Neurodynamics and the ‘Double Crush Syndrome’

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What do you think?

A nerve disorder in the upper limb or neck is a predisposition for the development of a second nerve disorder in the upper limb or neck eg. carpal tunnel syndrome following cervical radiculopathy.

- Strongly disagree
- Disagree
- Neither disagree nor agree
- Agree
- Strongly agree
History

- Onset of (R) wrist pain (arthritis)
- Steroid injections x 2
- STT fusion December 2008
- Infection++ (6 weeks of intravenous antibiotics, morphine, 7 plasters)
- Carpal tunnel release at 6 months – worse
- 5/12 post-carpal tunnel surgery: Widespread numbness and loss of dexterity in all fingers
Initial assessment

- Feels as if ‘arm doesn’t belong’
- Frustration +++
- ‘Clumsy’
- Difficulty tying fishing hooks
- Difficulty cooking, DIY, gardening, using computer keyboard
- Able to dress but difficulty with trouser zips
- Feeling angry – directed towards surgeon
Physical examination

- Restricted cervical movements (all except flexion)
- Restricted thoracic movements (all)
- Hypomobility 1\textsuperscript{st} rib
- All shoulder girdle movements restricted by pain
- Only 90° active glenohumeral flexion and & 90° abduction
- Non-dermatonal weakness at all levels from C5 to T1
- Highly positive neurodynamic tests - bilaterally
Neurodynamics

- How nerves slide and glide
- Return from an elongated position to a shortened one
- Withstand:
  - compression
  - jolting
  - repetitive forces
  - bending
Goal

- To be able to fish (teaching, competitions)

Expectations

- ‘Doubts’ about the future
- Wants hand to be as it was pre-operatively
- Realistic that it will not be.
Clinical conundrums

- ‘Far worse’ following surgery
- 12 months: symptoms did not fit with traditional healing timescales e.g. neuropraxia
- Distribution of numbness did not match nerve conduction findings
- Bilateral signs
- Mismatch in expected outcome between the surgeon and patient
Hypothesis: ‘Double crush’

- Single axons, compressed in one region, become susceptible to damage at another site.
- Electrophysiological & clinical evidence of nerve lesions at the neck in 81/115 patients with CTS or ulnar nerve lesions at the elbow.

www.vietnamchiropractic.com/wrist-en.html
Prevalence of cervical radiculopathy in patients with carpal tunnel syndrome as reported by different authors.

<table>
<thead>
<tr>
<th>Publication</th>
<th>Prevalence</th>
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<tbody>
<tr>
<td>Morgan and Wilbourn, 1998</td>
<td>5%</td>
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<tr>
<td>Kuntzer, 1994</td>
<td>6%</td>
</tr>
<tr>
<td>Yu et al., 1979</td>
<td>11%</td>
</tr>
<tr>
<td>Cassvan et al., 1986</td>
<td>14%</td>
</tr>
<tr>
<td>Pierre-Jerome and Bekkelund, 2003</td>
<td>16–53%</td>
</tr>
<tr>
<td>Osterman, 1988</td>
<td>18%</td>
</tr>
<tr>
<td>Moghtaderi and Izadi, 2008</td>
<td>24%</td>
</tr>
<tr>
<td>Herczeg et al., 1997</td>
<td>33%</td>
</tr>
<tr>
<td>Liveson, 1991</td>
<td>48%</td>
</tr>
<tr>
<td>Upton and McComas, 1973</td>
<td>76%</td>
</tr>
<tr>
<td>Golovchinsky, 1995</td>
<td>94%</td>
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### TABLE 1
The Biologic/Metabolic, Structural, Toxicogenic, and Vasculopathic Factors Seen in Neuropathies and Possibly Implicated in the Pathogenesis of Double Crush Syndrome

<table>
<thead>
<tr>
<th>Biologic/Metabolic</th>
<th>Structural</th>
<th>Toxicogenic</th>
<th>Vasculopathic</th>
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<tbody>
<tr>
<td>Amyotrophic lateral sclerosis (ALS); multiple sclerosis; Bell’s palsy; diabetes mellitus; autoimmune disease; connective tissue diseases; herpes zoster; HIV; Lyme borreliosis, leprosy, meningitis, sarcoidosis; Guillain Barré syndrome; meningitis; ethanol abuse; uremia-B₂ microglobulin amyloid deposition; hypothyroidism, B₆/B₁₂ (pyridoxine/cobalamin) deficiency; granulomatosis</td>
<td>Cervical ribs (thoracic outlet syndrome); Klippel Feil syndrome; ankylosing spondylitis; carpal tunnel syndrome; tarsal tunnel syndrome; meralgia paresthetica; cervical/lumbar spondylosis; Martin-Gruber anastomosis; enlarged bicipital bursa; accessory head of flexor pollicis longus (Gantzer muscle); thickened aponeurosis of flexor carpi ulnaris (between two heads of flexor carpi ulnaris); Guyon’s canal; fascial junction of brachioradialis and extensor carpi radialis longus</td>
<td>Ethanol abuse; drugs (cisplatin, vincristine, Nitrofurantoin, phenytin, reverse transcriptase inhibitors)</td>
<td>RA; scleroderma; polyarteritis nodosa; Sjögren’s syndrome; atherosclerosis; thrombosis; vascular-induced ischemia from local anesthetics that contain epinephrine; Churg-Strauss syndrome; diabetes mellitus</td>
</tr>
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*Childs SG. Orthopaedic Nursing 2003;22(2):117-21*
Delphi study

