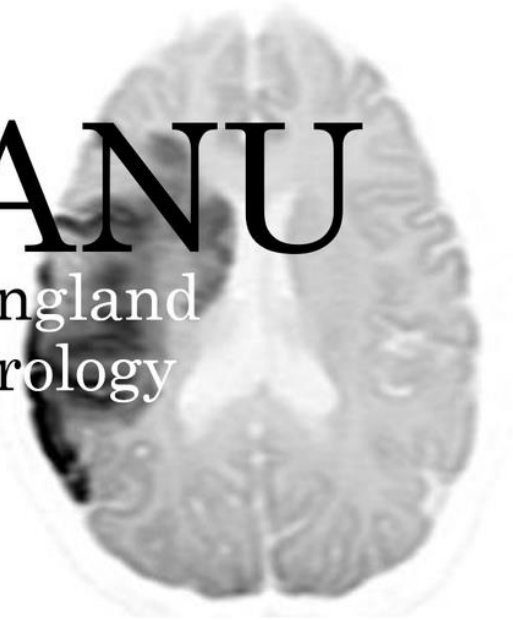
NEANU
North of England
Acute Neurology
Update



Electrodiagnostics: *NCS/EMG*

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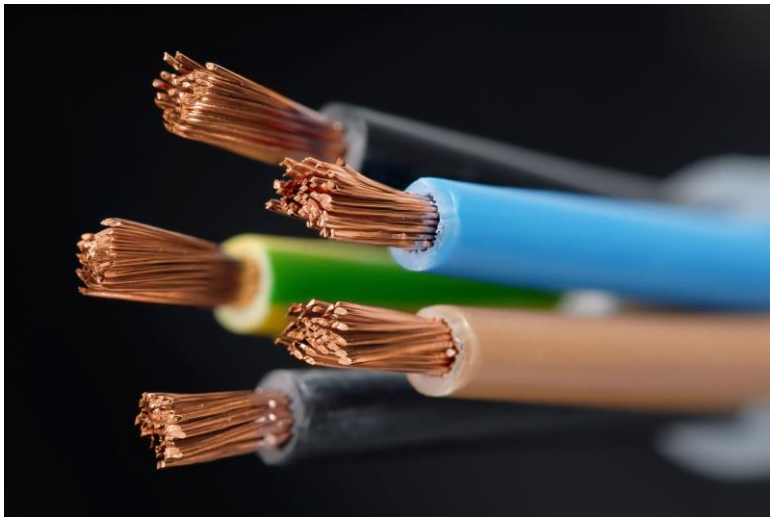
Manchester Centre for Clinical Neurosciences

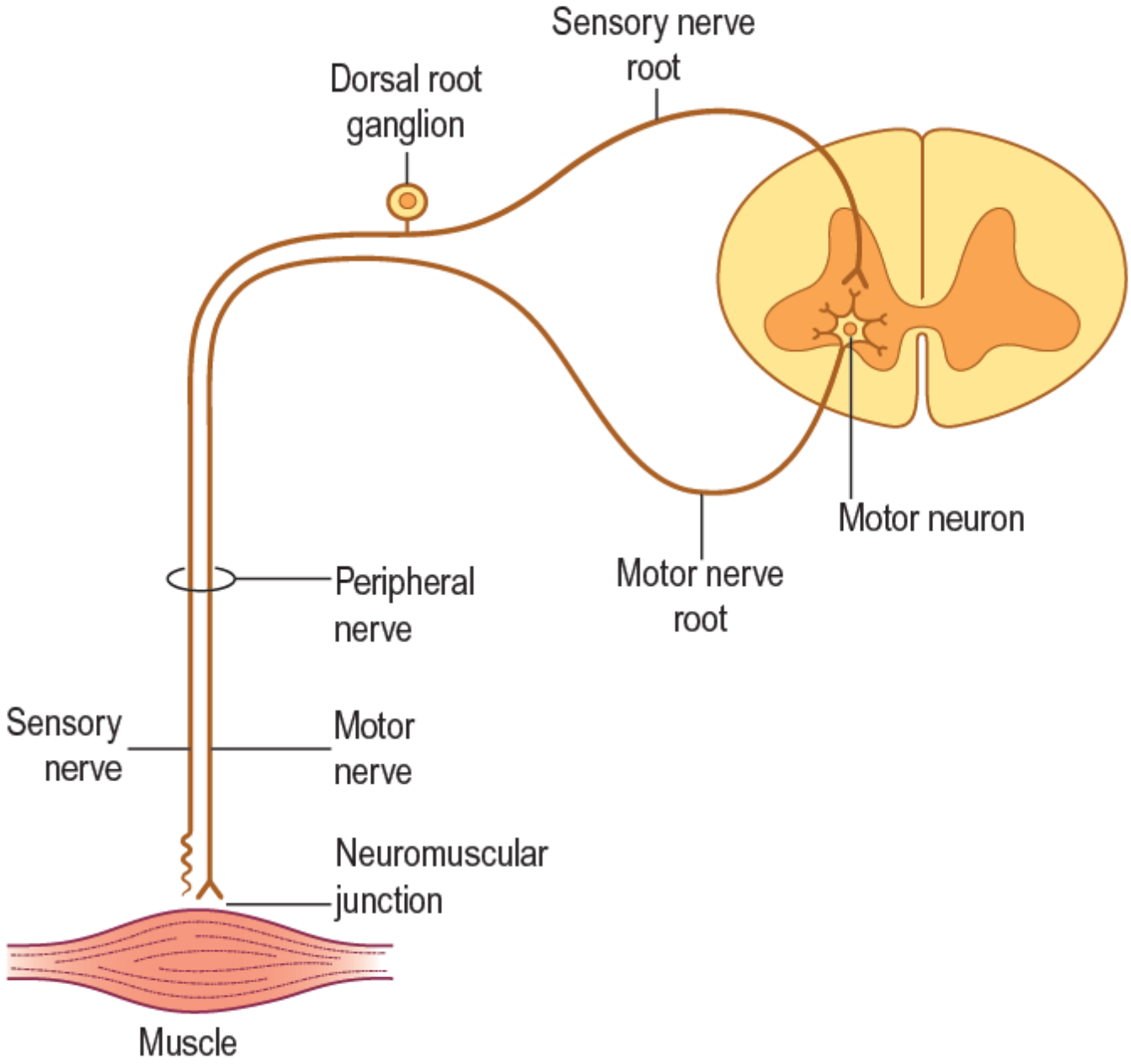
Salford Royal NHS
NHS Foundation Trust

Two principle components

1. Nerve conduction studies (NCS)
   - A test of *peripheral nerve* function
   - *Motor* and *sensory* nerves assessed separately
   - The function of the *axon, conducting sheath* and *neuromuscular junction* are tested

