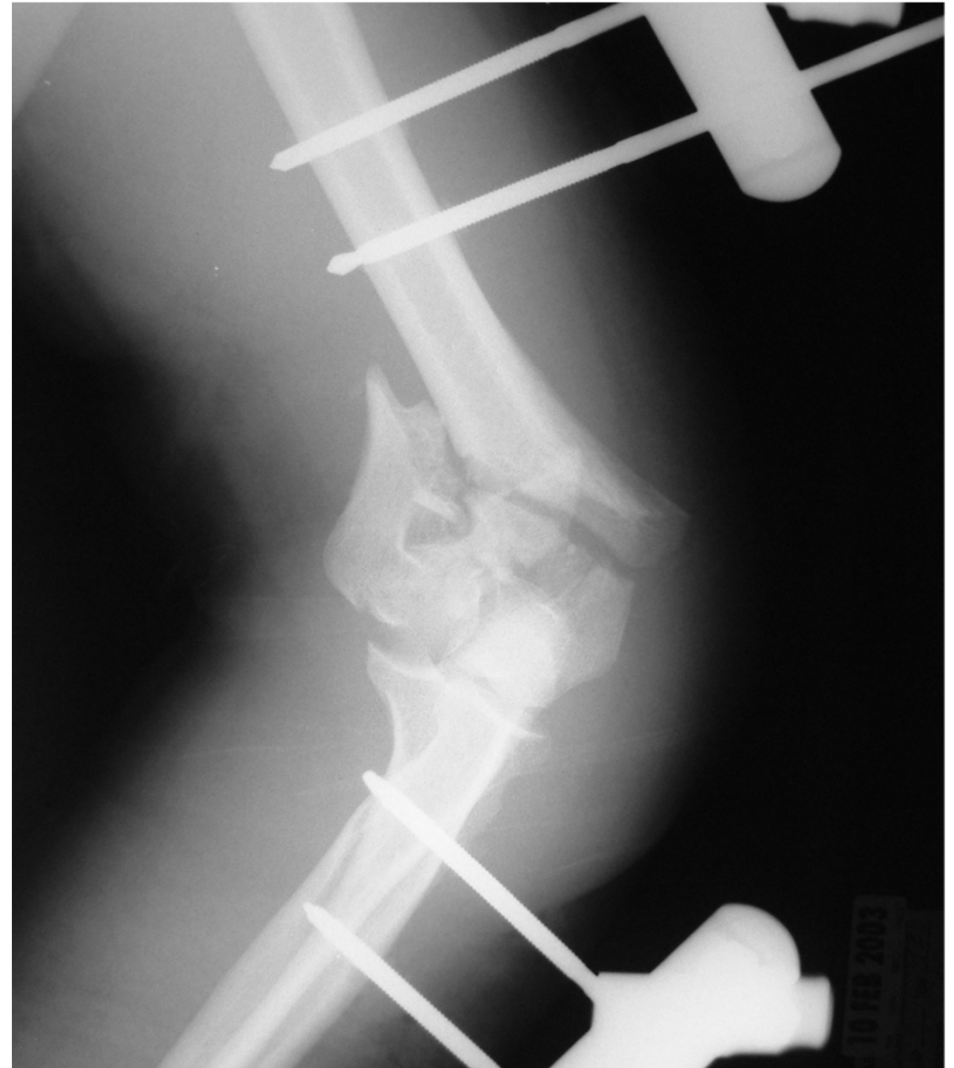
Elbow Trauma: GSW

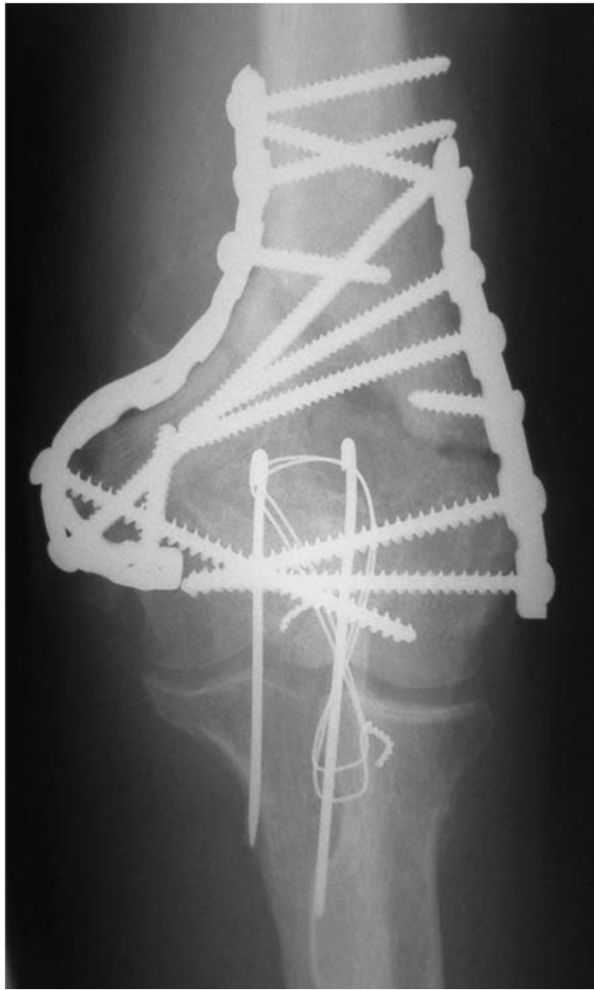


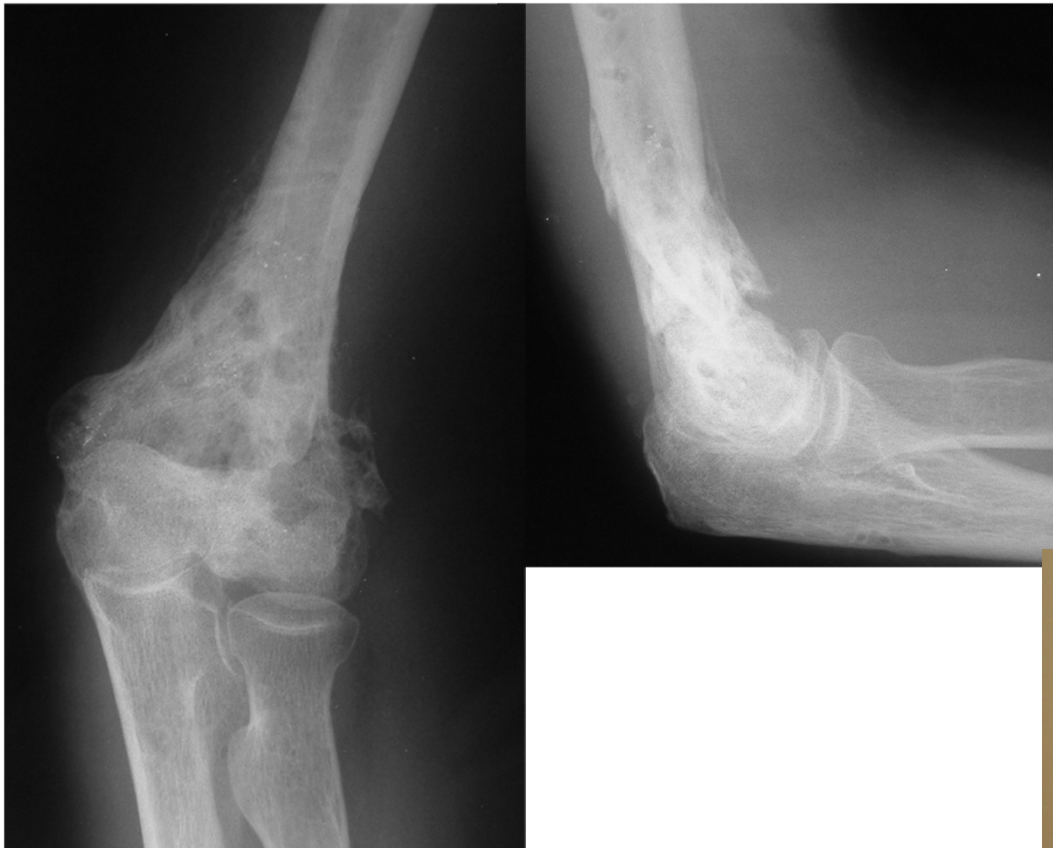


Case:

- 34 yo
- Polytrauma
 - Femur, tibia, TBI
- Open
- Radial Nerve Palsy







