“The Angry Mother-in-law”

Peripheral Neuropathic Syndromes in Orthopedic & Sports Medicine Patients: (Upper Quarter)

International Manual Therapy Seminars
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Course Description

Course 1A.: The Upper Quarter Pain Puzzle:

Peripheral Neuropathic Syndromes in Orthopedic & Sports Medicine Patients: (Upper Quarter)

This one-day lab course will present concepts of differential diagnosis and treatment of patients with upper extremity radicular symptoms pioneered by Bob Elvey P. T., GDMT. Emphasis will be placed on precise physical evaluation of neural, articular, and muscular tissues in order to allow for specific treatment of each. This precision evaluation allows for differential diagnosis of upper arm and arm pain syndromes with regard to the cervical spine, thoracic outlet, glenohumeral joint and peripheral nerves. Treatment of pain syndromes of neural origin will be explained and demonstrated along the lines of neural tissue bio-mechanics and physiological concepts. This course will cover well-recognized disorders commonly encountered by the physical and occupational therapist. Such topics as "frozen shoulder," CRPS, carpal tunnel, thoracic outlet, and other peripheral neuropathies and their treatment will be discussed. Neurogenic pain syndromes, associated neuropathic and neurogenic pain syndromes, including a review of new research into the treatment of CRPS I and CRPS II, will also be presented. Real patient videos, case presentation, and lab practicals will be used to further allow for application of the material into the participant's daily practice.
Who is Bob Elvey?

Bob is an internationally known clinician and lecturer from South Perth, Western Australia, who pioneered and developed evaluation and treatment techniques in the field of “Neural Mobilization”. He developed and researched upper quarter neural dysfunction’s and published his first articles on his innovative “Upper Limb Tension Tests (U.L.T.T.).” in the late 70’s. His research and innovative evaluation and treatment techniques have led to the worldwide development of treatments and research into neurogenic pain syndromes. Bob retired after many years in private practice and was coordinator and senior lecturer of the postgraduate manipulative therapy program at Curtin University in Perth Australia. Mr Elvey is a past president of the International Federation of Orthopaedic Manipulative Therapists and has received the prestigious lifetime achievement award. He is also past president of the Manipulative Physiotherapists Association of Australia. Bob Elvey has written and contributed to numerous articles and books on the subject of Neurogenic Pain Syndrome. He has taught his “Physical Evaluation and Treatment of Neural Tissues in Disorders of the Neuromusculoskeletal System” all over the world and has influenced countless numbers of Manual Therapists in the pursuit of excellence. Bob passed away in April of 2013. He left a legacy of compassion, excellence, and a never ending pursuit of knowledge. This presentation is a tribute to his spirit and his work.

About Our Instructor:

Jack M. Stagge P.T., OC.S.,F.A.A.O.M.P.T

Received his bachelor's degree in Physical Therapy from CSU Fresno in 1978. He received a specialty certification in Adult Neurology from U.S.C. in 1979. His postgraduate training includes extensive manual therapy course work in Norwegian, Australian, McKenzie and Cyriax protocols. He was chosen as a subject matter expert by the American Physical Therapy Board of Specialties in 1988 and helped in writing the first O.C.S. exam. He was Board Certified as an Orthopedic Clinical Specialist in May 1990 and 1999, and became a Fellow of the A.A.O.M.P.T. in 2002. Mr. Stagge taught “Physical Evaluation and Treatment of Neural Tissues in Disorders of the Neuromusculoskeletal System” with Mr. Robert L. Elvey, P.T., G.D.M.T. of Curtin University, Perth, Australia for several years throughout the United States. Mr. Stagge has been a featured presenter at the World Confederation for Physical Therapy, the APTA’s National Scientific meeting, Combined Section Meetings, State Conferences, and the I.F.O.M.P.T. in Perth Australia. He has served as a visiting professor and lecturer at several graduate programs and is a member of the International Teachers’ Forum of I.F.O.M.P.T. Mr. Stagge has also served as a book reviewer for the Journal of Orthopedic and Sports P.T., and as a consultant on several articles published on orthopedic manual therapy. Mr. Stagge is currently director of International Manual Therapy Seminars and continues to consult and teach in the area of neurogenic pain syndromes.
Learning Objectives

1. Participants will gain an understanding of the mechanisms by which neural tissue may in itself be a pain source.

2. Participants will gain an understanding of how neural tissue, when it is a pain source, may cause wide spread effects.