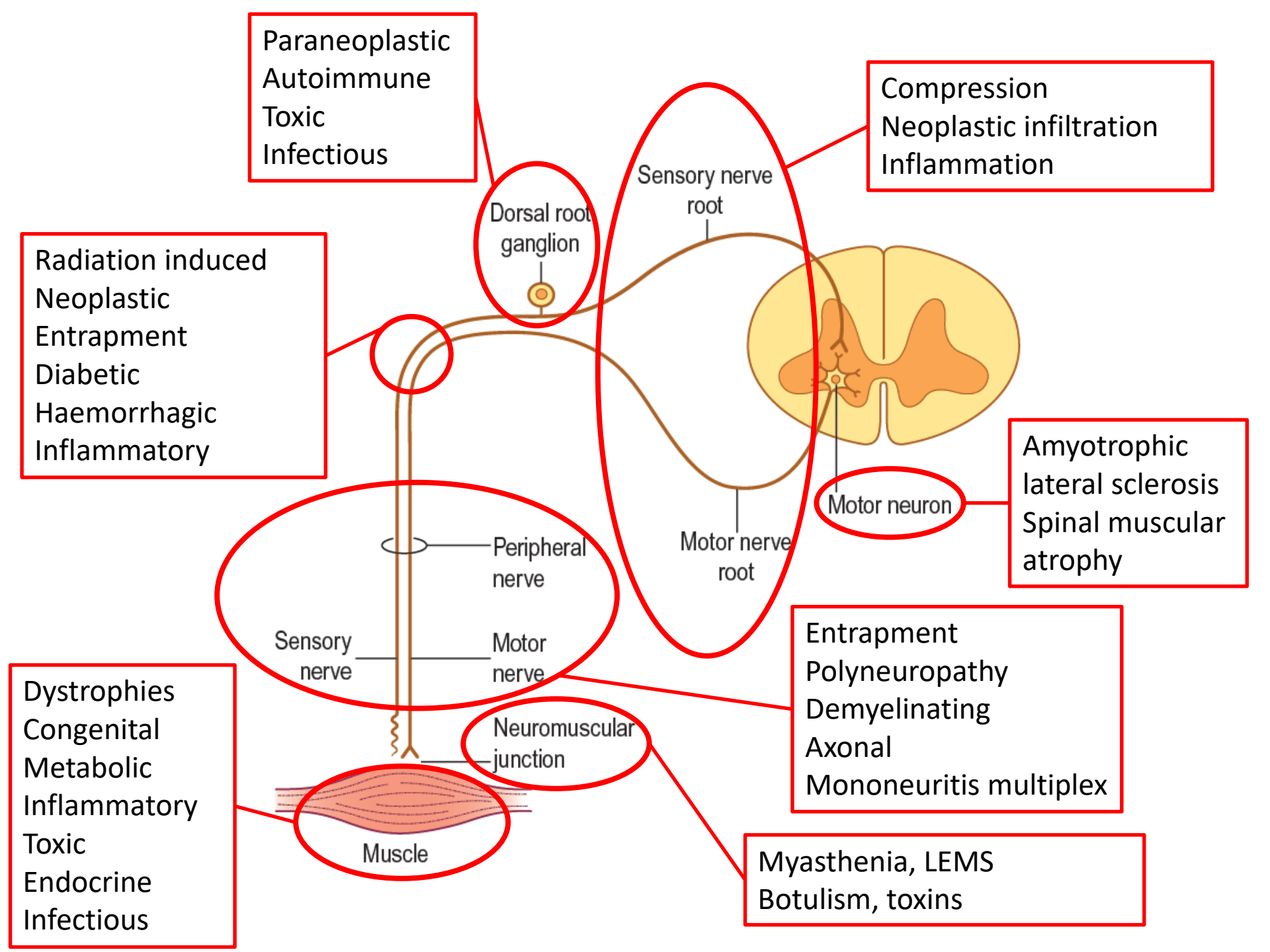


2. Electromyography (EMG)
   - Electrical recording of muscle to assess:
   - Motor innervation
   - Function


Sensory nerve root
Dorsal root ganglion
Motor neuron
Peripheral nerve
Motor nerve root
Sensory nerve
Motor nerve
Neuromuscular junction
Muscle

Key Principles of Electrodiagnostics

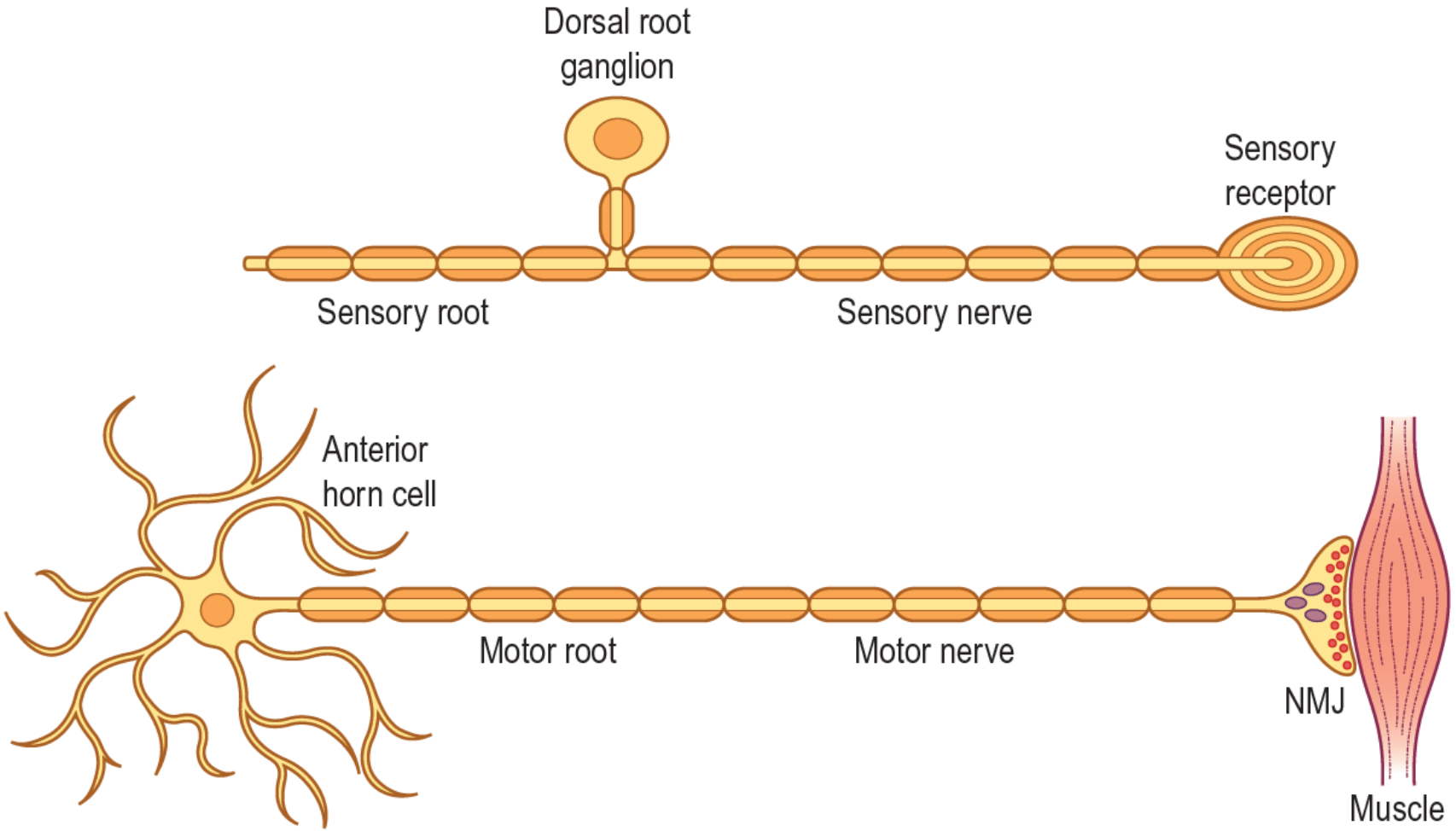
- Goal is to first localise the lesion
  - Work out which bit of the nervous system is affected
- Potentially aetiologies may *then* be considered
- An extension of the clinical examination
  - “rubbish in, rubbish out”


Paraneoplastic
Autoimmune
Toxic
Infectious
Compression
Neoplastic infiltration
Inflammation
Radiation induced
Neoplastic
Entrapment
Diabetic
Haemorrhagic
Inflammatory
Amyotrophic lateral sclerosis
Spinal muscular atrophy
Entrapment
Polyneuropathy
Demyelinating
Axonal
Mononeuritis multiplex
Dystrophies
Congenital
Metabolic
Inflammatory
Toxic
Endocrine
Infectious
Myasthenia, LEMS
Botulism, toxins
Dorsal root ganglion
Sensory nerve root
Motor neuron
Motor nerve root
Peripheral nerve
Sensory nerve
Motor nerve
Neuromuscular junction
Muscle

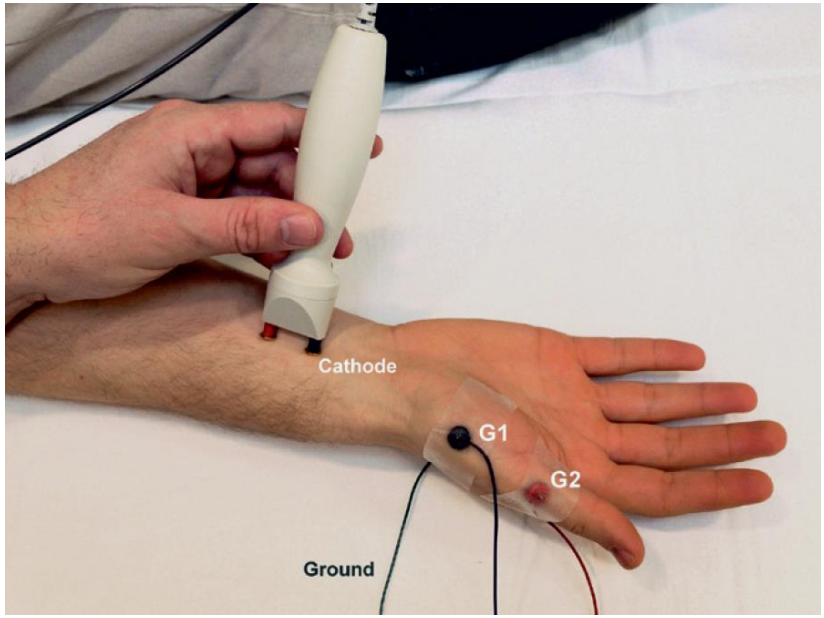
What to expect from your neurophysiologist

