Plexus and Peripheral Nerve Imaging

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Plexus and Peripheral Nerve Imaging

Leonardo Da Vinci illustration: brachial and LS plexus
Plexus and Nerve Imaging

- Anatomy
- MR neurography: technical considerations
- Diagnostic approach
- Clinical examples
  - Brachial plexus
  - Lumbosacral plexus
  - Peripheral nerves
Nerve Anatomy

Nerve Anatomy (Sciatic n.)

- Axon > Myelin > Endoneurium > Fascicle > Perineurium > Epineurium (internal and external)

3D T1 SPACE

DW PSIF
Nerve Anatomy (Sciatic n.)

3D T1 SPACE

DW PSIF

Fascicle/fascicle group
Diagnostic Strategies

- EMG/NCS
- Myelography
- MR neurography (MRN)
- US
- Exploration
MRN: Technical Considerations

♣ What is MR neurography?
  • Technically demanding, knowledge-intensive, time-consuming
  • High resolution and high contrast nerve-selective and nerve-nonselective imaging sequences
  • Multidisciplinary

♣ General considerations
  • 3T > 1.5T (except for metal), good coils, focus on limited anatomy
  • 2D and 3D, multiplanar reformatting and review
  • Axial (transverse) for nerves, long axis for regional evaluation
  • Fat saturation, flow suppression, limit motion, shim for 3D and diffusion
**Some specifics**

- Tailor fat sat
  - STIR for metal, plexus, larger patient/region 2D or 3D
  - SPAIR for peripheral nerves, small patient/region 2D or 3D
  - Dixon option with 2D
- Spatial resolution
  - 2D: 0.3-0.4mm in-plane, 2-3mm slice peripheral, 3-4mm proximal
  - 3D: 1-1.5mm isotropic
- 2D imaging
  - T1 (anatomy), T2 fat saturated (pathology)
- 3D imaging
  - Nerve nonselective (SPACE, CUBE, VISTA)
  - Nerve selective (DWI, DTI, DW-PSIF)
MRN: 1.5T vs. 3T with Hardware

T2 TSE FS pelvis 3T

T2 TSE L-spine 1.5T MAR
MRN: Fat Suppression
MRN: Flow Suppression

- 3D STIR – blood suppression with diffusion gradients
- PSIF with diffusion weighting
MRN: Flow Suppression

3D STIR SPACE (blood suppression)

DW-PSIF
MRN: Diagnostic Approach

- Identify spine and regional pathology (e.g., disc herniation)
- Look for focal nerve/plexus pathology
- Look for diffuse nerve/plexus pathology
- Assess for entrapment/compression
- Recognize denervation patterns in muscle
- Myopathy instead of neuropathy?
- Awareness of prior surgery
- Detailed assessment individual nerve and plexus elements increasingly possible, but question often more simple:
  - Abnormality present or absent?
  - Symmetric or asymmetric?
  - Diffuse or focal?
  - General anatomic localization (e.g., pre or post-ganglionic injury)
## MRN: Diagnostic Approach

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Normal</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Size</strong></td>
<td>Similar to adjacent artery Gradually decreases distally</td>
<td>Focal or diffuse enlargement Larger than the adjacent artery</td>
</tr>
<tr>
<td><strong>Signal intensity</strong></td>
<td>T1W &amp; T2W: isointense to skeletal muscle</td>
<td>T2 hyperintensity (becoming similar to adjacent veins)</td>
</tr>
<tr>
<td></td>
<td>STIR/fat suppressed T2W: isointense to minimally hyperintense</td>
<td>Asymmetric hyperintensity on 3D TSE</td>
</tr>
<tr>
<td></td>
<td>3D TSE (turbo spin echo): uniform and symmetric hyperintensity</td>
<td></td>
</tr>
<tr>
<td><strong>Fascicular pattern</strong></td>
<td>T1W &amp; T2W: present and uniform</td>
<td>Single or multiple fascicles enlargement/disruption Loss of fascicular pattern</td>
</tr>
<tr>
<td><strong>Course</strong></td>
<td>Smooth without focal deviations</td>
<td>Focal or diffuse deviations Discontinuity</td>
</tr>
<tr>
<td><strong>Enhancement</strong></td>
<td>Absent (except in areas of deficient blood-nerve barrier, such as dorsal nerve root ganglion)</td>
<td>In tumors &amp; infections (disruption of the blood-nerve barrier)</td>
</tr>
<tr>
<td><strong>Perineural fat</strong></td>
<td>Clean fat planes</td>
<td>Perineural strand like T1 &amp; T2 hypointensities/nerve encasement</td>
</tr>
<tr>
<td><strong>Diffusion tensor imaging, tracts</strong></td>
<td>Normal tracts</td>
<td>Abnormal tracts, disrupted or displaced</td>
</tr>
<tr>
<td><strong>Diffusion tensor imaging, quantitative</strong></td>
<td>Normal fractional anisotropy values (&gt;0.4–0.5)</td>
<td>Abnormal fractional anisotropy values (&lt;0.4–0.5)</td>
</tr>
<tr>
<td><strong>Diffusion tensor imaging, qualitative</strong></td>
<td>Symmetric brightness of nerves on tensor images</td>
<td>Asymmetrical hyperintensity of the neuropathic nerve</td>
</tr>
</tbody>
</table>