3. Participants will gain knowledge of neural tissue dynamics.

4. Participants will gain an understanding of how dysfunction may occur when Normal neural tissue dynamics is prevented by pain.

5. Participants will gain knowledge of physical examination techniques of neural tissue for its dynamic and functional compliance.

6. Participants will gain knowledge of the role neural tissue may play in conditions commonly evaluated and treated by physical therapists and manual medicine practitioners. This knowledge will allow for specificity of diagnosis and prevent treatment errors.

7. Participants will gain knowledge of physical treatment techniques directed towards neural tissues.

8. Participants will be presented with postulations as to the remedial effect of physical treatment techniques of neural tissues.

9. Participants will gain an understanding for caution in implying that neural tissue is the cause of a disorder and in the treatment of neural tissue.

10. Participants will gain knowledge of differential diagnostic techniques in neuromusculoskeletal disorders.

11. Participants will be able to differentially rule in or out neurogenic syndromes as either the cause or the central driver of diagnostic findings.
Schedule

8:30....Introduction
   Patient profiles

8:45....Orthodiagnosis Types

9:00....Term definition
   Compression vs. P.N.S.

9:15....Pathophysiology
   Research

9:30....Subjective Evaluation

9:45....Objective Evaluation
   DUCK
   Posture
   Active Testing

10:00....Break

10:15....Demo of Active Testing

10:30....Lab on Active Testing

11:00....Passive Testing

11:15....Demo of Passive Testing

11:30....Lab on Passive Testing

12:00....Lunch

1:00.....Neuropalpation

1:15.....Neuropalpation Demo

1:30.....Lab on Neuropalpation

2:00....Tender Points/Specific Signs of Local Dysfunction

2:15....Demo Trigger Point or Tender Point Palpation/Specific Signs of Local Dysfunction

2:30.... Putting it altogether.

2:45....Ortho and Sports Med Patient Presentation
3:00....Break
3:15....Treatment Demo
3:30....Lab on Treatment
4:00....Home Programs
4:15....Treatment Sequencing...
4:30....Further Considerations/Conclusion
4:45....End of Course
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Patient Profile

Subjective: Primary complaint of chronic pain and decrease function.
“Heaviness” and “weakness” reported without objective verification.

Objective: Often Negative MRI. Neg. Nerve Conduction Velocities. Active ROM often evaluated as WNL’s unless neural provocation positions are added or combined.

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E.M.G Findings

"E. M. G. will probably be negative if nerve is inflamed rather than compressed. Must lose 80% of conduction channels before tests are definitive."

Haldeman S., SPINE Vol. 9, No. 1, 1984
Sunderland S., Nerve and Nerve Injury, 1977
Yoshizawa, H. SPINE Vol. 20. No. 4, 1995

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Slide 6

Most patient’s have normally seen several practitioners some labeled as “Chronic Pain Personalities”. Patient’s with neural irritability many times report worsening symptoms with use of decompressive techniques such as traction or with patterned exercise such as PNF patterning of the lower extremity.

Patient’s who have had decompressive surgeries and had both compression and irritability prior to surgery may display decrease of compressive signs with continued pain complaints.
Athletes may have failed interventions directed to distal or protective tissues when neural irritability is the primary pain or movement driver. Noted functional loss and inability to correct normal patterning despite elite training.