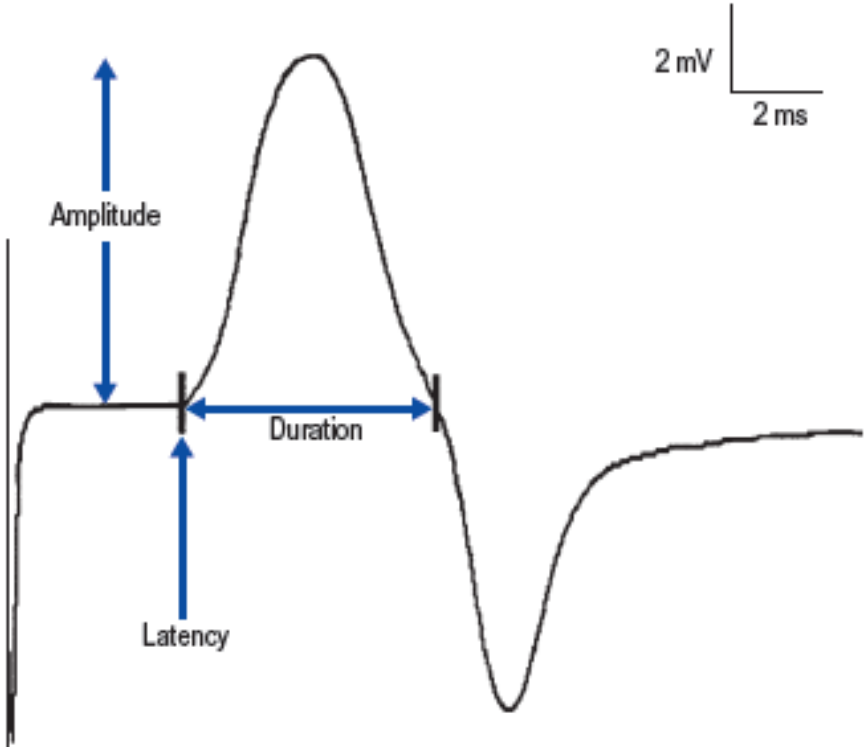
- Time is limited
- Need to be provided with key points of:
  - History
  - Examination
  - Differential diagnosis
- Hundreds or nerves and muscles could be studied:
  - Must be *individualised*