Table from Chhabra, NCNA 2014;24:79
Clinical: Brachial Plexus

- Anatomy
- Protocol
- Trauma
- Impingement: TOS, etc.
- Neoplasm
- Inflammatory, treatment-related
- Hereditary
Brachial Plexus Anatomy

Note: Usual composition shown. Prefixed plexus has large C4 contribution but lacks T1. Postfixed plexus lacks C5 but has T2 contribution.

Fig. 1. Schematic drawing of the normal right brachial plexus.
Brachial Plexus: Imaging Anatomy

Fig. 1. Schematic drawing of the normal right brachial plexus.
Brachial Plexus: Imaging Anatomy

Fig. 1. Schematic drawing of the normal right brachial plexus.

Divisions

ant post

ant post

Anterior scalene
Posterior scalene
Middle scalene
Anterior cord
Middle cord
Posterior cord
Neural plexus
C4
C5
C6
C7
T1
Clavicle
Subclavian artery
Subclavian vein
Clavicle
Manubrium

V A
Brachial Plexus: Imaging Anatomy

Fig. 1. Schematic drawing of the normal right brachial plexus.
Brachial Plexus: Imaging Anatomy

Fig. 1. Schematic drawing of the normal right brachial plexus.
**Protocol**

- **Side of interest**
  - T1 ax and sag
  - T2 fatsat (STIR) ax
  - STIR sag

- **Bilateral**
  - 3D T1 SPACE cor
  - 3D STIR SPACE cor

- **Options**
  - Post-contrast T1 (Dixon, IDEAL)
  - DWI/DTI
  - 3D T2 sag myelographic
  - Maneuvers, vascular imaging (TOS)

### 3T MRN examination protocol for the evaluation of the brachial plexus (FOVs from C2 to T2)

<table>
<thead>
<tr>
<th>MR imaging sequence</th>
<th>FOV</th>
<th>Section Thickness</th>
<th>TR/TE/TF (ms)</th>
<th>Matrix</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1 coronal (T1 axial)</td>
<td>30</td>
<td>4.0</td>
<td>881/11/7</td>
<td>512 × 512</td>
</tr>
<tr>
<td>3D coronal STIR SPACE</td>
<td>30</td>
<td>1.0</td>
<td>1500/97/53</td>
<td>256 × 256</td>
</tr>
<tr>
<td>3D sagittal T2 SPACE</td>
<td>25</td>
<td>1.0</td>
<td>1000/97/81</td>
<td>256 × 256</td>
</tr>
<tr>
<td>STIR sagittal, bilateral, affected side</td>
<td>22–24</td>
<td>3.0</td>
<td>5210/18/22</td>
<td>256 × 256</td>
</tr>
<tr>
<td>Additional arm examination if desired</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Axial T1</td>
<td>20–22</td>
<td>3.0–4.0</td>
<td>550/7.9/6</td>
<td>256 × 384</td>
</tr>
<tr>
<td>Axial T2 SPAIR</td>
<td>20–22</td>
<td>3.0–4.0</td>
<td>2840/70/13</td>
<td>256 × 384</td>
</tr>
</tbody>
</table>

**Table from Chhabra et al, AJNR 2013;34:486**
Trauma

- Birth trauma, MVA/MCC most common, “burners/stingers”
- OB trauma
  - Erb (Duchenne) paralysis (C5-7, more common)
  - Klumpke paralysis (C8-T1)
- Mechanisms of traction, direct blow, crush, laceration
  - Upper trunk (C5-6) or all roots more common than lower trunk
  - Stretching injury (usually recovers)
    - diffuse asymmetric thickening, T2 prolongation, enhancement
  - Pre-ganglionic avulsion: no recovery, early intervention (neurotization)
    - Pseudomeningocele, cord or paraspinal muscle signal abnormality/enhancement, hemidiaphragm elevation, other signs include Horner’s, scapular winging
  - Post-ganglionic rupture: variable recovery, intervention (neurotization, direct repair/graft, neurolysis) depends on injury
    - Neurotmesis, neuroma, hematoma