Research throughout the world is revealing that 17-37% of chronic pain patients “may have a neuropathic pain causation”


Upper Quarter “Orthopedic” Patient Diagnosis That May Have Neuropenic Causation or in whom symptoms are maintained by Neuropenic Input.

- Adhesive Capsulitis - “Frozen Shoulder”
  - Lateral Epicondylitis
  - “Whiplash Syndrome”
  - DeQuervain Tenosynovitis
  - Failed Cervical Decompression
- Tunnel Syndromes
  - “Pseudo” Sympathetic Reflex Dystrophy
  - Levator Scapular Syndrome
  - Shoulder Impingement Syndrome
  - Fibromyalgia TRP’s
  - Monarticular Arthritis
**Slide 10**

Objective of Orthopedic Manual Evaluation

Determine dysfunction and relate to subjective examination—Diagnosis:

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Areas That Must Be Considered In Evaluation

1. Motion segment
2. Neural tissue
3. Muscle and soft tissue
4. Functional Movement Patterns
5. Emotional aspects

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**Slide 12**

[Image of a diagram or chart, possibly related to orthopedic manual evaluation.]
Definitions and Terms in Neuropathic Syndromes

For the sake of this presentation:
Neuropathic Pain = CRPS I = Peripheral Nerve Pain = Peripheral Nerve Sensitization

AKA:: Angry Mother-in-Law


Schafier, A., Interrater Reliability of a New Classification System for Patients with Low Back Related Leg Pain. JMMT 2009: 17(2) pgs 109-117

Peripheral Nerve Sensitization (PNS):
"Sensitization arising from nerve trunk inflammation causing increased axonal mechanosensitivity with absent significant denervation."

Elow EJ., Inflammation with no axonal damage of the rat saphenous nerve trunk induces ectopic discharge and mechanosensitivity in myelinated axons. Neurosci Letters 2003; 333(1): 49-52

### NeuPSIG Guidelines  
**IASP 2011**

**“Common Denominators” that are found in Neuropathic Pain**

1. Area of symptoms fits the distribution of a nerve  
2. Quantitative—hyper or hypoesthesia  
3. Qualitative—alodynia or dyesthesias  
4. Temporal—after sensation, summation.

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### Neuropathological Definitions by Treatment Categories  
(Instructors Definition Only)

- **1. Compressive**
- **2. Inflammatory AKA... P.N.S or Peripheral Nerve Pain**
- **3. Combinations**

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### Compressive Loss of Neural Function

- Example: Bony or soft tissue stenosis, tunnel syndromes, scar tissue “strangulation”, HNP sequestration, foreign bodies, etc.
Comparison of Compressive versus Irritative Evaluation Findings

<table>
<thead>
<tr>
<th>Compressive</th>
<th>Irritative-Inflammatory</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Loss of conductivity)</td>
<td>(Pseudoneuroma, Central Sens.)</td>
</tr>
<tr>
<td>– Loss of Reflex</td>
<td>– Possible Hyper-reflexive</td>
</tr>
<tr>
<td>– Hypoesthesia</td>
<td>– Hyperesthesia/After sensation</td>
</tr>
<tr>
<td>– Loss of strength</td>
<td>– Irritation with Provocation positions</td>
</tr>
<tr>
<td>– Irritation with compressive positioning</td>
<td>– Distal &quot;tender points&quot;</td>
</tr>
<tr>
<td>– Hair loss</td>
<td>– Mechanical Hypalgiesia</td>
</tr>
<tr>
<td>– Possible positive electrodiagnostics</td>
<td>– Neg. Electrodiagnostics</td>
</tr>
<tr>
<td></td>
<td>– Pain inhibition</td>
</tr>
</tbody>
</table>

Three Major Causations for Symptom or Sign Manifestations With Peripheral Nerve Pathology

1. Adherance of the epi-endo-or perineum leading to loss of elongation for normal functional patterns.
2. Pseudoneuroma formation leading to pain avoidance or direct distal inflammatory responses.
3. Prior peripheral nerve injury leading to excessive protection and physiological reaction to second load or trauma.
Physiological, Inflammatory and Neuropathic Pain

“A region of potential abnormal impulse initiation may not become symptomatic until local adhesions or a change in posture causes undue mechanical forces to be brought to bear.”