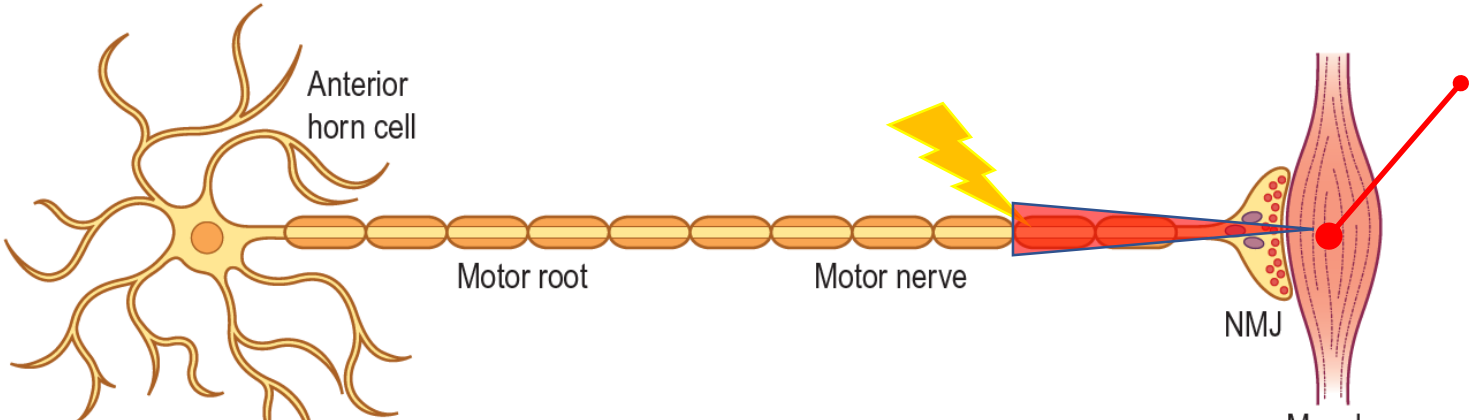
Nerve Conduction Studies



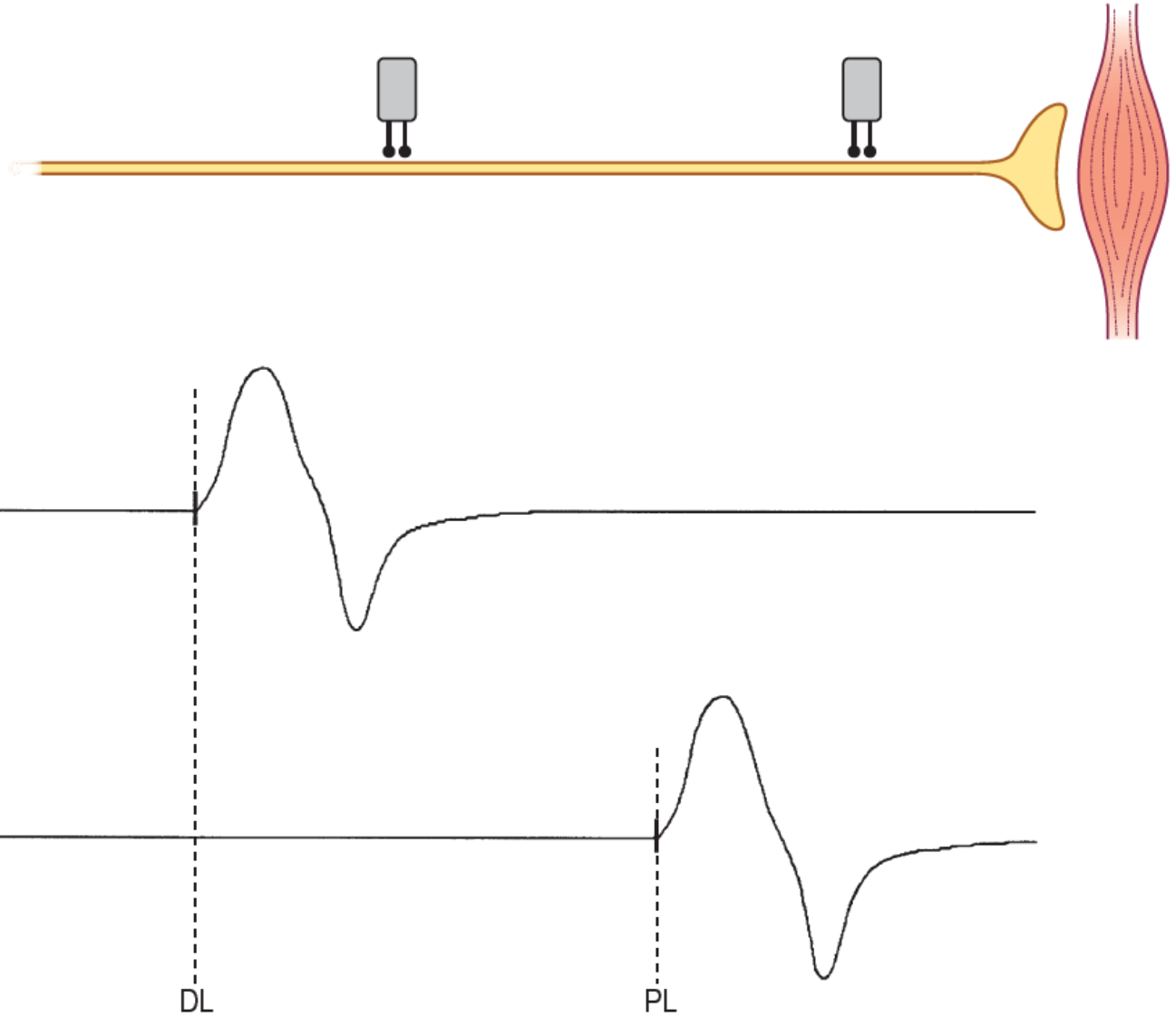
Motor nerves



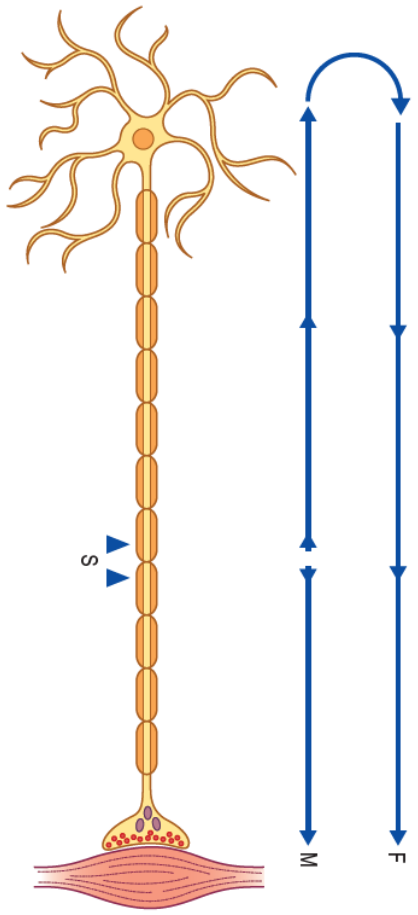


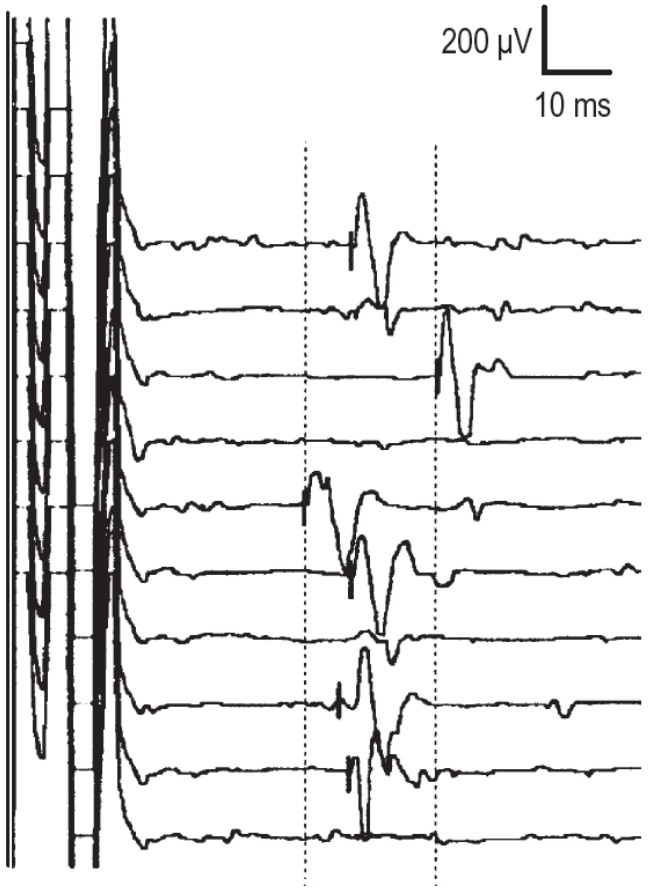


Distal latency and velocity



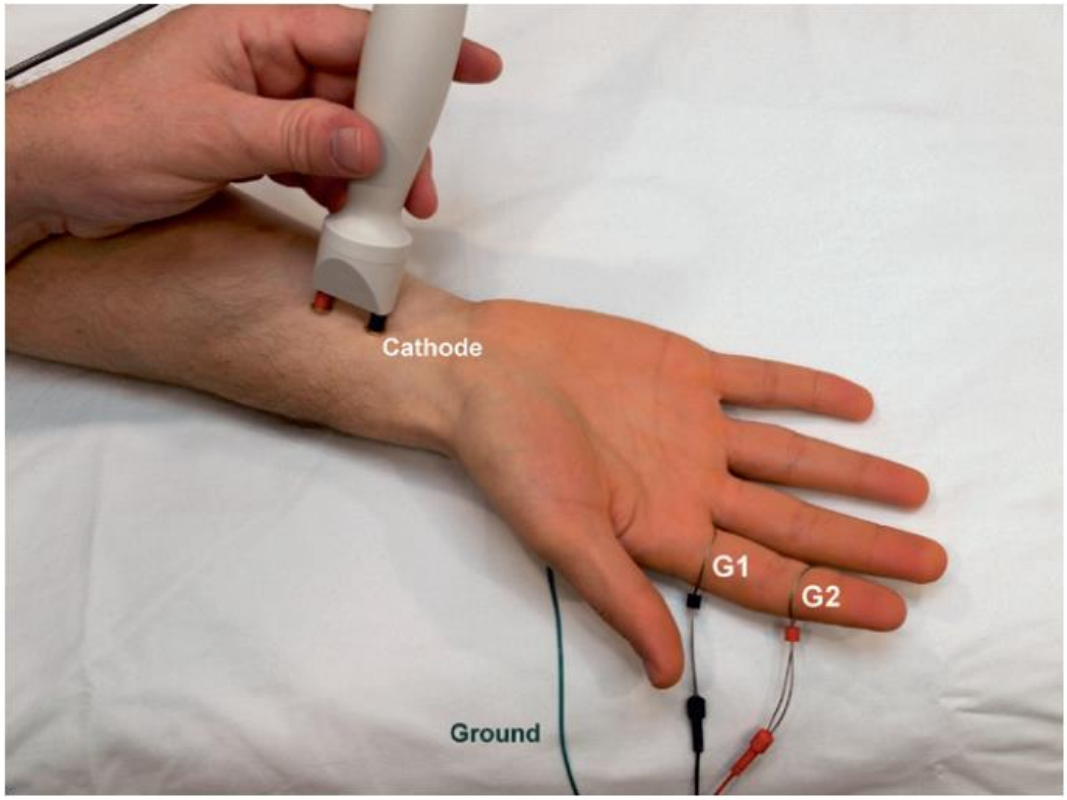
Assessing the proximal nerve – F wave responses