Mauricio Castillo. Imaging the Brachial Plexus. AJR:185, December 2005
MRI and CT myelography
- 80% neurosurgeons prefer postmyelography CT, 20% prefer MRI, and 41% use both methods for surgical planning.
- CT myelography remains standard for root avulsion

MR differentiation between pre- and postganglionic lesions
- Pseudomeningoceles
  - 15% without avulsion
  - 20% avulsions without pseudomeningocele
- Cord signal abnormality
  - 20% preganglionic injuries
  - Enhancement at avulsion site
- Paraspinal muscle enhancement (esp. multifidus) in 24 hrs, edema within a few days

Postganglionic
- Laceration, posttraumatic neuromas
- Hematomas, vascular injury including pseudoaneurysm, fibrosis

Plain film (hemidiaphragm elevation), US

Mauricio Castillo. Imaging the Brachial Plexus. AJR:185, December 2005
Trauma: Pre-Ganglionic
Trauma: Pre-Ganglionic
Trauma: Pre-Ganglionic

- Multilevel avulsions without clear pseudomeningoceles
Trauma: Pre-Ganglionic

- Multilevel avulsions with pseudomeningoceles
Trauma: Pre-Ganglionic

- Cord abnormality s/p avulsions
Trauma: Post-Ganglionic

- Multilevel avulsions with pseudomeningoceles and stretch + probable postganglionic avulsion/division
Trauma: Post-Ganglionic

Division with neuromas

Avulsion

Fig. 7 from Chhabra AJNR 2013; 34: 486

Fig. 10A from Lutz et al, Neuroimag Clin N Am 2014; 24: 91
Trauma: Traction

- LUE weakness after injury, weight fell on shoulder

Courtesy of Igor Mikityansky, MD, MPH
Trauma: Post-Ganglionic

- iatrogenic: positioning injury upper trunk, complete recovery
## Surgical Intervention

### Table 1
Classification and expected recovery of nerve injuries

<table>
<thead>
<tr>
<th>Classification</th>
<th>Seddon</th>
<th>Sunderland Degree (Modified)</th>
<th>Nerve Component Injured</th>
<th>Expected Recovery</th>
<th>Surgery Indicated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurapraxia</td>
<td>First</td>
<td>Yes</td>
<td>No</td>
<td>Complete</td>
<td>None</td>
</tr>
<tr>
<td>Axonotmesis</td>
<td>Second</td>
<td>Yes</td>
<td>Yes</td>
<td>Good</td>
<td>None</td>
</tr>
<tr>
<td>Axonotmesis</td>
<td>Third</td>
<td>Yes</td>
<td>Yes</td>
<td>Variable</td>
<td>None or neurolysis</td>
</tr>
<tr>
<td>Axonotmesis</td>
<td>Fourth</td>
<td>Yes</td>
<td>Yes</td>
<td>None</td>
<td>None or neurolysis</td>
</tr>
<tr>
<td>Neurotmesis</td>
<td>Fifth</td>
<td>Yes</td>
<td>Yes</td>
<td>None</td>
<td>Nerve repair</td>
</tr>
<tr>
<td></td>
<td>Sixth</td>
<td>Combination of injury</td>
<td></td>
<td>Variable</td>
<td>Variable</td>
</tr>
</tbody>
</table>


Table from Baltodano et al, Neuroimag Clin N Am 2014;24:235

See also:

- Sunderland, Brain 1951;74:491
- Pindrik and Belzberg, Neuroimag Clin N Am 2014;24:193
Surgical Intervention

- Timing important, e.g., severed nerves can be repaired immediately, avulsions may get nerve transfer early or late (within 6 months), but subacute phase problematic
- Surgical options include neurolysis, direct repair, nerve grafting (autologous, biologic and synthetic nerve guides), neurotization (nerve transfers)