- Round 1: Ascertaining beliefs about the double crush hypothesis
- Round 2: Identified 22 possible mechanisms
- Round 3: Rated plausibility:
  - $\geq 7$ = Highly plausible
  - $>4 \& <7$ = Plausible
  - $\leq 4$ = Implausible

Axonal transport

- Animal studies: axonal transport impaired at pressure levels commonly seen in patients
- Chemical blockage of axonal transport increases mechanosensitivity of axons locally & just proximal to blocking site, but not distally
- But: proposed that peripheral nerve & dorsal root have separate axonal transport systems
- Impaired axonal transport by itself cannot explain CTS & ulnar neuropathy as different nerves have different transport systems
Ion channel up or downregulation

- Upregulation (sodium channels) & downregulation (potassium channels), distal as well as proximal to the primary nerve injury may lower the firing threshold of neurones
- Growing evidence for upregulation of specific sodium channels locally as well as in the dorsal root ganglia, dorsal horn and thalamus
- Sodium channels rapidly recover from inactivation and are excitable by depolarisations below the action potential threshold (associated with neuropathic pain)
Immune-inflammation of the dorsal root ganglia

- Animal studies demonstrated an invasion of immune cells in the dorsal root ganglia following peripheral nerve injury.
- Excitatory cytokines released by immune cells may lower the firing threshold of sensory neurones.
- Cell bodies of intact & injured neurones lie in very close proximity in the dorsal root ganglia.
- Immune-inflammation at this site may affect intact axons originating from a site distant to the injury.
Neuroma-in-continuity

- If the epineurium remains intact after peripheral nerve injury, regenerating axons sprout along the nerve trunk
- Axonal sprouts may fail to reach their peripheral targets, forming ‘neuroma-in-continuity’
- Regenerating axons are more sensitive to mechanical and thermal stimuli & may exhibit ectopic activity
Mechanical considerations

- Tight muscles (Butler, 2000).
- Some scarring, creating foci of mechanical pressure (Butler, 2000).
- Abnormal impulse generating site (Butler 2000).
- Fibrotic & shrunk peripheral nerve sheath (Millesi et al., 1995).
- Possible connections between peripheral nerves, ‘common’ (Butler, 2000).
- Irritation, compression or traction of the brachial plexus → neurological thoracic outlet syndrome (Watson et al., 2009).
Cadaveric evidence

- Zoech et al. 10 unfixed cadavers
- At 90° abduction, 100 mm of excursion from median nerve bed in wrist flexion + elbow flexion to wrist + elbow extension
- Average nerve length of 517mm, this represents 19% length difference
Problems with the term ‘double crush’

- Proximal focal disturbance could result from traction rather than compression
- Could be more than 2 sites of injury (‘triple-crush’, ‘quadruple-crush’, ‘multiple-crush’)
- Generalised subclinical polyneuropathy in all peripheral nerve fibres could serve as the proximal compression site
Arguments against ‘double crush’ hypothesis

- There must be anatomic continuity of nerve fibres between the lesion sites. If this is lacking, sequential impairment of axoplasmic flow cannot occur.
- Lesions within the intraspinal canal do not support the hypothesis when they involve sensory fibres.
- Sensory fibres injured proximal to cell bodies of origin in DRG: distal lesions damage post-ganglionic sensory fibres.
- Pre- & post-ganglionic sensory fibres are not anatomically continuous, even though they share the same unipolar cell body. Injury to one has no effect on the other unless it affects their common cell body in the DRG.
### Predictive factors for poor outcome for carpal tunnel release

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<th>Co-morbid factors</th>
<th>Diabetes</th>
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<tr>
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<td>Poor health status</td>
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<td></td>
<td>Smoking, alcohol</td>
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<tr>
<td>Misdiagnosis</td>
<td>Thoracic outlet syndrome</td>
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<tr>
<td></td>
<td><strong>Double crush</strong></td>
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<td></td>
<td>Normal nerve conduction studies</td>
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<tr>
<td>Physical findings</td>
<td>Abductor pollicis brevis atrophy</td>
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<td>Other factors</td>
<td>Workers’ compensation with legal involvement preoperatively</td>
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Clinical implications: some challenges

- ‘An incidental effect of this theory is that it provides an acceptable explanation for two or even more failed operative procedures performed to treat essentially the same symptoms’
  
  Wilbourn AJ & Gilliatt RW

- Complaints and medico-legal action
- Should reviewing the spine (including neurodynamic testing) be undertaken prior to every carpal tunnel release?
- Could it be considered negligent to operate without having checked the spine?
Treatment

- Manual therapy (cervical & thoracic regions)
- Soft tissue techniques
- Neurodynamic home exercises
- Mobilisation of the scapula and first rib
- Local friction massage to the carpal tunnel scar site
Outcome

- Able to fish in competitions for 6+ hours
- ‘100% recovery’
- Arm is ‘Stronger than ever’
- Climbed a tree to retrieve a child’s fishing tackle!
Case report

Clinical conundrums in a case of upper quadrant dysfunction

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Summary

- ‘Double crush’ is a useful hypothesis
- Mechanical and physiological mechanisms are still unknown
- 22 mechanisms have been suggested
- History taking is paramount (keep an open mind)
- Challenge when the evidence does not appear to ‘fit’
- Remember the neurodynamic tests in your clinical kit-bag
- Proposal: That neurodynamic testing should be mandatory prior to all carpal tunnel surgery.