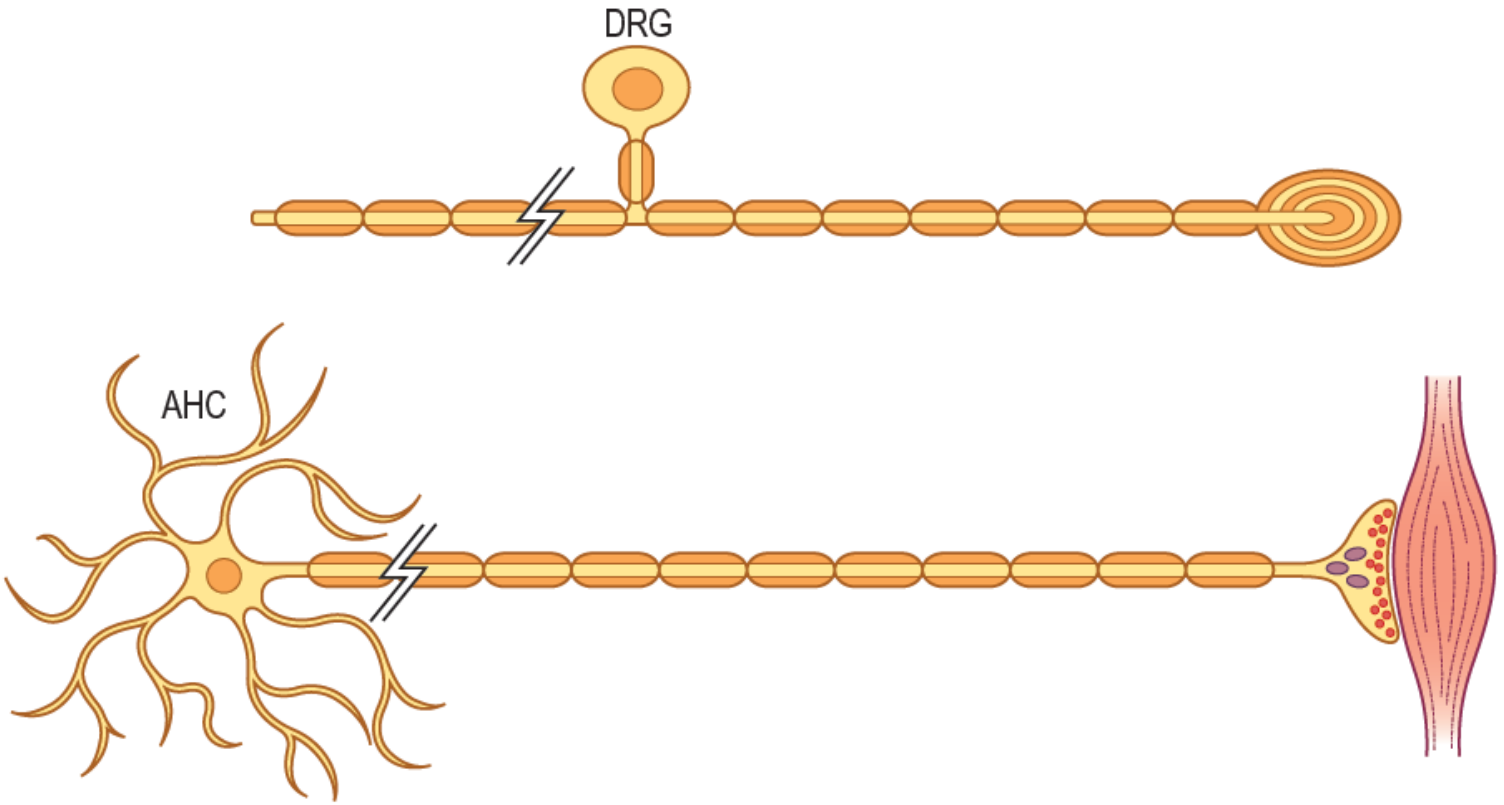


Sensory nerves

- Same principles
- More difficult to study
  - No end organ
  - Smaller amplitudes

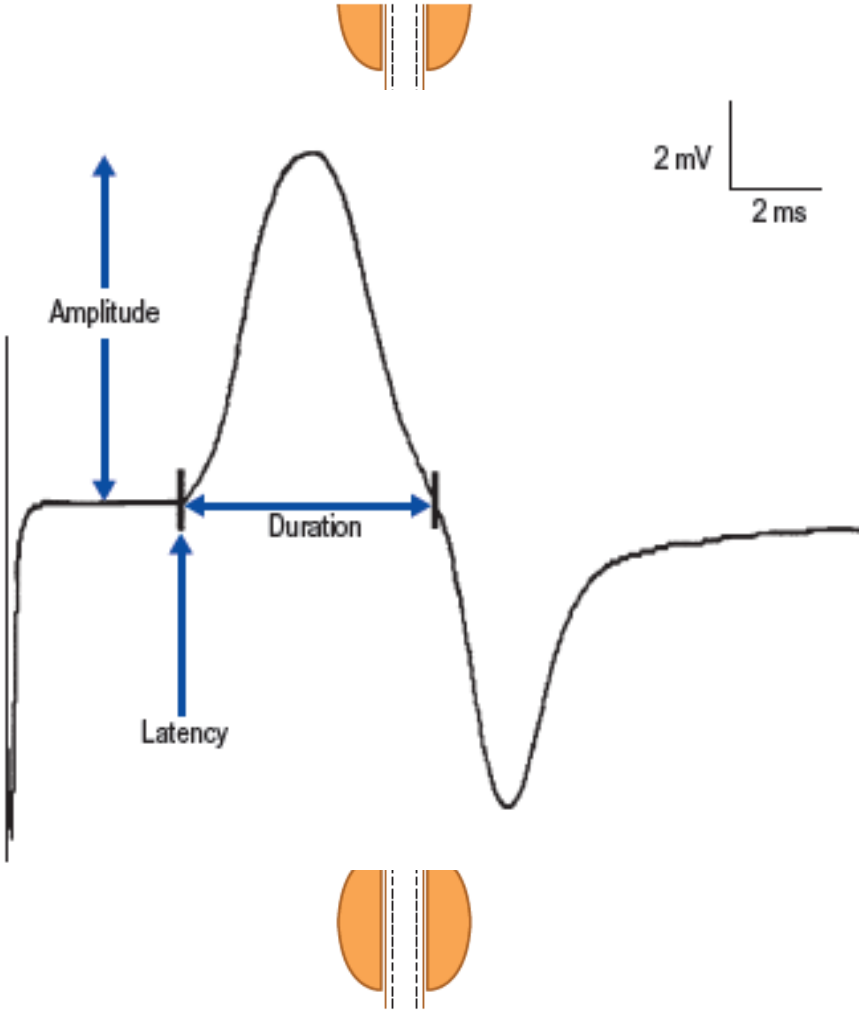


Motor *versus* sensory



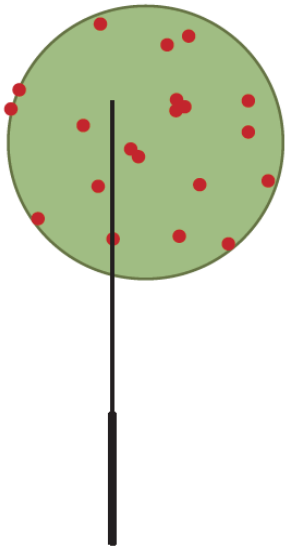
Interpretation

- Key information
  - CMAP **amplitude**
  - Conduction **velocity**
- **Axonal neuropathy**
  - Reduced *amplitude*
- **Demyelinating neuropathy**
  - Reduced *velocity*
  - Temporal dispersion

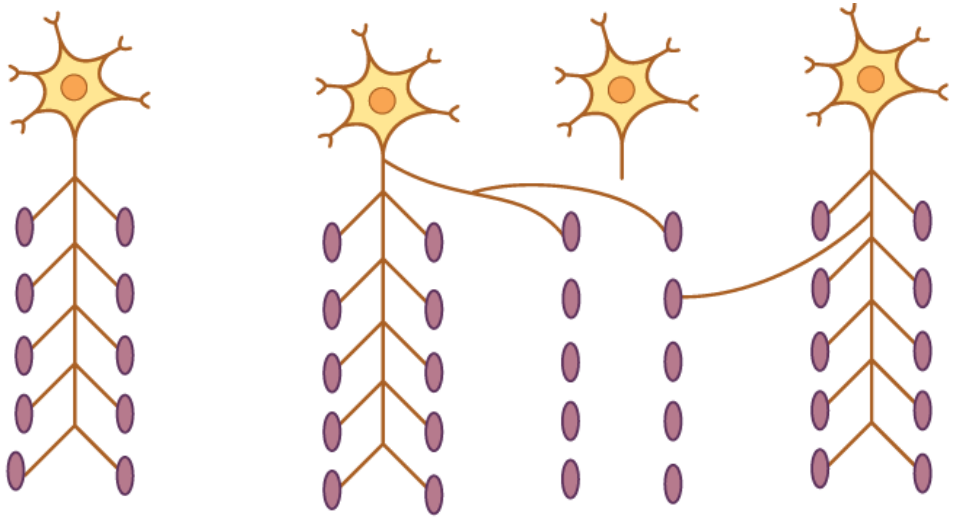


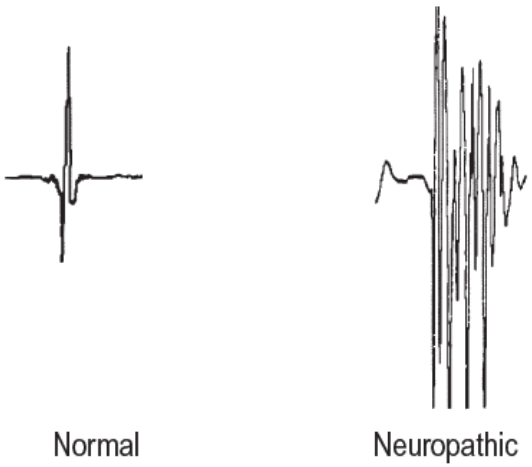
EMG

- Recording of the *motor unit action potential (MUAP)*
- But not just a test of muscle!
  - Important information about motor nerve function
- Muscles examined *at rest* and *during contraction*
- Think about anticoagulants etc.