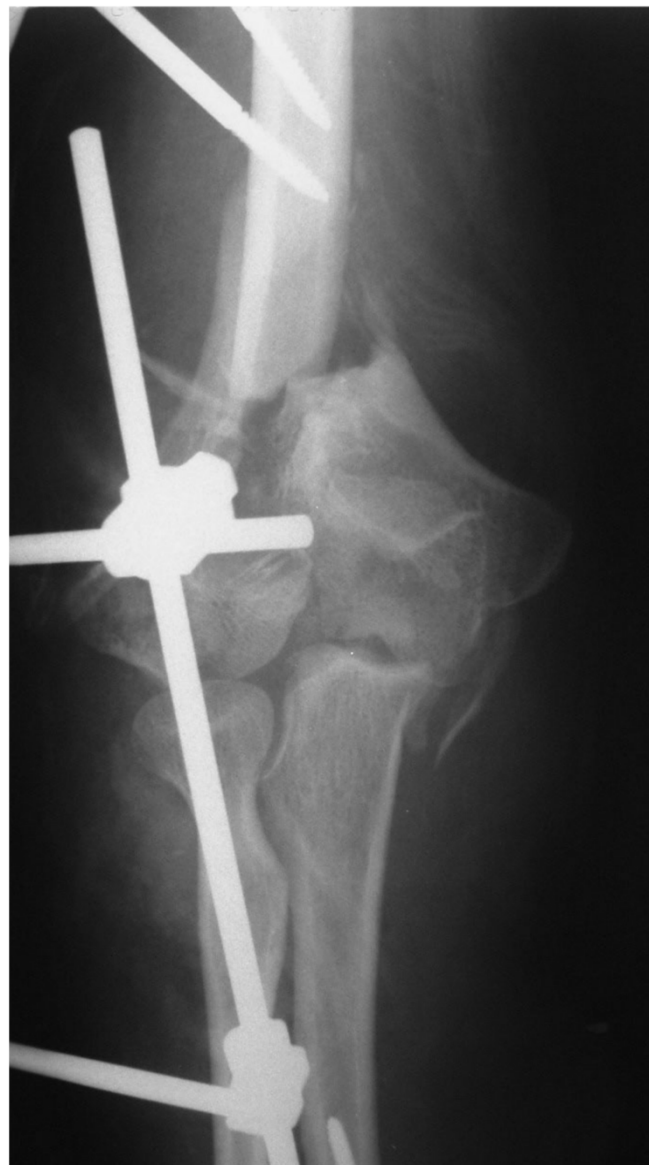
1 year post injury

Case

- 28 yo female
- MVA
- Open Fracture
- Radial Nerve Injury
- TBI

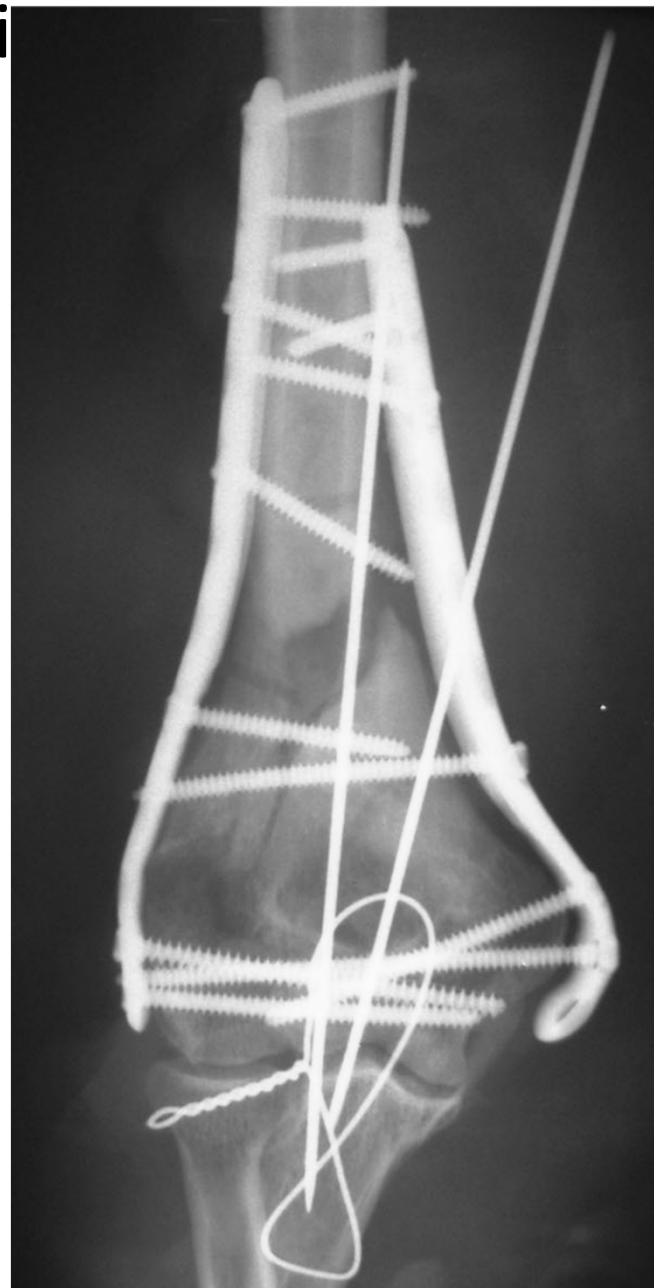
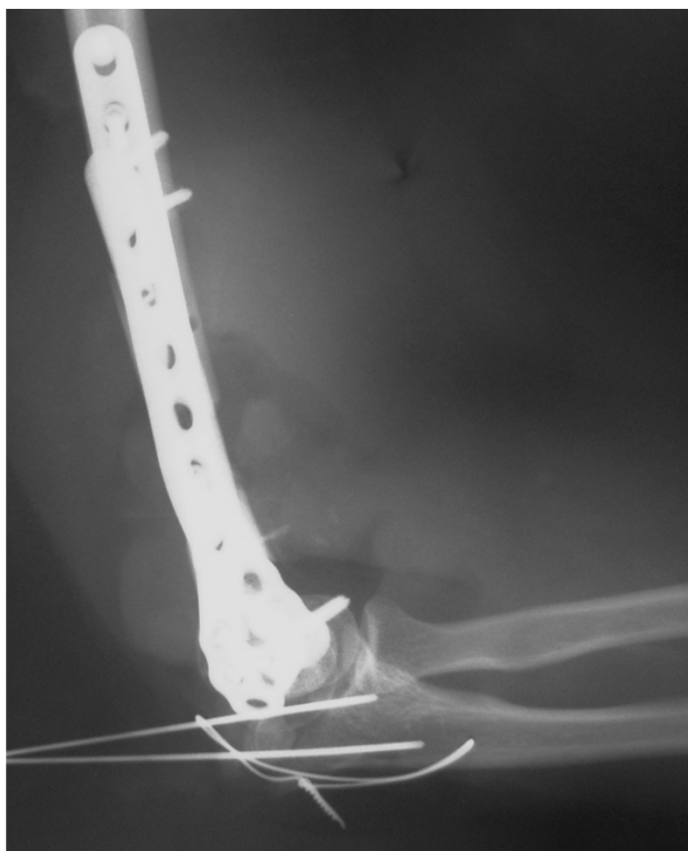


