Primer in Acute Neuromuscular Disease

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NHS
Northern Care Alliance
NHS Foundation Trust

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North of England Acute Neurology Update

Disclosures

James Lilleker

- Employed by the Northern Care Alliance NHS foundation Trust
- Speakers bureau, conference expenses, and/or advisory board fees from Roche, Sanofi Genzyme and Biogen

Katy Dodd

- Employed by the Northern Care Alliance NHS foundation Trust
- PhD funding from the Manchester Myasthenia Gravis Research Fund
- Research grants from Manchester neurological research fund, Myaware and the Muscle Study Group

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Overview

- Six **acute neuromuscular** cases
- **Common** presentations on the acute take
- Focus on **practical** management
- Improve your skills in:
  - Prompt and accurate diagnosis
  - Timely commencement of appropriate treatment
  - Avoidance of preventable harm

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Diagnostic Possibilities

***New* primary neuromuscular disorder**	GBS, Myasthenia Gravis, Motor Neurone Disease, Myopathy, Muscular dystrophy…
***Flare* of pre-existing neuromuscular disorder**	Myasthenia Gravis, CIDP
***Progression* of pre-existing neuromuscular disorder**	MND, Muscular dystrophy

History

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Cadence is key:

Seconds	→ vascular trauma	Mononeuritis multiplex Compressive neuropathies
Hours-days	→ inflammation infection	GBS HIV
Weeks+	→ degenerative genetic metabolic toxic malignant	MND CMT B12 deficiency Alcohol Infiltration / paraneoplastic