Patterns of EMG abnormality





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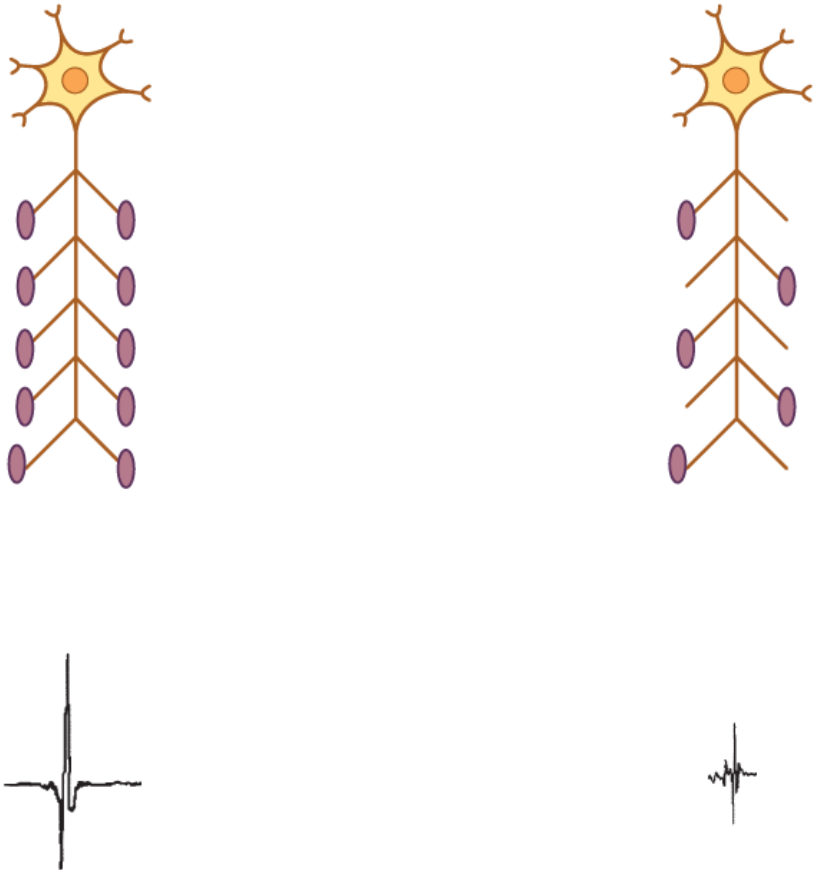


Normal

Myopathic

Putting it all together

- Formulating an electro-clinical syndrome

Demonstration

Case examples

1 - Progressive weakness

- 35 year old female
- No PMH
- Tingling in feet 2 days ago, gradually spreading up the legs
- Followed by progressive weakness in arms and legs
- Now struggling to mobilise
- Urinary retention, 1.5 L residual

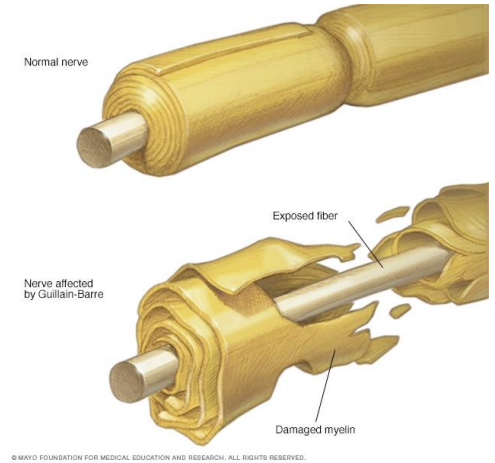
Examination

- Normal cranial nerves
- Flaccid limbs
- Can just about wiggle fingers and toes
- Areflexic
- Sensory level T4

Acute Symmetrical Limb Weakness

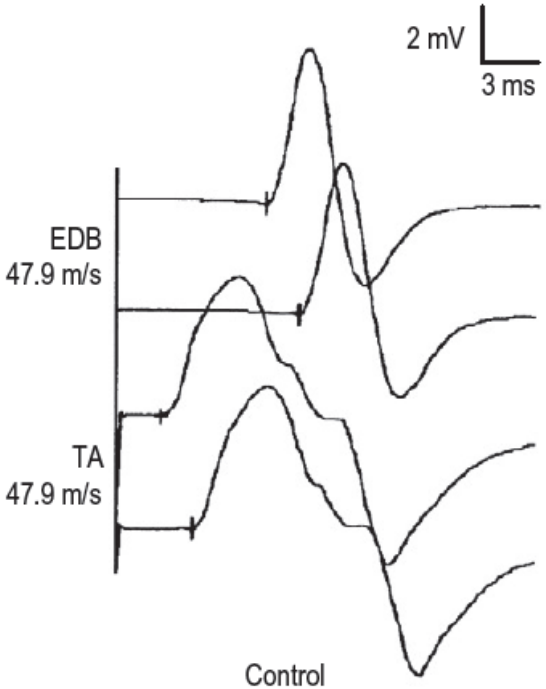
Myelopathic	•Acute transverse myelitis •Anterior spinal artery syndrome
Neuropathic	•Guillain–Barré syndrome •Toxin exposure: e.g. lead, organophosphates
Neuromuscular junction	•Myasthenia gravis/LEMS •Botulism •Iatrogenic (e.g. neuromuscular blocking agents)
Muscle	•Inflammatory myopathy •Hypo/hyper-kalaemic periodic paralysis

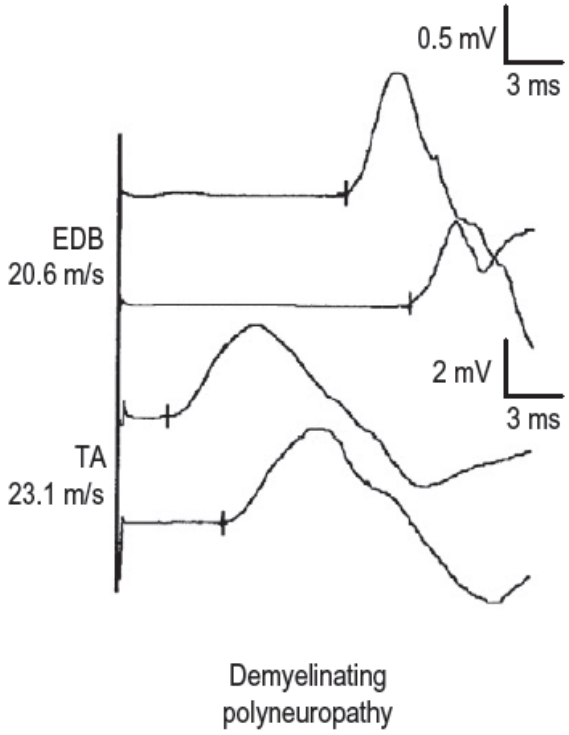
Neurophysiology



- Delayed F-waves
- Evidence of demyelinating neuropathy

- Delayed F-waves
- Prolong distal latency
- Reduced velocities
- Temporal dispersion