<table>
<thead>
<tr>
<th>Type of Nerve Injury</th>
<th>Acute Setting</th>
<th>Medium Term Follow-Up</th>
<th>Longer Term Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nerve laceration</td>
<td>Early surgical intervention after stabilization of other traumatic injuries</td>
<td>Postoperative clinical examination</td>
<td>Adjuvant surgery as required (tendon or muscle transfers)</td>
</tr>
<tr>
<td>Progressive loss of function over 12–24 h</td>
<td>Urgent diagnostic evaluation, surgery for compressive hematoma, pseudoaneurysm, etc</td>
<td>Postoperative clinical examination</td>
<td>Clinical follow-up to assess recovery</td>
</tr>
<tr>
<td>Blunt or stretch injury</td>
<td>Initial clinical examination, stabilization of other traumatic injuries</td>
<td>EMG and NCS at 2 wk following injury</td>
<td>Clinical examinations and repeat EMG/NCS at 3-mo intervals; surgery as needed</td>
</tr>
<tr>
<td>Obstetric brachial plexus injury</td>
<td>Initial clinical examination</td>
<td>Repeat clinical assessment at age 3 mo; consider surgical intervention at age 3–6 mo</td>
<td>Delayed repair as needed (tendon or muscle transfers)</td>
</tr>
<tr>
<td>Entrapment neuropathy</td>
<td>Clinical history and examination; EMG and NCS</td>
<td>Conservative management, physical therapy, analgesic medications</td>
<td>Surgical considerations if conservative management ineffective</td>
</tr>
</tbody>
</table>

Pindrik & Belzberg, NCNA2014;24:193
Nerve Transfer

- **Common nerve transfers**
  - Oberlin: ulnar > musculocutaneous
  - Radial > axillary

- **Basic goals**
  - Shoulder stability
  - Arm abduction
  - Elbow flexion

---

Table 10: Examples of nerve transfer in brachial plexus injury

<table>
<thead>
<tr>
<th>Brachial Plexus Components Injured</th>
<th>Donor Nerve</th>
<th>Recipient Nerve</th>
<th>Desired Functional Recovery</th>
<th>Additional Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper trunk, C5 root</td>
<td>Spinal accessory n.</td>
<td>Suprascapular n.</td>
<td>Shoulder stability, abduction</td>
<td>Requires bridging sural n. graft</td>
</tr>
<tr>
<td>Upper trunk</td>
<td>Spinal accessory n.</td>
<td>Musculocutaneous n.</td>
<td>Elbow flexion, shoulder abduction</td>
<td>May be performed in acute or late setting</td>
</tr>
<tr>
<td>Upper trunk</td>
<td>Spinal accessory n.</td>
<td>Obturator n., gracilis muscle transfer</td>
<td>Elbow flexion, wrist extension</td>
<td>Requires 2-3 intercostal n. fibers per transfer</td>
</tr>
<tr>
<td>Upper trunk, C5-C6 avulsion</td>
<td>Intercostal n.</td>
<td>Musculocutaneous n.</td>
<td>Elbow flexion</td>
<td>Requires 2-3 intercostal n. fibers per transfer</td>
</tr>
<tr>
<td>Complete plexus avulsion</td>
<td>Intercostal n.</td>
<td>Obturator n., gracilis muscle transfer</td>
<td>Finger flexion, elbow extension</td>
<td>Requires 2-3 intercostal n. fibers per transfer</td>
</tr>
<tr>
<td>Complete plexus avulsion</td>
<td>Contralateral C7 nerve root</td>
<td>Median n.</td>
<td>Hand function, shoulder stability</td>
<td>Requires bridging ulnar n. graft</td>
</tr>
<tr>
<td>Complete plexus avulsion</td>
<td>Phrenic n.</td>
<td>Suprascapular n.</td>
<td>Shoulder stability, abduction</td>
<td>Risk of pulmonary compromise, contraindicated in children</td>
</tr>
<tr>
<td>Complete plexus avulsion</td>
<td>Phrenic n.</td>
<td>Axillary n.</td>
<td>Shoulder stability, abduction</td>
<td>Requires bridging n. graft, same risks as above</td>
</tr>
<tr>
<td>Upper trunk, C5-C6 roots</td>
<td>Ulnar n.</td>
<td>Musculocutaneous n.</td>
<td>Elbow flexion</td>
<td>Classic Oberlin technique, modified Oberlin technique</td>
</tr>
<tr>
<td>Upper trunk, C5-C6 roots</td>
<td>Ulnar n.</td>
<td>Obturator n., gracilis muscle transfer</td>
<td>Elbow flexion</td>
<td></td>
</tr>
<tr>
<td>C5-C6 avulsion</td>
<td>Radial n.</td>
<td>Axillary n.</td>
<td>Shoulder stability</td>
<td>Short distance of reinnervation through quadrangular space</td>
</tr>
</tbody>
</table>

Abbreviation: n., nerve(s).