I and D, Ext Fixation

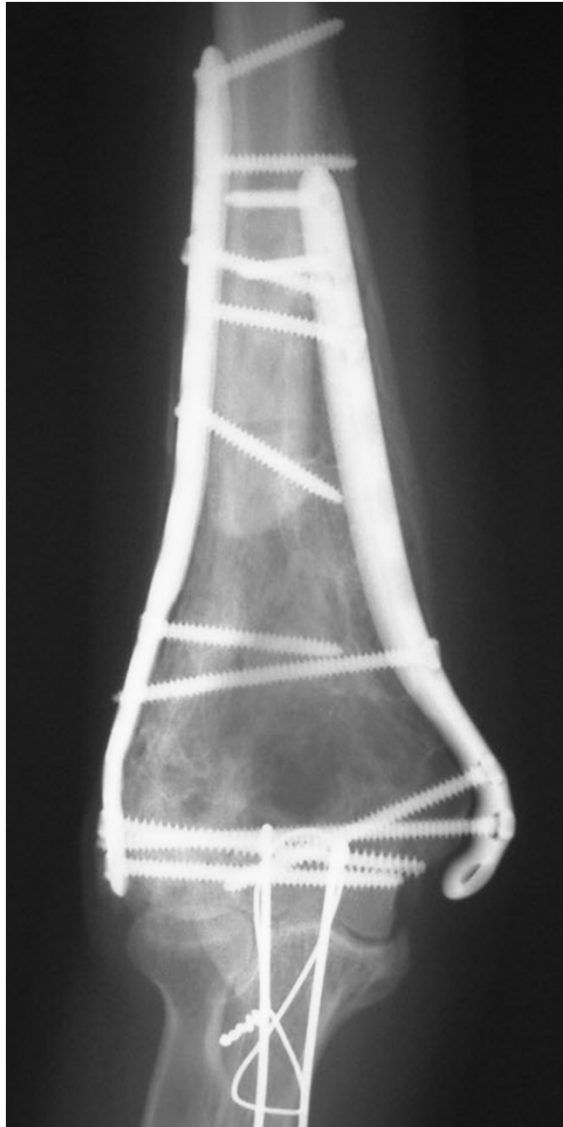


ORIF

Intraoperative



Heterotopic Ossification



Resection HO



12 month f/u



Heterotopic Ossification

Bony encasement of the ulnar nerve secondary to heterotopic ossification of the elbow: an evaluation of long-term outcomes



Consecutive series
32 elbow (27 patients)
Surgical Excision

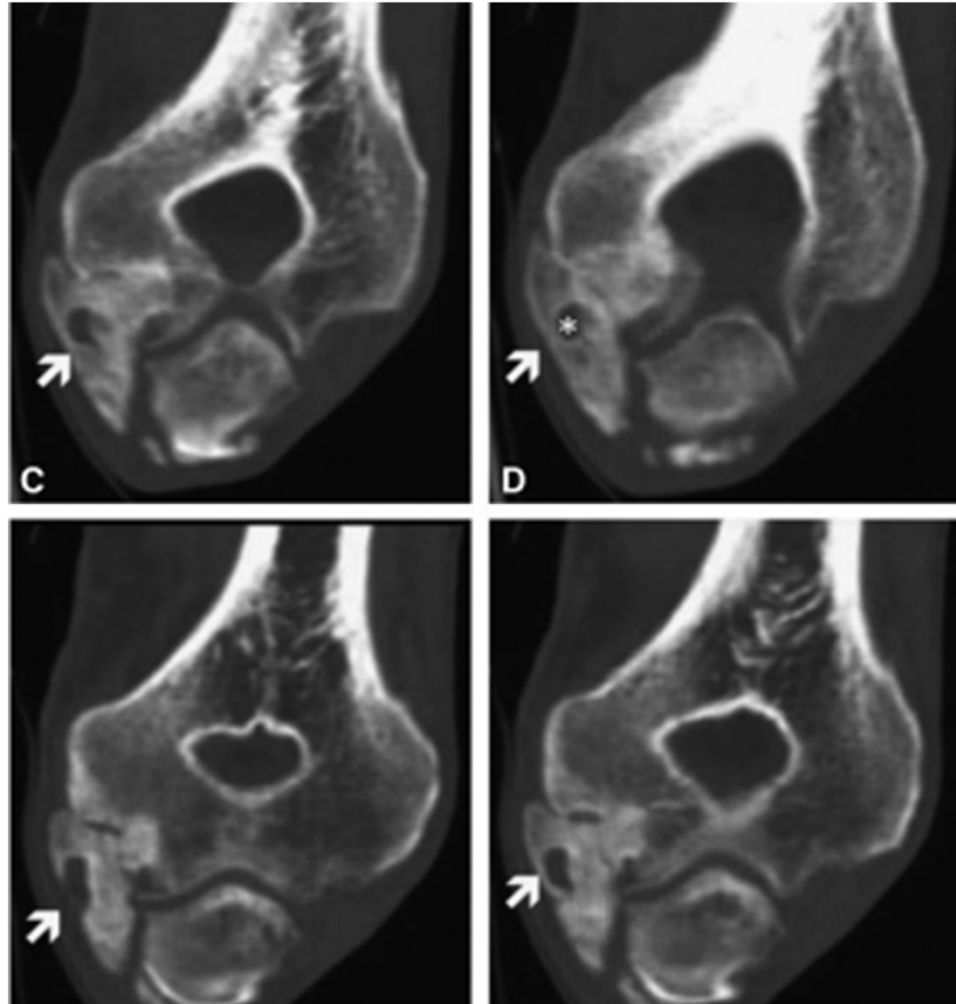
Krishin Shivdasani, MPH^{a,b,*}, Robert Kelly, BS^b, Michael Scheidt, MD^b,
Amir Boubekri, MD^b, Guido Marra, MD^c, Nickolas Garbis, MD^b,
Dane Salazar, MD, MBA^b