Outcome

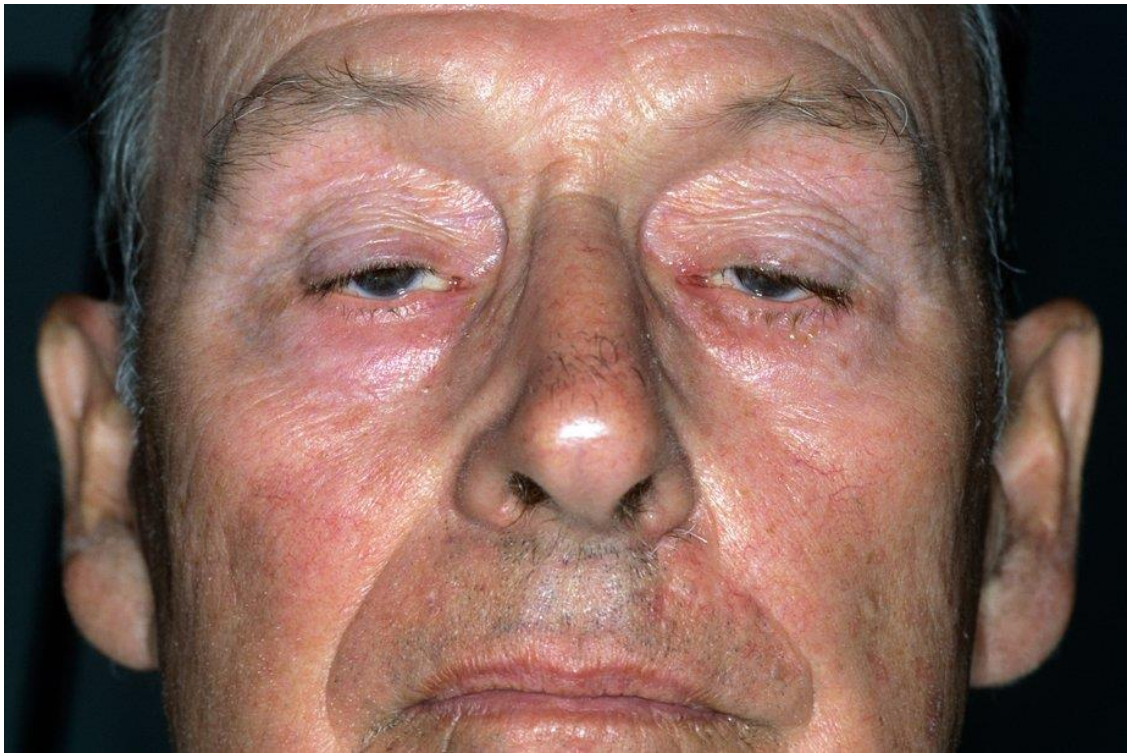
- CSF:
  - WCC<1, RBC<1
  - Protein 1.2g
  - Glucose 4.2 (6.0)
- **Diagnosis: Guillain Barre Syndrome**
- FVC 1L (peak flow normal)
- Given IVIG (2g/kg)
- Required respiratory support on ITU
- Eventually became ambulant after 6 months

2 – Eyes not moving

- 55 year old man
- 3 weeks ago – diarrhoeal illness
- Double vision, getting worse over last 2 weeks
- Voice has changed – slurring
- Feels unsteady on feet, but slower going up stairs

Examination

- Complex ophthalmoplegia, restricted in all directions
- Mild ptosis
- Bulbar dysarthria
- Mild proximal limb weakness
- Areflexic
- No ataxia



Differential diagnosis

- Myasthenia gravis
  - On close questioning, ptosis is variable, worse at end of day. Dysarthria worsens with prolonged speaking, noticed by friends on the ‘phone
- Miller Fisher syndrome
  - Usual triad of ataxia, **ophthalmoplegia and areflexia**
  - A variant of GBS – more central involvement
  - Associated with GQ1B antibodies

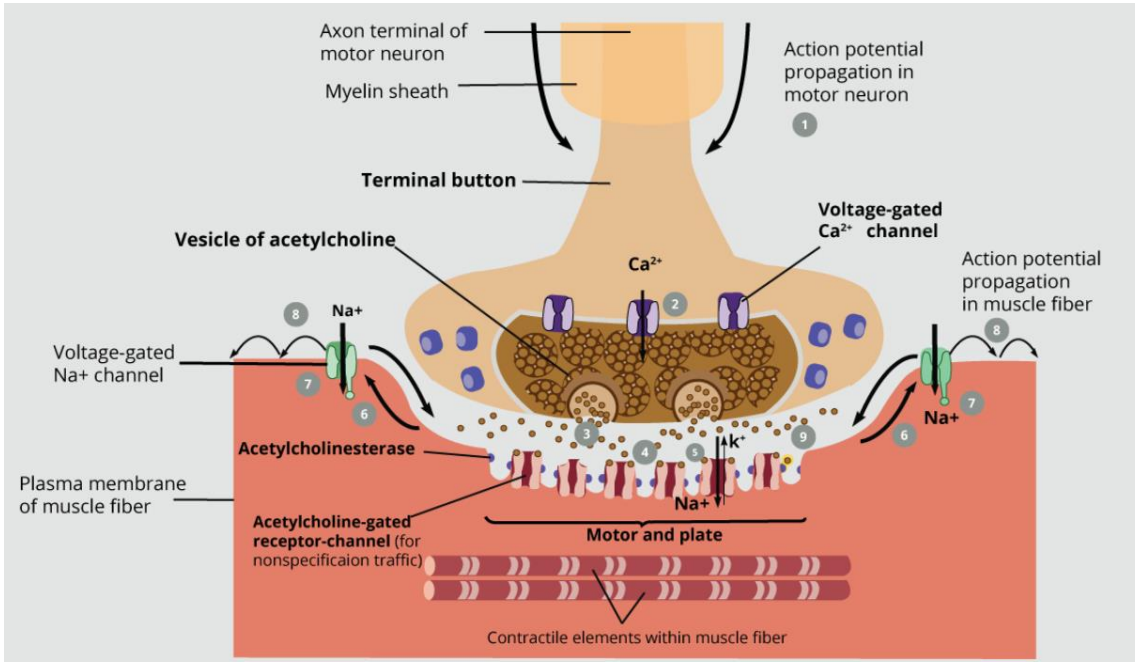
Neurophysiology

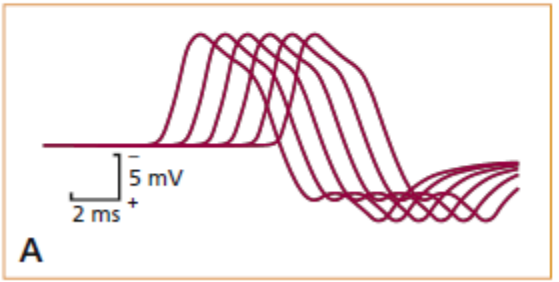
- Motor studies NORMAL
- Sensory studies NORMAL
- EMG NORMAL

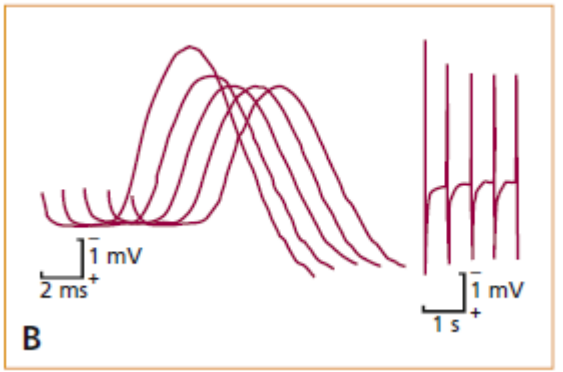
- Makes Miller Fisher unlikely
- Leave it there?

Repetitive nerve stimulation

- Neurophysiologist recognises potential for neuromuscular junction problem
- Significant decrement (>10%) identified