Woolf C J
Advances and Technical Standards in Neurosurgery

Development of a Pseudoneuroma

Caused by:
- Direct mechanical disruption of the epineureum, perineurium, or endoneurium.
- Rupture of epi or endoneural blood vessels.
- Chemical disruption of the epineural barrier such as exposure to nucleus pulposus in annular tears.
**Slide 25**

**Development of a “Pseudoneuroma”**

“Obstruction to the venous outflow from a funiculus slows intrafunicula circulation.”

Increase intrafunicular pressure→Vascular collapse→Breakdown of Blood Nerve Barrier→Decrease axonal Transport→Stasis→Hypoxia=Excitability

“Painful Lesion”... Sjostrands J et al, Lundberg et al.

“Pseudoneuroma”... Le Min M ET Al, Cavanagh et al.

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**Slide 27**

“When pain from localised peripheral neural pathology becomes widespread, tenderness can be found along the course of the affected nerve.”

- Devor, Lishman, Quitner, etc.
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**Neurogenic Rheumatica**

The usual diagnosis of arthritis, bursitis, neuritis, muscular rheumatism, fibrositis should not be made until cervical nerve root irritation has been considered. Joint swelling may be directly caused by inflamed nerve roots.


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Research into "algias" suggest that the reason that many problems once referred to as "itis", do not have normal inflammatory product findings. Instead neurogenic inflammatory products are found such as PABA etc. Possible neurogenic causation of Lat. Epicondalgia...?

Copepeters, Fernandez De-Los-Peñas

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**What Causes Severe Reactions to Minor Afferent Input?**
Causations of Central Sensitization

- Long standing nociceptive input. (Cavenaugh, Chang, Cheng, Haroutounian, etc.)

- Prior history of radiculopathy or CRPS involving ipsilateral or contralateral limb with new pain inputs involving the same nerve root. Could be Antidromic or Orthodromic. (Kim, Hunt, etc.)

- "Double Crush" (Summation of inputs) (Osterman, Ang, etc.)
1st Stage: Peripheral Afferent Sensitivity
(Pathological Changes That Occur After Injury)
• Increased Mechano-sensitivity. (As Stretch Receptor, hyper-to-compression due to hypoxia)
• Altered Chemosensitivity. (Epi, Peri, Endoneural Breakdown)
• Altered Trophic Influence on Peripheral Target Tissue. (Neurogenic rheumatica.)
• Altered Connections (C-fiber re-investment, Phenotypic Switch)
• Pain Avoidance Movement Patterns (Nerve Elongation Avoided)
Orthodromic vs Antidromic
Afferent Sensory Patterns

Orthodromic Impulses
Conduction along an axon (normally thought to be sensory) in its normal direction, away from the target structures. Modified at spinal synapse. Summates. Repetitive input causes ramping.

Antidromic Impulses
Conduction along the axon (thought to be sensory) away from the axon terminal(s) and towards the distal target structures. Monitored by the CNS and both modifies and contributes to pain syndromes. May cause release of chemicals at distal site.

Does Antidromic Activation of Nociceptors Play a Role in Sciatic Radicular Pain
(Sauer ET AL PAIN No 40, pg. 17-79, 1990)

Primary afferent input critical for maintaining spontaneous pain in peripheral neuropathy.