^aCarle Illinois College of Medicine, University of Illinois Urbana Champaign, Champaign, IL, USA

^bDepartment of Orthopaedic Surgery & Rehabilitation, Loyola University Health System, Maywood, IL, USA

^cDepartment of Orthopaedic Surgery, Northwestern University, Feinberg School of Medicine, Chicago, IL, USA

Bony Encasement: CT



The combination of:

- 1) Operative Management
- 2) PostOperative HO Prophylaxis
- 3) Regimented Rehabilitation

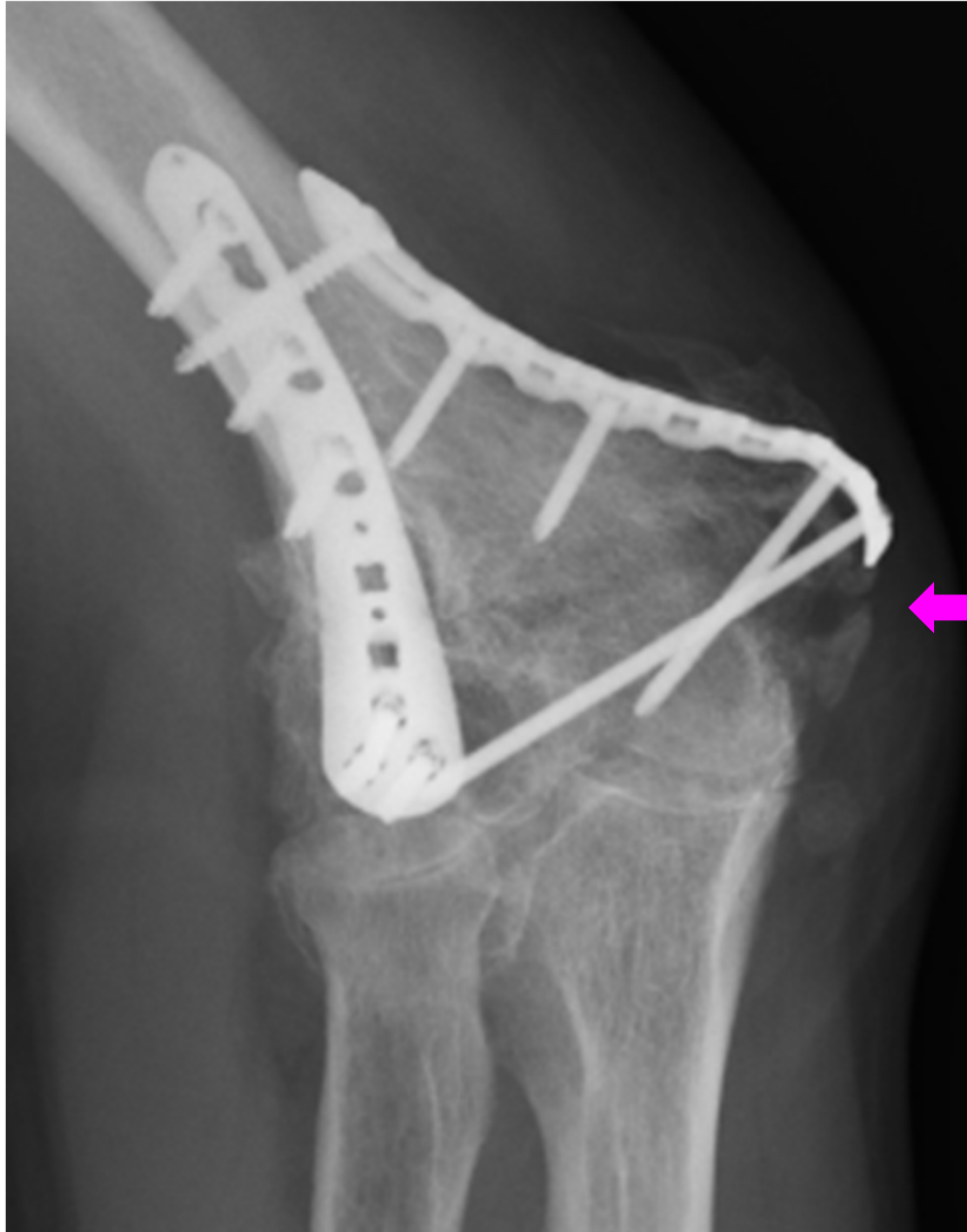
Can lead to durable, long term improvements in:

- 1) ROM
- 2) Function
- 3) Improved function of ulnar nerve
- 4) Resolution of Ulnar Neuropathy