Pindrik & Belzberg, NCNA2014;24:193
Thoracic Outlet Syndrome (TOS)

- **Entrapment**
  - Neurogenic much more common than vascular
- **Interscalene and costoclavicular space > retropectoral**
- **Etiology**
  - Fibro-osseus
    - Cervical rib
    - Elongated transverse process C7
    - Exostosis or anomaly first rib or clavicle
    - Scarring
  - Muscular hypertrophy
    - Scalene, subclavius, pectoralis minor
- **Symptoms**
  - Pain, Raynaud phenomenon
  - Arterial insufficiency, edema
  - Vascular injury/thrombosis (e.g., Paget-Schroetter or effort induced venous thrombosis, pseudoaneurysm)
TOS: Etiologies
TOS: Neurogenic
Thoracic Outlet Syndrome
Thoracic Outlet Syndrome
Thoracic Outlet Syndrome
TOS: Vascular
TOS: Vascular (Paget-Schroetter)
TOS: Vascular (Paget-Schroetter)
Neoplasm: Intrinsic

- **Most common**
  - Neurofibroma: nerve inseparable, not encapsulated
  - Schwannoma: eccentric to nerve, encapsulated
  - 1/3 neurofibromas in neurocutaneous syndromes (plexiform neurofibromas in NF 1)
  - Target, fascicular, tail sign on T2 and split fat sign on T1, but imaging not always clear and does not distinguish between PNST

- **MPNST (malignant PNST)**
  - Reliable differentiation from benign is difficult
  - Local invasion, indistinct margin, absence of target sign (or loss), large size, bone destruction
  - Significant change or rapid growth, change in symptoms
  - Biopsy ultimately required

- **Non-neurogenic**
  - Desmoid most common
  - Lipoma
  - Lymphoma

Neoplasm: Extrinsic

- More for planning than neurography per se
- Adjacent neoplasm or treatment related change
  - Primary or secondary neoplasm most common (e.g., Pancoast)
  - Other neurogenic tumors like vagus nerve schwannoma, paraganglioma, neuroblastoma, ganglioneuroblastoma, ganglioneuroma
    - Non-BP neurogenic tumors more vertical orientation
  - Direct extension from primary or secondary bone neoplasm
  - Postsurgical and/or postradiation scarring, fibrosis
- Primary or secondary involvement with some pathology
  - Lymphoma, sarcoma, venolymphatic malformation
- Hematogenous metastases to BP are rare
Neoplasm: Schwannoma

Fusiform mass along axis of C6, UT and posterior cord
Neoplasm: Plexiform Neurofibroma

“Target” sign
Neoplasm MPNST

T1 pre

T1 post

lateral

medial

T1+C

STIR
Neoplasm: Non-Neurogenetic (Desmoid)
Neoplasm: Non-Neurogenic (Pancoast)

- Superior sulcus lung cancer
- Pancoast syndrome: pain shoulder to arm and along ulnar aspect, muscle atrophy, Horner’s, possible vascular involvement
Neoplasm: Non-Neurogenic (Lymphoma)
Neurolymphomatosis

- Waldenstrom macroglobulinemia: bilateral arm numbness, weakness, LUE pain
- Lymphoma can primarily involve plexus, more commonly secondary
Neurolymphomatosis (Waldenstrom)

Left distal

Left proximal

Courtesy of Igor Mikityansky, MD, MPH
Neoplasm versus Treatment

- Breast CA, prior radiation: rapid onset pain and weakness
- Unilateral diffuse enlargement and T2 prolongation
Inflammatory: Radiation Plexopathy
Inflammatory Plexopathy

- Sudden onset pain, weakness, paresthesias
- Can be sensory only
- Most common etiology is radiation (plexitis or fibrosis)
  - 5-30 months after treatment, usually > 6000cGy
  - Prognosis worse for acute (<6mo) than chronic
- Other etiologies
  - Infection (e.g., viral, lyme, atypicals like leprosy)
  - Immune-mediated, toxic, iatrogenic (e.g., nerve blocks)
  - Idiopathic
- Imaging findings in irradiated region
  - T2 prolongation, mild enhancement, loss of intraneural/intraplexal architecture
  - Diffuse thickening, nonfocal
  - Denervation
Muscle Findings

- Diffuse plexopathy
- Patchy muscle involvement, no specific innervation pattern
Denervation: Muscle Findings

- Early: normal, possible enhancement (e.g., cervical avulsions)
- Subacute: edema, enhancement
- Chronic: volume loss, fatty replacement

Note this can be confusing with plexus pathology – muscle groups corresponding to multiple nerve distributions can create confusion with myositis.