2nd Stage (normally)
isCentral Sensitization
Central Sensitization

A) Dorsal Horn Sensitization (WDR Neuron) Cavenagh, Geppetti, Svendsen

B) CNS Sensitization (Thalmus-Red Nucleus, Descending Fiber Excitation, Changes in Cortex Mapping, etc) Butler, Sluka, Kramis

C) Autonomic Over Reactivity Bennett, Geppetti,
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**Newly Developed Subjective Exams**


**painDetect** Freynhagen, Current Medical Research and Opinion: vol.22, pgs 1911-1920 2006

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**Pain Drawings**

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**DERMATOMES** vs **DYNATOMES**

"Not Consistent"

Dalton, P: Australian Journal of Physiotherapy Vol. 35, No. 1

Curtis, Slipman – SPINE Vol. 23, No. 20, 1998
Pain Descriptions
(Individualized)

- May describe “burning”, heaviness, “deep bone ache”, “a headache in my foot”, “cold water running down my leg”, “feels numb”, “hurts to touch”, “lightning”, “tingles and hurts at the same time”, etc. etc. etc.
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Need Analogue Pain Scales

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Objective Evaluation:

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[Image]
Objective: Use of Standard Neurological Eval

- Reflexes: Maybe hyper-reflexive.
- Pinwheel: Hyper/hypo pin point sensation, may have increased receptor fields, after sensation. Rate pain responses.
- Vibratome: 128 HZ Tuning Fork: Mechanical Hypoesthesia
- Von Frey Fibers: Impaired tactile discrimination, two point discrimination.
- Strength: Test in provoked position.
- Clonus, Babinski etc.

Used to help determine the "health" of the nerve and to rule out other neurological considerations.

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Review of the "Duck"

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Signs of Adverse Response to the Physical Examination of Neural tissues "THE DUCK"

- Posture
- Active Dysfunction
- Passive Dysfunction
- Nerve Trunk Hyperalgesia
- Tender Points
- Specific Signs of Local Dysfunction
Neural Tissue Sensitivity and Protective Reflex Muscle Activity.

Protective antalgic posture. The recognition of protective muscle hypertonicity.

Hall T 1996, Sherrington CS 1900
"Transformation of flexion withdrawal reflex from high threshold phasic to low threshold tonic."


Biomechanics of Neuromeningeal Tissues Involved in Active and Passive Testing
Validity of Upper Limb Neurodynamic Tests for Detecting Peripheral Neuropathic Pain.

Active Dysfunction
Add and Subtract Specific Nerve Provocation Positions
Passive Dysfunction
Add and Subtract Nerve Provocation Positions

Ectopic Sensory Discharges & Paraesthesiae in Patients with Disorders of Peripheral Nerves, Dorsal Roots & Dorsal Columns.

Provocation Tests showed abnormal discharges on sensory pathways using microneurography

Nordin M. ET AL PAIN Vol. 20, Pgs. 231-245, 1984
Muscular Recruitment Patterns with and without Provocation.
Use of surface electrode EMG
Using Nerve Bias Position to Rule in and Rule Out Muscle Tightness

**Example:** Scalenes, Levator, Shoulder External Rotators

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Nerve Trunk Hyperalgesia (Mechanical Alodynia)

Elvey 1981
Fernandez de Las Penas 2010
Hall and Marshall 2009
Hall and Quintner 1996
Lishman, Nordin, M 1994
Devor, M 1991
“When pain from localised peripheral neural pathology becomes widespread, tenderness can be found along the course of the affected nerve.”

- Devor, Lishman, Quitner, etc.

Neural Hyperalgesia can be Objectified by use of Algometry

- Test Pain Threshold
- Test Pain Tolerance


Specific Mechanical Pain Hypersensitivity Over Peripheral Nerve Trunks in Women with Either Unilateral Epicondylalgia or Carpal Tunnel Syndrome

Nerve Palpation: In Supine

Trunks of the brachial plexus. C5, 6, 7, 8, T1.
Posterior Triangle of the neck at the interscalene shelf and the supraclevicular fossa, over the first rib. In the infraclevicular fossa. Caudal/medial to the coracoids.