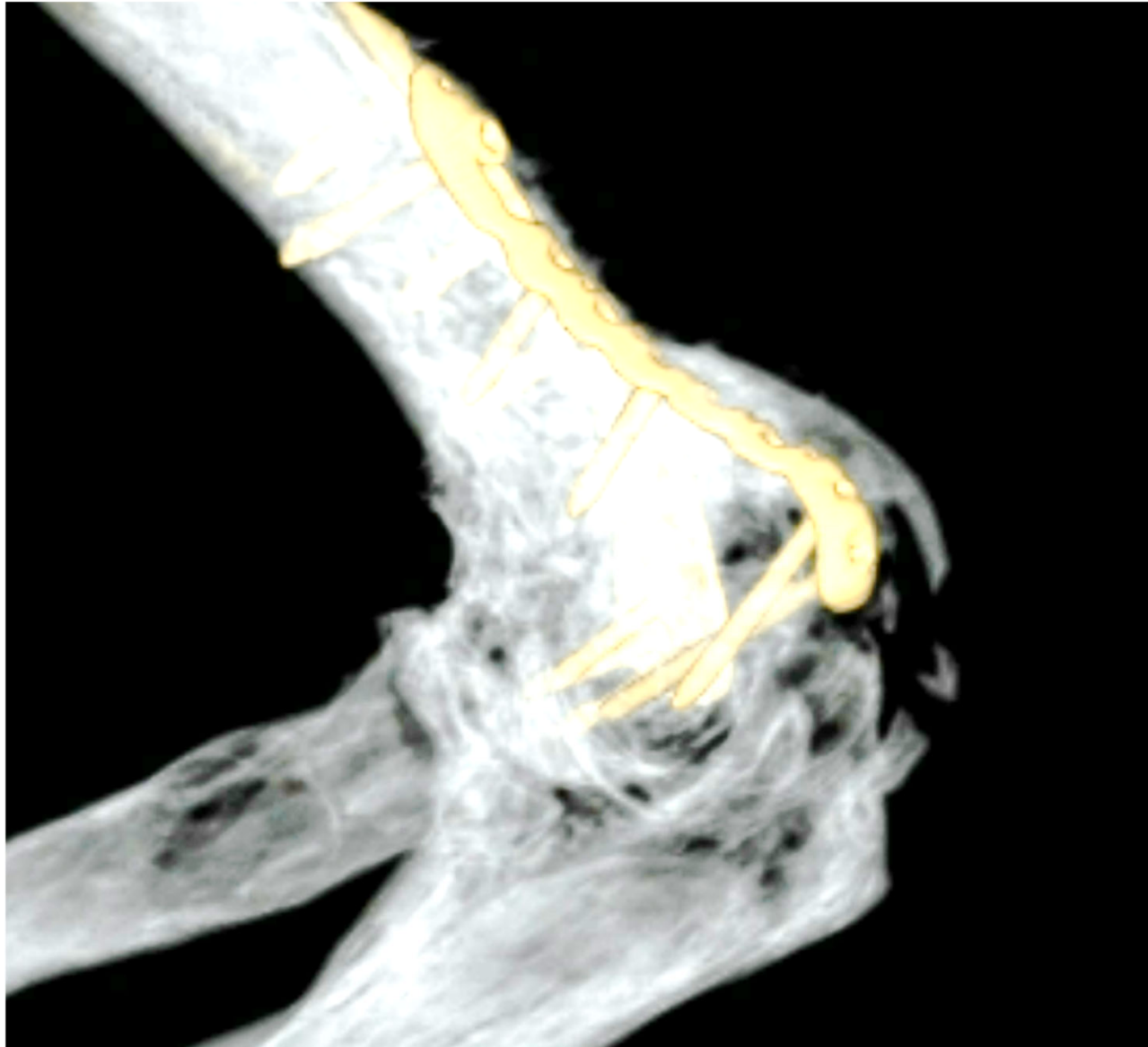
Example: Elbow HO, Ulnar Neuropathy

- GM, 64 yo, ORIF right elbow
- Ulnar neuropathy, stiff, painful, + tinels medially





CT Scan ??



EMG ??

Nerve Conduction Studies Motor Summary Table

Stim Site	N R	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Median Motor (Abd Poll Brev)											
Wrist		3.9	<4.5	8.0	>4.0	Wrist	Abd Poll Brev	3.9	8.0		
Elbow		8.1		7.2	>4.0	Elbow	Wrist	4.2	22.0	52	>50.0
Right Ulnar Seg Motor (Abd Dig Minimi)											
Wrist		3.4	<4.5	2.8	>3.2	Wrist	Abd Dig Minimi	3.4	8.0		
B Elbow		6.4		2.3	>3.2	B Elbow	Wrist	3.0	17.0	57	>50.0
A Elbow		9.2		3.6	>3.2	A Elbow	B Elbow	2.8	10.0	36	>50.0

Sensory Summary Table

Stim Site	N R	Peak (ms)	Norm Peak (ms)	O-P Amp (μV)	Norm O-P Amp	Site 1	Site2	Delta-0 (ms)	Dist (cm)
Right Median D2 Sensory (2nd Digit)									
Wrist		4.0	<3.7	23.0	>15.0	Wrist	2nd Digit	2.7	14.0
Right Ulnar Sensory (5th Digit)									
Wrist	N R		<3.7		>15.0	Wrist	5th Digit		14.0

- HO excision, Ulnar Neurolysis, Hardware Removal, Capsulectomy
- Post OP
 - Improved motion
 - Ulnar nerve improved
 - Pain Improved



- 32 yo male
- ORIF distal humerus fracture
 - Medial and Lateral Plates
 - Ulnar Nerve not Transposed
- Post Operative Paresthesias Ulnar Distribution with a little (4/5) weakness

What should be done ??

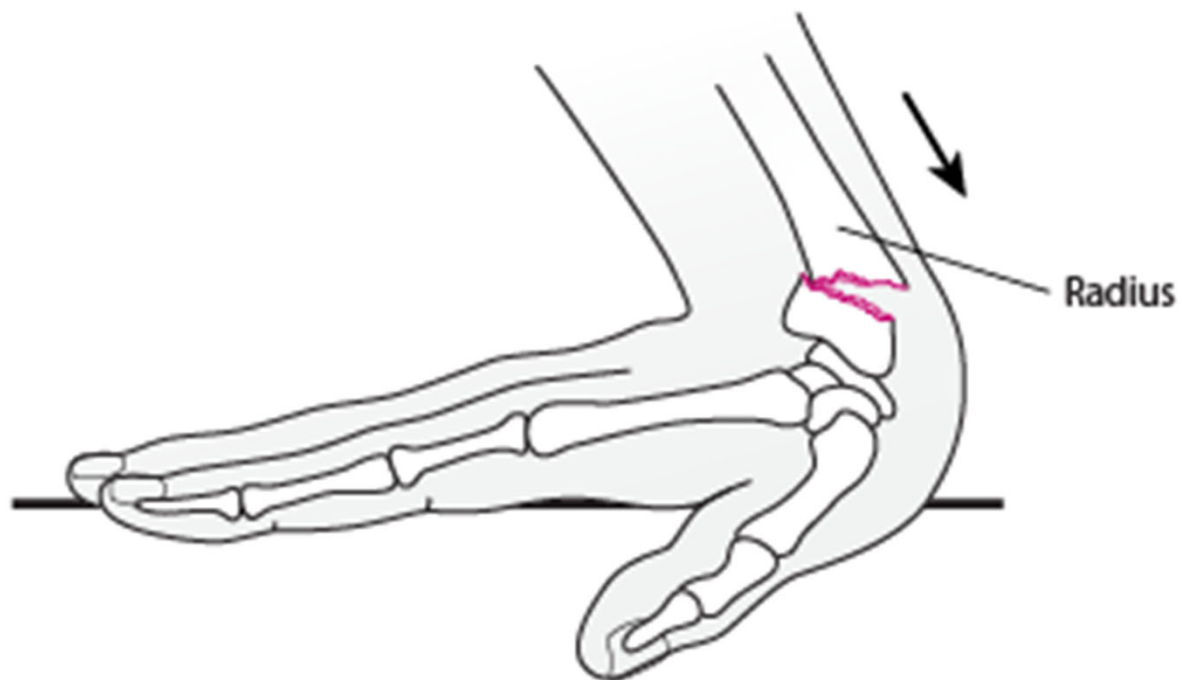
Wrist and Hand Surgery for which Post-Surgical Nerve Issues can Develop:

Wrist Fractures

Carpal Tunnel Surgery

recognition, prevention, treatment, legal aspect

Mechanism



What's happening to the Median (and ulnar) Nerve ?