Table 6
Differential diagnosis of abnormal muscle signal on MR imaging

<table>
<thead>
<tr>
<th>Focal/Patchy</th>
<th>Diffuse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious myositis</td>
<td>Denervation</td>
</tr>
<tr>
<td>Delayed-onset muscle soreness</td>
<td>Postinfectious/inflammatory (Parsonage-Turner syndrome)</td>
</tr>
<tr>
<td>Trauma (contusion)</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Muscular strain</td>
<td>Disuse atrophy</td>
</tr>
<tr>
<td>Polymyositis/myopathy</td>
<td>Polymyositis/myopathy</td>
</tr>
<tr>
<td>Tumor</td>
<td></td>
</tr>
</tbody>
</table>

Delaney et al, NCNA 2014;24:127
Inflammatory: Muscle Findings

- Brachial neuritis (Parsonage Turner), subacute denervation
Inflammatory: Parsonage Turner Syndrome

- Usually self-limited, duration months to years
- Men > women, may be bilateral
- Presents with pain, then weakness
- Imaging findings
  - Diffuse thickening and T2 prolongation
  - Denervation pattern may not follow typical innervation distributions
Inflammatory

- Radiation, Parsonage Turner Syndrome
- GBS (AIDP, axonal GBS, MFS/Bickerstaff)
- CIDP (Chronic Inflammatory Demyelinating Polyneuropathy)
  - Typical
    - Clinical: symmetric, motor and sensory, elevated CSF protein
    - Imaging pattern: symmetric, more proximal nerves and plexus, diffuse, ± enhancement
  - Atypical
    - MADSAM (Multifocal Acquired Demyelinating Sensory and Motor) or Lewis-Sumner
    - Clinical: acquired, immune-mediated multifocal demyelinating polyneuropathy
    - Imaging pattern: symmetric, more peripheral, multifocal, ± enhancement
- MMN (Multifocal Motor Neuropathy)
  - Immune-mediated demyelinating neuropathy/polyneuropathy
  - Asymmetric motor involvement, sensory spared
  - More peripheral nerves especially UE
  - Increased GM1 specific IgM
- Others including paraproteinemic, vasculitic, autoimmune
- Hereditary neuropathies can mimic
CIDP

37 YF with quadriplegia and tremors
AJNR Case of the Week July 14, 2014
Estela Gomez, MD; Agustin Marrero, MD; Eduardo Cosci, MD
Investigaciones Medicas, Buenos Aires, Argentina
CIDP?

A, B: typical CIDP

C, D: atypical CIDP (MADSAM or Lewis-Sumner)

E: CMT 1A
F: spondylotic

Fig. 1, from Shibuya et al, Ann Neurol 2015; 77:333, A-F are STIR MIPs
### Table 1 | Differential diagnosis of multifocal motor neuropathy

<table>
<thead>
<tr>
<th>Feature</th>
<th>Multifocal motor neuropathy</th>
<th>Amyotrophic lateral sclerosis</th>
<th>Lower motor neuron disease</th>
<th>Chronic inflammatory demyelinating polyneuropathy</th>
<th>Lewis–Sümmner syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distribution of weakness</td>
<td>Asymmetric</td>
<td>Asymmetric</td>
<td>Asymmetric</td>
<td>Symmetric</td>
<td>Asymmetric</td>
</tr>
<tr>
<td>Prominent sensory symptoms</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Tendon reflexes</td>
<td>Normal or decreased in weakened muscles*</td>
<td>Increased in weakened muscles</td>
<td>Decreased in weakened muscles</td>
<td>General hyporeflexia or areflexia</td>
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<tr>
<td>Disease course</td>
<td>Slowly progressive</td>
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<td>Progressive or relapsing</td>
<td>Progressive or relapsing</td>
</tr>
<tr>
<td>Cerebrospinal fluid protein &gt;1g/l</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Rare</td>
</tr>
<tr>
<td>Increased titers of GM1-specific IgM antibodies</td>
<td>Common</td>
<td>Rare</td>
<td>Rare</td>
<td>Rare</td>
<td>Rare</td>
</tr>
<tr>
<td>Abnormal MRI signal in the brachial plexus</td>
<td>Asymmetric</td>
<td>No</td>
<td>No</td>
<td>Symmetric</td>
<td>Asymmetric</td>
</tr>
<tr>
<td>Response to intravenous immunoglobulin</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Response to corticosteroids</td>
<td>No†</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

*In some patients, reflexes are brisk. †May aggravate symptoms.

Clinical: Lumbar Plexus

- Protocol
- Impingement: Piriformis, etc.
- Tumors/treatment
- Inflammatory
- Hereditary
Lumbosacral Plexus

Figures 1, 2. (1) Illustration shows the anatomy of the lumbar plexus from T12 to L5, with “T” denoting thoracic and “L” denoting lumbar nerve roots. Peripheral nerve branches (*) to the psoas and iliacus muscles are also illustrated. (2) Illustration shows the anatomy of the sacral plexus from L4 to S5, with “L” denoting lumbar and “S” denoting sacral nerve roots. + = peripheral nerve branches to the piriformis, * = quadratus femoris and inferior gemellus, ** = obturator internus and superior gemellus, x = levator ani, coccygeus, and sphincter ani externus.