The Peripheral Nerve Trunks: Below the clavicle, proximal to the deltoids.
The Median Nerve C5,6,7,8, T1. Along the distal third of the anterior/medial upper arm between biceps and brachialis muscles.
The Ulnar Nerve C7,8, T1. At the posterio/medial elbow. Proximal and distal to the Ulnar Groove.
The Radial Nerve C5,6,7 (primarily). At the posterior arm in the lower one-third of the Radial Groove.

In Prone:
The Axillary Nerve C5,6. between posterior deltoideus and teres minor.
The Suprascapular Nerve C5,6. Supraspinous and Infraspinous Fossa
Radial Nerve as described in other slide.
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**Tender Points**
Will be Found in Muscles Innervated by Involved Nerve


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**Specific Signs of Local Dysfunction**

- Loss of passive joint motion at specific levels of dysfunction. Will have a painful, springy or bogey end feel often with a muscular rebound noted in multifidi etc.

- Should display tenderness from problem site distally, rarely proximally.

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**Putting it All Together**

- Meshing Subjective, Objective, and Patient History to come to a Differential Diagnosis
Subjective
1. Pain diagram and reproduction of symptoms during exam should match. Often produce dominant and specific pain symptom related to original complaints.
2. Complaints of sensitivity (hyperesthesia, allodynia, and temperature abnormalities) should be objectified.
3. Patients lack of ability to precisely describe abnormal sensations should not be regarded as signs of malingering. Often use words such as “heavy”, “puffy”, “electrical”, “burns”, “like a toothache in my...... etc.
4. Pay close attention to reports of multiple trauma involving the same nerve roots. Make sure to question prior history.

Working With the Duck (Objective Findings)
1. Must determine peripheral neuropathy vs spinal neuropathy or radiculopathy, vs. systemic disease?: (Reflex, Pinwheel, Strength,Tinel’s, Distalization etc.)
2. Anatomical Relationships between pain locality and spinal motion segments must match if central problem exists.
   (Example: Lateral Epicondylitis C45, C56)
3. Must have specific correlation between provocation tests of neural tissue and spinal segmental signs.
   (ex. Positive Ulnar Nerve Tests and C4C5 Segment Dysfunction= NO CORRELATION)
4. There must be specific correlation between neural palpation tests and provocation tests of neural tissue.
   (Example: Median Nerve Palpation with Positive Median Provocation Tests= CORRELATION)
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5. Trigger Points or "Tender Points" should correlate with Spinal Level of Involvement unless only postural... i.e. Levator, Upper Traps etc. (Example: C5C6 dysfunction and Trigger Point or Tender point in the Teres Minor)

6. Should be able to “turn on” and “turn off” findings by use of specific provoking and relieving positions.

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Medical Considerations

• Systemic Disease changes...Diabetes...Shingles...ART meds., radiation
  • Sleep Deprivation
  • Emotional Overlay

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“Ortho”
Case Presentation
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“Sports Medicine”
Patient Presentation

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Treatment of Irritative-Inflammatory Neural Pain Syndromes, PNP’s, PNS

Patient’s who meet the criteria for irritative or inflammatory dysfunctions, are initiated on the following treatment protocols, dependent on the severity. Chronicity and complexity of their problems.
Contraindications

- Some conditions with neurological deficit. CNS disorders or compressive radiculopathies.
- Progressive neurological pathology.
- When treatment exacerbates a condition or causes distal symptoms.
- When another form of treatment or management is indicated. Eg. joint disorder, metabolic neuropathy, etc.

Treatment of neural tissues by techniques of mobilization of surrounding anatomical tissues or of the neural tissues.