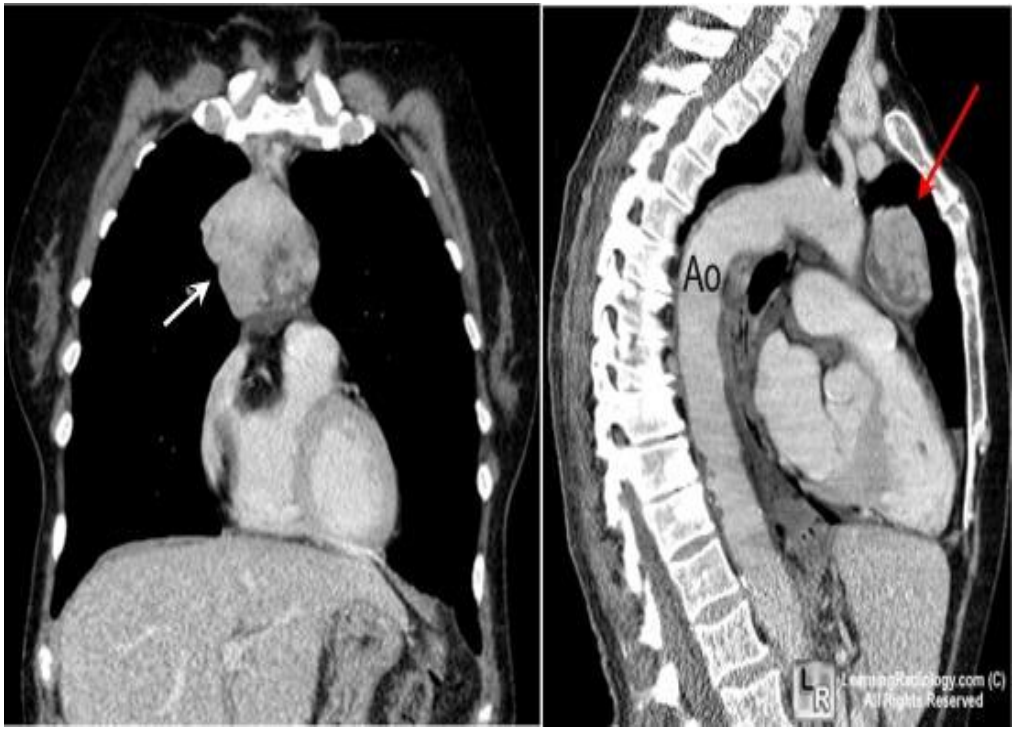






Outcome

- Diagnosis:
  Myasthenia Gravis
- Started on steroids
- CT thorax demonstrated thymoma
  - → resected
- Back to normal within 2 months. Azathioprine started

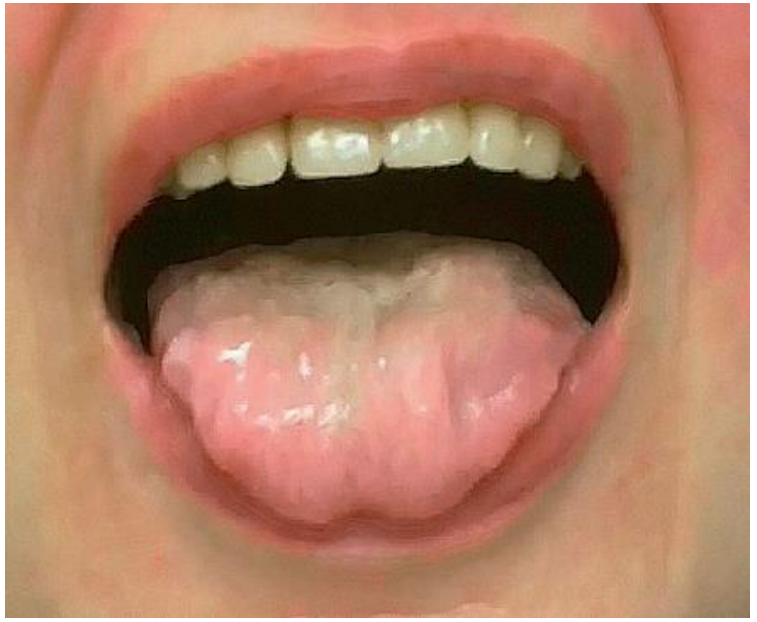


3 – Swallowing problems

- 70 year old man
- Progressive swallowing problems over 6 months
- Losing weight
- Slowing down, struggling to perform ADLs independently
- Some pain down legs
- GP checked the CK **– 900 IU/L**
- Referred in ?myopathy

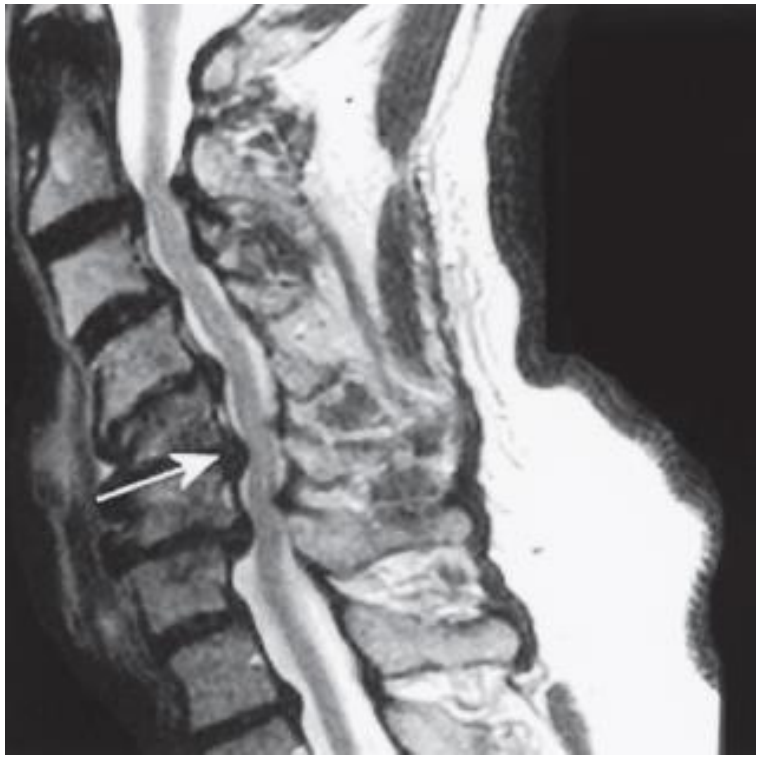
Examination

- Normal eye movements
- Tongue wasted and fasciculating
- Pseudobulbar speech, slow tongue movements, weak cough
- Widespread muscle wasting
- No fasciculations seen
- Reflexes + throughout
- Mute plantars
- Normal sensory exam



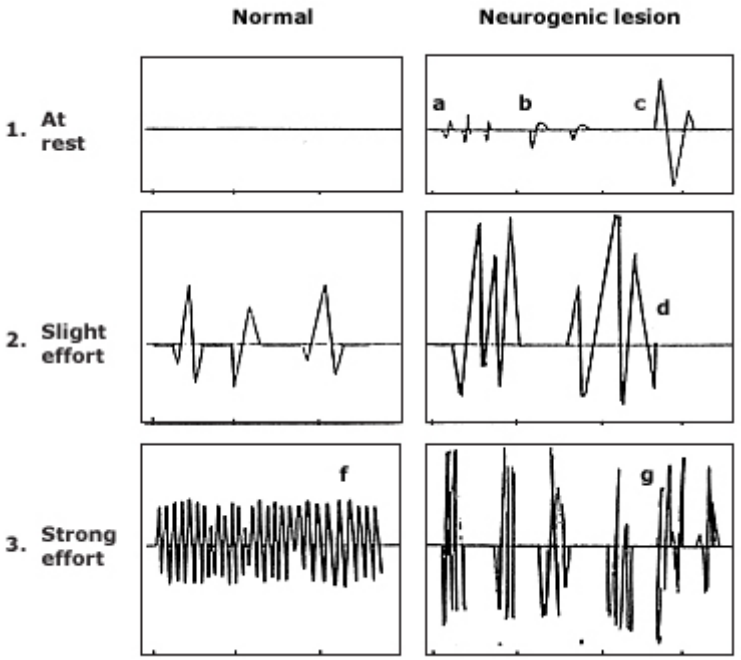
Differential diagnosis

- Brain?
  - Seems unlikely – clear LMN signs
- Spinal cord?
  - Wouldn't explain cranial nerve signs
- Neuropathy?
  - Doesn't explain the UMN signs, normal sensory exam
- Neuromuscular junction
  - No ophthalmoplegia, no fatigue
- Muscle
  - Doesn't fit – clear mix of UNM and LMN abnormality
- **Anterior horn cell**



Electrophysiology

- Normal sensory study
- Slightly reduced CMAP amplitudes
- Normal conduction velocities (and no block)
- EMG:
  - Changes in keeping with widespread acute and chronic denervation
  - Increased spontaneous activity: fibrillation potentials
  - Long-duration, high-amplitude, polyphasic MUAPs



Outcome

- Diagnosis: Motor neurone disease (ALS)
- Riluzole started
- Not a candidate for NIV
- Died 6 months later

Summary

- NCS/EMG should be viewed as an extension of the clinical examination
- Neurophysiologist should be asked to try and distinguish between clinical differential diagnoses
- A key benefit of NCS/EMG is the ability to localise neurological problems
- Final diagnosis made after consideration of the “electroclinical syndrome”
- If in doubt, discuss