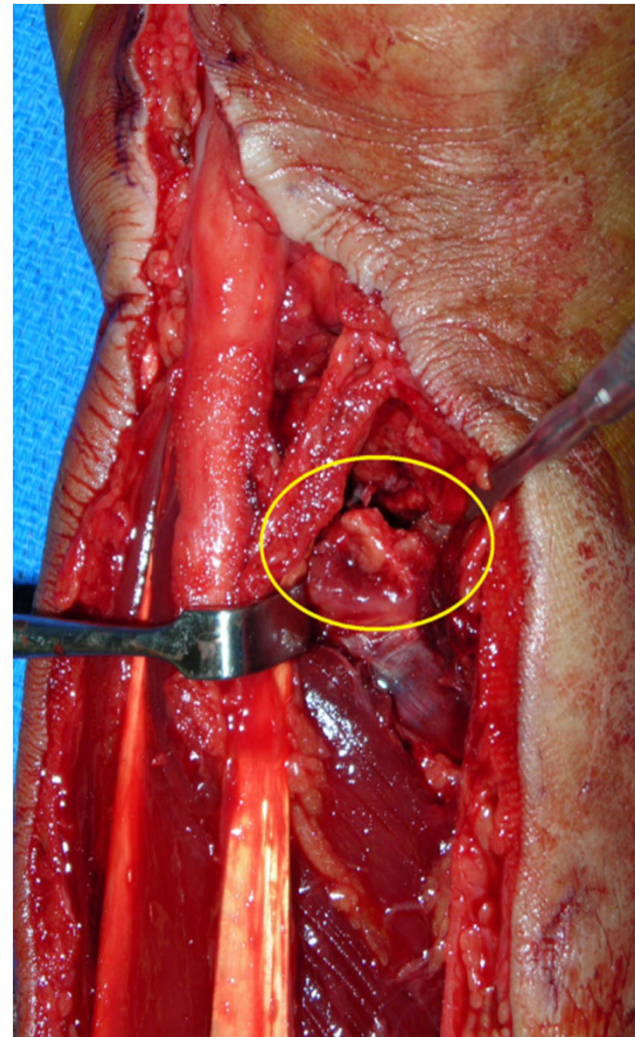
61 yo f, fall at home, diabetes



Post operative xray



CTS after distal radius fracture



Carpal Tunnel Syndrome After Distal Radius Fracture

Genghis E. Niver, MD, Asif M. Ilyas, MD*

Orthop Clin N Am 43 (2012) 521–527

- CTS and DRF are among the most common diagnoses treated by surgeons
- Their relationship is poorly understood
- CTS is a known complication of DRF
- ACUTE vs DELAYED CTS

- Nerve issues more likely due to indirect causes
 - Swelling
 - Retractors
 - Contusion (injury related)
- Direct Injury is possible
- Early intervention may be indicated

CARPAL CANAL PRESSURES AFTER VOLAR PLATING OF DISTAL RADIUS FRACTURES

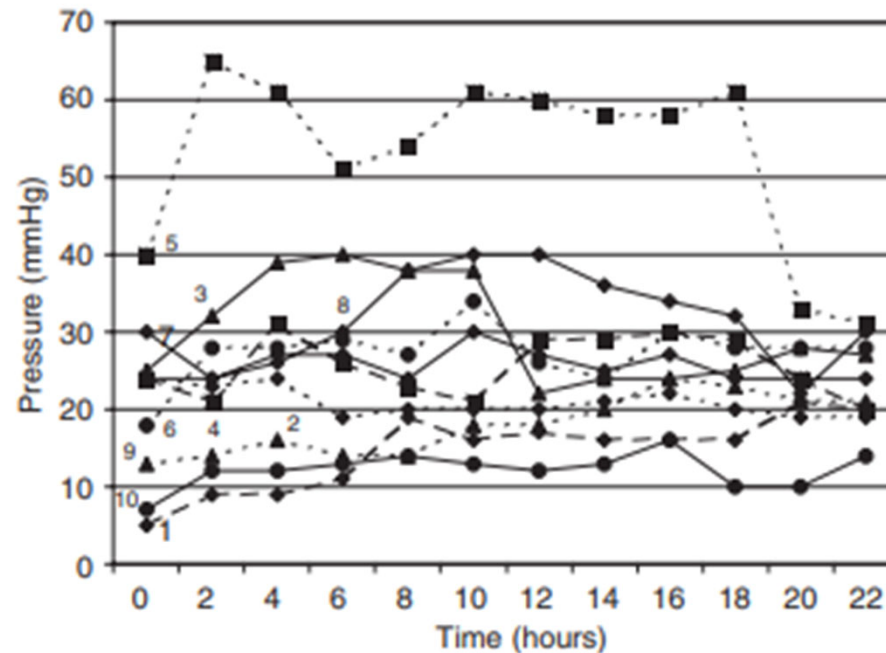
D. A. FULLER, M. BARRETT, R. K. MARBURGER and R. HIRSCH

From the Department of Surgery, Division of Orthopaedics, University of Medicine and Dentistry of New Jersey, Camden, NJ, USA

Do pressures rise in the carpal canal that put the median nerve at risk after volar plating of a distal radius fracture ?

Carpal canal pressures after volar plating of wrist fracture

10 consecutive patients, 24 hours monitoring



All pressures ≤ 31 mm Hg by 24 hours without CTR

Based upon pressure measurements,
routine prophylactic CTR does not appear necessary

- Role for Fasciotomies (compartment syndromes)
- Should we perform Prophylactic Carpal Tunnel Releases, and perhaps Guyon's canal release also, when we operate around the wrist ??