Techniques DO NOT involve stretching. 15% elongation NORMAL neural tissue causes microvascular stoppage (Lundberg G. et al)
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**POSTULATION for Treatment Effect**

- Mechanical effect
- Neurophysiological modulation (Central Desensitization)
- Physiological effect

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**1. Therapist’s Intervention:**

Passive Mobilization of the environment surrounding the affected nerve in very specific positions and levels determined by the evaluation. May also include the use of modalities such as Heat, Ultrasound, and/or Electrical Stimulation to decreased muscle guarding or help with neural irritability. Only used to allow therapist to administer mobilization more effectively. In many cases modalities are not needed. Direct segmental mobilization may of course be needed.

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2. Patient Self Treatment:

Once therapist’s intervention has a carry over between treatments, patients are initiated on home programs of neuro-mobilization exercises to allow for continued benefit of movement and to restore function around inflamed nerve.
Home Programs

Active Neural Mobilization
Standing
Seated
Flossing

Home Nerve Gliding Techniques
“Flossing”
Additional Home Programs

In addition to Neural “Mobilization”
May include:
Desensitizing, Mirror Box, Loading
Techniques etc.

3. Therapist’s Specific Muscle Re-
education:
Patient’s normally have developed loss of specific
muscular strength and endurance. This is due to
disuse atrophy, reflexive inhibition, or neural
compression. May require modification of
positioning into unprovoked position to “honor”
the “Mother in Law”.
Remember fear avoidance issues!!! Good time to
initiate “general ex” programs to break patterns of
disability and increase overall physiological return.

Patient’s Self Specific Movement Re-
education:
Home exercise, guided by the therapist.
Patterned movement ie. Functional patterns of
movement that were substituted for when the
nerve was injured and need to be restored to
allow for return to function including work
activities without re-injury or injury of another
area. Once again remember provoked
positioning and pain memory!!
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Specific Functional Task Education and Rehabilitation:

Certain heavy demand or labor intensive job tasks require specific “work hardening” procedures to return many of these “Chronic Pain Patient’s” to prior job or modified job status.

Communication with treating Therapist and Rehab Counselor is essential.

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Conclusion

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Patient Specificity
1. Each patient is unique! Find and treat “Their Pain”
2. Must be able to turn on and turn off symptoms.
3. Goal is to reverse process of sensitization, self perpetuation and allow for normal healing of pseudoneuroma.
4. Must remember all aspects of the treatment environment are critical.
5. Hope is given and healing begins with proving diagnosis to yourself and especially the patient. This requires a thorough exam and good communication.
Remember Regional Intervention.

Treatment of the “Inflamed Nerve”, is only the beginning of functional restoration. By decreasing and often eliminating the input and secondary reactions to the “Angry Mother in Law”, we can begin the process of rehabilitation that the pain input did not allow and may have led to an incorrect diagnosis and treatment plan. None of the treatments presented are an “End All”, but rather a beginning and a component of the final restorative process.

Thank You Very Much for Your Participation
Specific Physical Therapy Evaluation

Signs and Features must be re-assessable:
Patients symptoms should be reproduced at some point of this process!!

1. **Observation**: Body positioning that decreases elongation or provocation of nerve.

2. **Active Movement**: Specific motions with and without nerve provocation positions.

3. **Passive Movement**: Specific motions with and without nerve provocation positions. These are repeated provocation tests with “Central” and Peripheral Positions.

4. **Nerve and nerve trunk hyperalgesia** to mechanical pressures.

5. **Trigger Point or “Tender Point” palpation**. Look for myotomal distribution matching neural irritability.

6. **Evidence of Central or Peripheral Causation**. Must find localized source of irritability, adhesion, or compression or any combination to effectively treat.