Soldatos et al, Radiographics 2013;33:971
Lumbosacral Plexus: Imaging Anatomy

3D T1 SPACE

3DSTIR SPACE

SN

LFCN

FN

SGN

SN

LS trunk
Lumbosacral Plexus: Imaging Anatomy

FN (STIR SPACE)

ON (T1 SPACE)
Segmental schwannomatosis
Protocol

♦ Bilateral
  • T1 ax
  • T1 fatsat ax
  • 3D T1 SPACE cor (or PD)
  • 3D STIR SPACE ax or cor
  • 3D PSIF ax

♦ Options
  • Post-contrast T1 (Dixon or IDEAL)
  • DWI/DTI
  • 3D T2 sag myelo spine

Table 3
Dedicated pelvic MR neurography protocol

<table>
<thead>
<tr>
<th>MR Imaging Sequence</th>
<th>Plane</th>
<th>FOV (cm)</th>
<th>Slice Thickness (mm)</th>
<th>TR (ms)</th>
<th>TE (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>Axial</td>
<td>38</td>
<td>3</td>
<td>640</td>
<td>6.3</td>
</tr>
<tr>
<td>T2 FS</td>
<td>Axial</td>
<td>38</td>
<td>3</td>
<td>5420</td>
<td>63</td>
</tr>
<tr>
<td>PD</td>
<td>Coronal</td>
<td>38</td>
<td>3</td>
<td>4000</td>
<td>39</td>
</tr>
<tr>
<td>3D SPACE-SPACE</td>
<td>Axial</td>
<td>38</td>
<td>0.8</td>
<td>1000</td>
<td>92</td>
</tr>
<tr>
<td>3D PSIF</td>
<td>Axial</td>
<td>38</td>
<td>1.1</td>
<td>12.19</td>
<td>2.38</td>
</tr>
<tr>
<td>T1 FS C+</td>
<td>Axial</td>
<td>38</td>
<td>3</td>
<td>400-800</td>
<td>min</td>
</tr>
</tbody>
</table>

Siemens Verio 3T MR imaging scanner, Erlangen, Germany.
* Optional.

Delaney et al, NCNA 2014;24:127
As for brachial plexus, any mass can cause compression
- Benign: bursae, paralabral cysts (even clothing)
- Neoplastic: neurogenic (more commonly benign), any primary and secondary neoplasm from or to adjacent structures

Some locations/nerves with predilection for compression or surgical or other traumatic injury
- Traction injuries: sciatic, femoral, obturator nerves with abdominal, hip and GU surgery, stretch injuries with trauma (avulsions less common than BP)
- Direct: femoral nerve with vascular intervention, SGN with hip replacement, obturator nerve with GU surgery
Compression

- Sciatic nerve dysfunction after long flight related to paralabral cyst tracking to nerve
Compression: Paralabral Cyst (US)
Neoplasm: Schwannoma

Tail sign, split fat sign, and orientation along axis for PNSTs in general, but eccentric to S2 nerve so favors schwannoma over neurofibroma.
Neoplasm
Neoplasm: Ganglioneuroma
Neoplasm: Perineuriroma
Chronic Denervation (Perineuriroma)
MPNST: Bone Involvement
Inflammatory

- Pancreatic cancer s/p chemotherapy
- Indolent LLE pain
Inflammatory

Pre-T1

Post-T1

Pre-T1
Multidisciplinary Input Needed

Table 1 | Differential diagnosis of multifocal motor neuropathy

<table>
<thead>
<tr>
<th>Feature</th>
<th>Multifocal motor neuropathy</th>
<th>Amyotrophic lateral sclerosis</th>
<th>Lower motor neuron disease</th>
<th>Chronic inflammatory demyelinating polyneuropathy</th>
<th>Lewis–Sumner syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distribution of weakness</td>
<td>Asymmetric</td>
<td>Asymmetric</td>
<td>Asymmetric</td>
<td>Symmetric</td>
<td>Asymmetric</td>
</tr>
<tr>
<td>Prominent sensory symptoms</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Tendon reflexes</td>
<td>Normal or decreased in weakened muscles*</td>
<td>Increased in weakened muscles</td>
<td>Decreased in weakened muscles</td>
<td>General hyporeflexia or areflexia</td>
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<td>No</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
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<td>No†</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

*In some patients, reflexes are brisk. †May aggravate symptoms.