Wrist Fractures & Prophylactic CTR 2001

Prophylactic carpal tunnel decompression during buttress plating of the distal radius — is it justified?☆

O. Odumala *, C. Ayekoloye, G. Packer

Injury, Int. J. Care Injured 32 (2001) 577–579

Retrospective, Clinical Series, patients managed with volar plating
24 patients had prophylactic CTR, 45 patients did not

Prophylactic Decompressed Patients had more than twice
the relative odds (2.7x) of developing median nerve dysfunction

A wait and see attitude is better in these patients

Systematic review and pooled analysis of the rate of carpal tunnel syndrome after prophylactic carpal tunnel release in patients with a distal radius fracture

Z. Al-Amin, S.A. Senyürek, E.M.M. Van Lieshout, M.M.E. Wijffels *

Hand Surgery and Rehabilitation 37 (2018) 155–159

- PMID Review, about 2000 patients

Carpal Tunnel Release is of NO PROPHYLACTIC value for postoperative CTS in volar plated DRF patients

CTS after distal radius fracture

- Acute CTS presenting with painful paraesthesias in the hand requires urgent decompression and fixation of the fracture.
- Prophylactic release of the carpal tunnel during fracture fixation is not recommended unless the patient has a prior history of CTS or is demonstrating active median nerve paraesthesias preoperatively.
- If a concomitant CTR is to be performed, it should be performed through a separate incision or through a radial-sided hybrid FCR approach.
- Delayed presentation of CTS following a healed distal radius fracture is best treated as an idiopathic CTS, including evaluation with electrodiagnostic testing.



Carpal Tunnel Syndrome After Distal Radius Fracture

Genghis E. Niver, MD, Asif M. Ilyas,

Orthop Clin N Am 43 (2012) 521–527

Carpal Tunnel: Post Surgical Nerve Deficits

- Colleague (surgeon), 68 yo male
- Post Operative Nerve Deficit(s)



Carpal Tunnel Releases: PostSurgical Nerve Deficits

- Median
 - Complete
 - Palmar Cutaneous
 - Recurrent Motor
- Ulnar
- Risk < 1 % but can be devastating

Example: MiniOpen CTR

- 77 yo male
- History and PE consistent with bilateral CTS
- Diabetes, obesity, gout
- EMG done

Nerve Conduction Studies
Motor Summary Table

Stim Site	N R	Onset (ms)	Norm Onset (ms)	O-P Amp (mV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Median Motor (Abd Poll Brev)											
Wrist		7.5	<4.5	3.2	>4.0	Wrist	Abd Poll Brev	7.5	8.0		
Elbow		12.2		2.5	>4.0	Elbow	Wrist	4.7	22.0	47	>50.0
Left Median Motor (Abd Poll Brev)											
Wrist		5.2	<4.5	5.6	>4.0	Wrist	Abd Poll Brev	5.2	8.0		
Elbow		9.5		5.1	>4.0	Elbow	Wrist	4.3	22.0	51	>50.0

Sensory Summary Table

Stim Site	N R	Peak (ms)	Norm Peak (ms)	O-P Amp (μV)	Norm O-P Amp	Site 1	Site2	Delta-0 (ms)	Dist (cm)
Left Median D2 Sensory (2nd Digit)									
Wrist		4.7	<3.7	6.0	>15.0	Wrist	2nd Digit	3.5	14.0
Right Median D2 Sensory (2nd Digit)									
Wrist		4.8	<3.7	4.3	>15.0	Wrist	2nd Digit	3.7	14.0

EMG

Sid e	Muscle	Nerve	Root	Ins Act	Fib s	Ps w	Am p	Du r	Pol y	Rec rt	Int Pat	Comme nt
Rig ht	Deltoid	Axillary	C5-6	Nml	1+	2+	Nml	Nm l	Nm l	Nml	Comp	
Rig ht	Biceps	Musculoc ut	C5-6	Nml	2+	2+	Nml	Nm l	Nm l	Nml	Comp	
Rig ht	Triceps	Radial	C6-7-8	Nml	0	0	Nml	Nm l	Nm l	Nml	Comp	
Rig ht	PronatorTeres	Median	C6-7	Nml	1+	1+	Nml	Nm l	Nm l	Nml	Comp	
Rig ht	FlexCarpiUln	Ulnar	C8,T1	Nml	0	0	Nml	Nm l	Nm l	Nml	Comp	
Rig ht	1stDorInt	Ulnar	C8-T1	Nml	0	0	Nml	Nm l	Nm l	Nml	Comp	
Rig ht	Abd Poll Brev	Median	C8-T1	Nml	2+	2+	Nml	Nm l	Nm l	Nml	Comp	
Left	Deltoid	Axillary	C5-6	Nml	2+	2+	Nml	Nm l	Nm l	Nml	Comp	
Left	Biceps	Musculoc ut	C5-6	Nml	1+	2+	Nml	Nm l	Nm l	Nml	Comp	
Left	Triceps	Radial	C6-7-8	Nml	0	0	Nml	Nm l	Nm l	Nml	Comp	
Left	PronatorTeres	Median	C6-7	Nml	0	0	Nml	Nm l	Nm l	Nml	Comp	
Left	FlexCarpiUln	Ulnar	C8,T1	Nml	0	0	Nml	Nm l	Nm l	Nml	Comp	
Left	1stDorInt	Ulnar	C8-T1	Nml	2+	2+	Nml	Nm l	Nm l	Nml	Comp	
Left	Abd Poll Brev	Median	C8-T1	Nml	2+	2+	Nml	Nm l	Nm l	Nml	Comp	
Rig ht	CervParasp Middle	Rami		Nml	2+	2+						
Left	CervParasp Middle	Rami		Nml	2+	2+						

There is electrodiagnostic evidence of a bilateral C5-6 and a left sided C8 cervical radiculopathy.

There is electrodiagnostic evidence of a bilateral moderate-severe primarily demyelinating median neuropathy at the wrist consistent with a clinical diagnosis of a bilateral moderate-severe carpal tunnel syndrome (right worse than left).

- Undergoes Right CTR
- 6 weeks post op: *“right hand is more numb than before surgery & thumb is completely numb”*
- Next Steps ?
 - PE
 - Surgical Exploration, repeat EMG, MRI, U/S ?
 - Other

Open Vs Endoscopic CTR: Risks

Open versus Endoscopic Carpal Tunnel Release: A Meta-analysis of Randomized Controlled Trials