**Neural Provocation Evaluation**

____________(Upper Quarter)______________

**Postural Postioning:**
Forward Head___Cervical Lateral Tilt___Shldr. Elevation R_L_
Elbow Position_______ Wrist Position_____ Fingers___

**Active Motion Tests:**

**Shoulder Active Abduction ROM**

Neutral Cervical Position____ Scapula Fixation___________
Scapula Fixated and Head in Ipsilateral Flexion__________
    Contralateral Flexion_________
**Plus:** Elbow Flexed______ Elbow Extended___________
Noted Substitution________________________

**Passive Motion Tests (N.T.P.T.)**

**Peripheral to Central**

**Median Nerve:** Cervical Neutral___Contralateral___Ipsilateral___
**Radial Nerve:** Cervical Neutral___Contralateral___Ipsilateral___
**Ulnar Nerve:** Cervical Neutral___Contralateral___Ipsilateral___

**Central to Peripheral**

Degrees of Cervical Lateral Flexion R___L___ with Upper Extremity in
________________________position. Increases with ____________

**Neural Palpation**

**Trunks:** Superior____Middle____Inferior_____
**Axillary Palpation**______________________________
**Median Nerve** ____Location____________________
Radial Nerve Location

Ulnar Nerve Location

Axillary Nerve Location

Tender Points

Specific Signs of Local Dysfunction

C3C4__C4C5__C5C6__C6C7__C8T1__T1T2__T2T3
NEURAL PALPATION

In Supine:

Trunks of the brachial plexus. C5, 6, 7, 8, T1.

- Posterior Triangle of the neck at the interscalene cleft and the supraclavicular fossa, over the first rib.
- In the infraclavicular fossa. Caudal/medial to the coracoids.

The Peripheral Nerve Trunks: Below the coracobrachialis.

- The Median Nerve C5,6,7,8,T1. Along the distal third of the anterior/medial upper arm between biceps and brachialis muscles.
- The Ulnar Nerve C7,8,T1. At the posterior/medial elbow. Proximal and distal to the Ulnar Groove.
- The Radial Nerve 5,6,7 (primarily) At the posterior arm in the lower one-third of the Radial Groove.

In Prone:

- The Axillary Nerve C5,6 between posterior deltoid and teres minor.
- The Suprascapular Nerve C5,6. Supraspinous and Infraspinous Fossa
- Radial Nerve as described above.
Home Programs

Once the Patient has displayed carryover from therapist intervention with manual techniques and the patient displays good understanding of irritative factors, the patient may be started on self-treatment. The patient stands or sits in front of a mirror (elbow supported) and first tests their active ROM of Cervical Lateral Flexion away from the symptomatic side with upper extremity in neutral. The patient then adds the specific provoked position only up until approximately one third of the Cervical Lateral Flexion is lost. Seven to Ten repetitions are completed without producing symptoms. Patient can expect to feel tightness in the antagonistic musculature, however, symptoms should not distalize. Patient can repeat these “treatments” one or two times daily. Do not refer to them as exercises. If symptoms increase the next day, rest twenty four to forty eight hours and retry. It is imperative that patients recheck positioning each time and treat each session as separate from the last one.
Home Nerve Gliding Techniques
“Flossing”


35. Coppieters MW, The immediate effects of a cervical lateral glide technique in patients with


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**Abbreviated Biblio for Neurogenic Rheumatica:**

In addition to others listed in the manual, there has been several published studies in veterinary journals using isotope tracers on neuropeptides at the proximal nerve roots and tracing them out to their terminal branches in the joints, skin, muscle etc.

These were presented by a Dr. Bove, D.C., PhD. Neurophysiologists from the U.S. At a pain seminar I attended in 2001. Michelle Cooputers P.T., G.D.M.T., (see your manual biblio) has been working on similar yet unpublished research on the distal transport of systemic or inflammatory waste down the axon in peripheral nerve inflammation. Herpes Zoster research gives a glimpse also into this phenomena as the cause of cutaneous pustule formations. Recent research by Fernandez-De-Las-Penas (see biblio and other available articles) suggest that the distal nerve is responsible for maintenance of the inflammatory responses of pain and sensitivity and this is why no normal inflammatory products are found at the tendon sites. (I will cover this in the Advanced Course) Here are a few other references.


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