Nat. Rev. Neurol. doi:10.1038/nrneurol.2011.175
Clinical: Peripheral Nerves

- For neurologist, distribution, temporal progression and electrophysiology are key
- MR neurography not part of standard workup but evolving
- Neuropathies
  - Entrapment
  - Toxic/Metabolic (Diabetes most common, hypothyroid, B12 deficiency, renal/hepatic, chemotherapy)
  - Inflammatory and immune-mediated
    - GBS (AIDP, axonal GBS, MFS/Bickerstaff)
    - CIDP
    - MMN
    - Others including paraproteinemic, vasculitic, autoimmune
  - Infection (leprosy, Lyme, HIV, CMV, EBV, VZV, others)
  - Neoplastic/paraneoplastic (SCLC anti-Hu, direct involvement, lymphoproliferative (gammopathies), GVHD)
  - Hereditary
Peripheral Nerve Injury

- **Wide spectrum**
  - Birth injury: Erb, Klumpke palsy
  - Young adults: trauma
  - Older adults: entrapment, neoplasm, iatrogenic

- **Types of injury**
  - Penetrating/blunt trauma, compression, crush, stretch/traction, avulsion, iatrogenic

- **Nerve injury classification**
  - Neuropraxia: conduction block, myelin not axon, recovery weeks to months
  - Axonotmesis: loss of conduction at injury and distal, axon interrupted but perineurium and epineurium are intact, recovery longer
    - Prognosis good if endoneurial tube intact
    - Prognosis poor if endoneurial tube is not intact
  - Neurotmesis: loss of nerve conduction at injury and distal, nerve severed, prognosis poor
Conclusions

- **MR neurography**
  - Technically demanding, knowledge-intensive, time-consuming
  - High resolution and high contrast nerve-selective and nerve-nonselective imaging sequences
  - **Multidisciplinary**
    - Clinical input from neurology, neurosurgery, orthopedic surgery, pain medicine
    - Knowledge base across multiple radiology subspecialties including musculoskeletal and neuroradiology

- **Base concepts**
  - 3T > 1.5T (except for metal), good coils, focus on limited anatomy
  - 2D and 3D, multiplanar reformatting and review
  - Axial (transverse) for nerves, long axis for regional evaluation
  - Fat saturation, flow suppression, limit motion, shim for 3D and diffusion
## Surgical Intervention

### Table 1: Classification and expected recovery of nerve injuries

<table>
<thead>
<tr>
<th>Seddon Degree (Modified)</th>
<th>Myelin</th>
<th>Axon</th>
<th>Endoneurium</th>
<th>Perineurium</th>
<th>Epineurium</th>
<th>Expected Recovery</th>
<th>Surgery Indicated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurapraxia</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Complete</td>
<td>None</td>
</tr>
<tr>
<td>Axonotmesis First</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Good</td>
<td>None</td>
</tr>
<tr>
<td>Axonotmesis Second</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Variable</td>
<td>None</td>
</tr>
<tr>
<td>Axonotmesis Third</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>None</td>
<td>None or neurolysis</td>
</tr>
<tr>
<td>Axonotmesis Fourth</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>None</td>
<td>Nerve repair</td>
</tr>
<tr>
<td>Neurotmesis Fifth</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>None</td>
<td>None</td>
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<tr>
<td>Sixth</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Variable</td>
<td>Variable</td>
</tr>
</tbody>
</table>


### Table 4: Sunderland classification of peripheral nerve injury

<table>
<thead>
<tr>
<th>Sunderland Degree</th>
<th>Components Damaged</th>
<th>Clinical Outcome Expected</th>
<th>Additional Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No structural damage; only conduction loss</td>
<td>Complete recovery</td>
<td>Consistent with neurapraxia</td>
</tr>
<tr>
<td>II</td>
<td>Axon, myelin</td>
<td>Complete recovery</td>
<td>Longer recovery period than degree I injuries</td>
</tr>
<tr>
<td>III</td>
<td>Axon, endoneurium, fascicle</td>
<td>Incomplete recovery with residual deficit</td>
<td>Intrafascicular hemorrhage and fibrosis complicate recovery</td>
</tr>
<tr>
<td>IV</td>
<td>Axon, endoneurium, fascicle, perineurium, internal epineurium</td>
<td>Partial spontaneous recovery; limited function of involved distal targets</td>
<td>Typically requires surgical repair</td>
</tr>
<tr>
<td>V</td>
<td>Entire nerve structure</td>
<td>Negligible recovery</td>
<td>Typically requires surgical repair</td>
</tr>
</tbody>
</table>

Data from Sunderland S. A classification of peripheral nerve injuries producing loss of function. Brain 1951;74:491–516.

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Pindrik and Belzberg, NCNA 2014;24:193

Baltodano et al, NCNA 2014;24:235