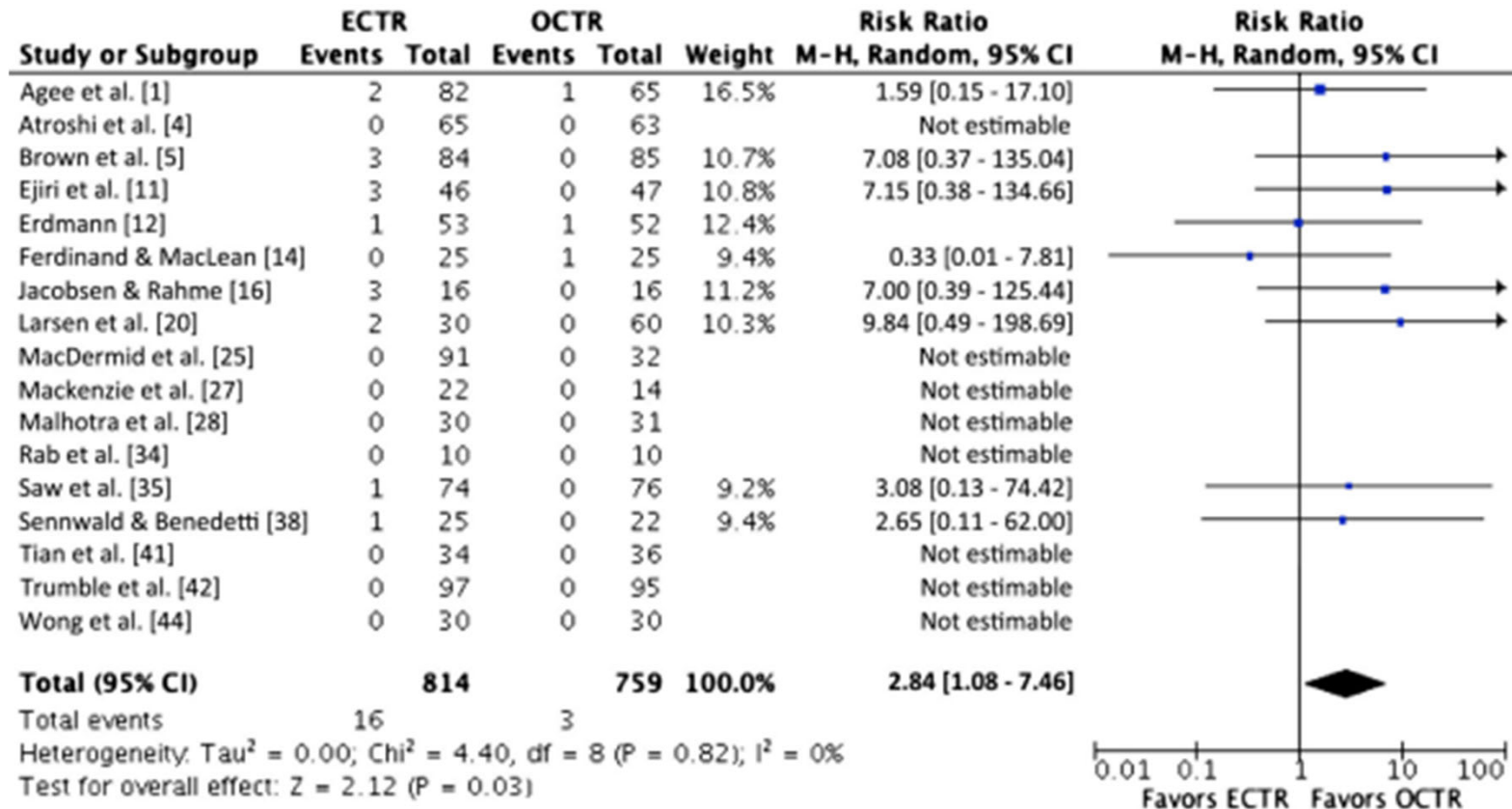
Eli T. Sayegh BS, Robert J. Strauch MD

Clin Orthop Relat Res (2015) 473:1120–1132

The Relative Risk of a Nerve Injury favors Open CTR over Endoscopic CTR

The RR of Nerve Injury = 2.84 (eCTR v OCTR)

Nerve Injury: eCTR v OCTR



Forest Plot Risk Ratio for Nerve Injury

Reawakening Neuritis of the Median Nerve after Carpal Tunnel Release: Defining and Predicting Patients at Risk

3.9% of patients undergoing CTR

Plastic and Reconstructive Surgery • July 2022

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Reawakening Neuritis after CTR: Risk Factors

- Advanced Age
- Evidence of APB atrophy on EMG
- Longer duration of symptoms

Recommendation: Preoperative Counseling

- Discussed Incidence of Intraoperative Nerve Injuries with Upper Extremity Surgeries
- Discussed Diagnosis and Prevention Strategies
- Discussed Treatment Strategies for Such Nerve Issues

Reminder



But Hector, in his shining helm, hit him with the barren stone at the shoulder, where the clavicle divides the neck from the chest, a most opportune point, and broke the nerve.

The arm fell numb to the wrist, he (Teucus) fell on his knees and lay still, the bow fallen from his hand.

Homer's Iliad
- 8th century BC

Tangled Up in Blue



Keep on Keeping on

Conclusion: Core Values

Service-Oriented

Because we value people above all else, we serve each other, our patients and clients, our communities, and the world.



Rocky Mountain
University
of Health Professions

Thank you



Cooper Medical School
of Rowan University

Question 1:

Intraoperative Neuromonitoring for Spinal Surgery:

- 1) is required for spine surgery
- 2) is always done using only somatosensory and motor evoked potentials
- 3) reduces the risk of malpractice litigation
- 4) lacks consensus and conclusive evidence for routine usage
- 5) is reimbursed as part of the procedural fee

Question 2:

A 68 year old female undergoes a total shoulder arthroplasty (TSA). Which of the following is true:

- 1) TSA is routinely performed with intraoperative neuromonitoring
- 2) TSA has a lower nerve injury rate than knee or hip arthroplasty surgery
- 3) Preoperative shoulder stiffness is associated with greater risk of nerve injury for patients undergoing TSA
- 4) Electrodiagnostic studies reveal nerve trauma to be rare during TSA
- 5) The Ulnar nerve is the most commonly injured nerve associated with TSA

Question 3:

A 32 year old male undergoes surgical fixation of a distal humerus fracture with medial and lateral plates. Which of the following is true:

- 1) The lateral plate is most likely to cause ulnar neuritis
- 2) An anterior transposition is associated with lower risk of ulnar neuritis
- 3) He has a $> 50\%$ chance of having abnormal EMG findings in his ulnar nerve 6 weeks after surgery
- 4) If he develops clinically symptomatic ulnar nerve symptoms after surgery, it should always be observed for 12 months
- 5) Heterotopic bone will not encase an ulnar nerve after elbow trauma

Question 4:

A 68 year old female falls from a standing height and breaks her wrist. She undergoes volar plating of her distal radius fracture. Which of the following is true:

- 1) There is no association between distal radius fractures and the development of Carpal Tunnel Syndrome
- 2) Carpal canal pressures do not change after volar plating of distal radius fractures
- 3) Prophylactic carpal tunnel release is indicated when a distal radius fracture is treated surgically
- 4) Acute carpal tunnel syndrome can develop and requires urgent decompression
- 5) Median nerve dysfunction occurs with twice the frequency if she does not undergo a prophylactic carpal tunnel release

Question 5:

A 68 year old male undergoes a mini-open carpal tunnel release for idiopathic carpal tunnel syndrome. His surgical incision is about 1 inch long. Which of the following is true:

- 1) There is no risk of intraoperative nerve injury because the surgery was done in an open fashion
- 2) If the surgery had been done endoscopically, his relative risk of nerve injury would have been higher
- 3) All nerve injuries after a carpal tunnel release require immediate exploration
- 4) EMG/NCS are of no value in a post surgical nerve injury after a carpal tunnel release
- 5) Carpal tunnel release should not be done endoscopically