Examination: localise the lesion

UMN

Atrophy if chronic

Pyramidal weakness

↑ Reflexes

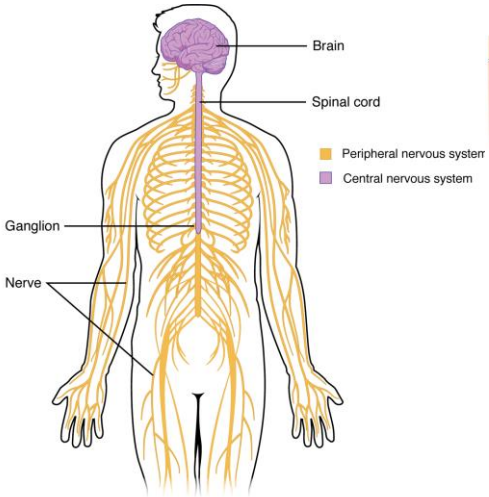
↑ Tone

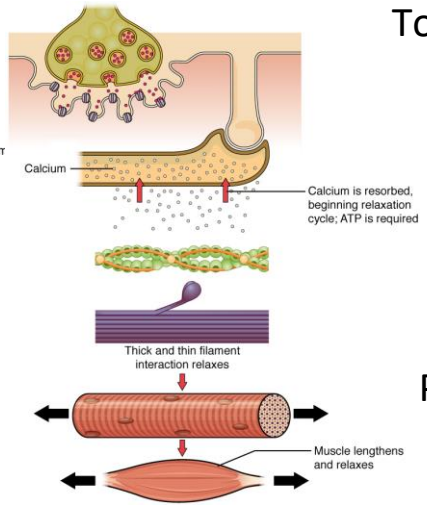
LMN

Wasting/fasciculation

↓ Reflexes

↓ Tone





NMJ

Tone and reflexes usually normal

Fatigable proximal weakness

No sensory involvement

Muscle

Tone and reflexes normal

Proximal / specific patterns of weakness

May have wasting

No sensory involvement

Examination: localise the lesion

UMN

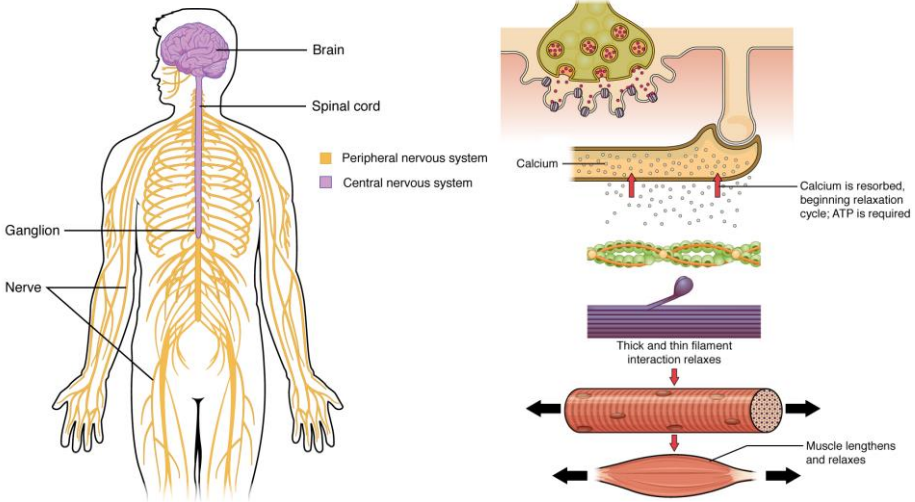
MND

LMN

GBS

CIDP

Nutritional



NMJ

MG

LEMS

Botulism

Muscle

Inflammatory

Dystrophic

Mrs A presents to you complaining of new weakness and numbness in her limbs

60 year old nurse. Usually fit and well.

“Over the past week I have becomes weak and numb in my arms and legs.”

On Examination:

Cranial nerves normal

Bilateral arm and leg weakness:

MRC 4 / 5 in a pyramidal pattern

Reduced pinprick sensation over legs and arms in a patchy distribution

Areflexic, plantars mute

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Where is the lesion?

1. Brain
2. Spinal cord
3. Peripheral nerve
4. Neuromuscular junction
5. Muscle
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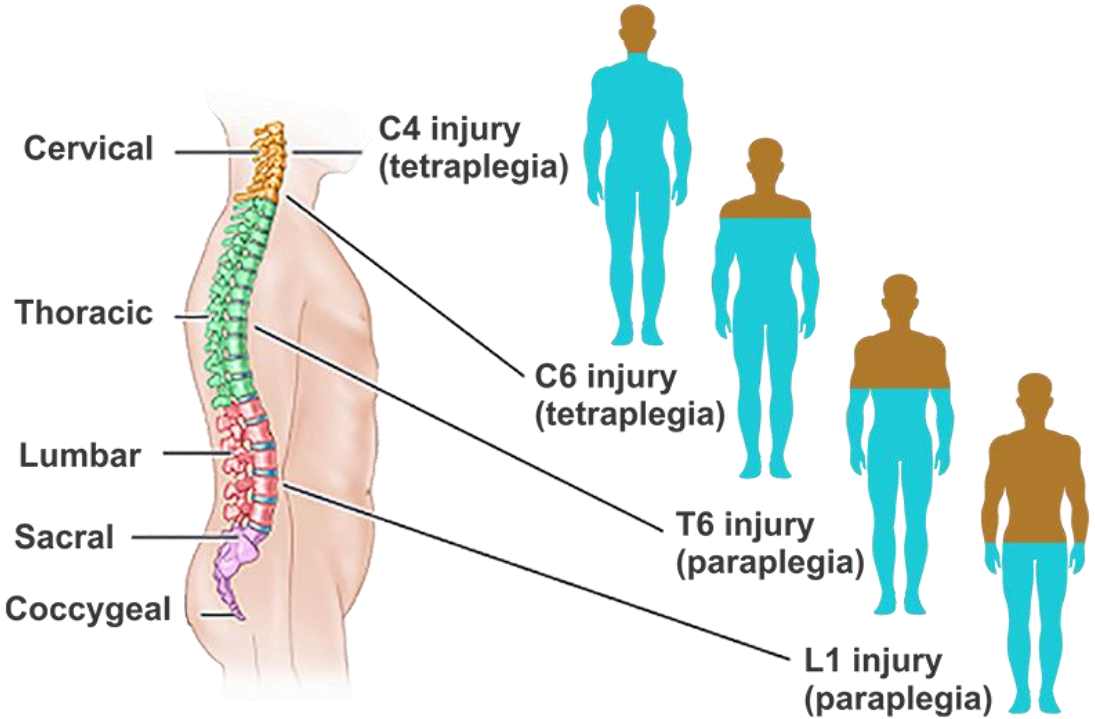
UMN lesions = pyramidal weakness

But, LMN lesions can cause pseudopyramidal weakness

UMN lesions = spasticity

But, acute spinal cord injury can cause “spinal shock” - reduced tone and reduced/absent reflexes

Spinal cord lesions usually have a sensory level on the torso



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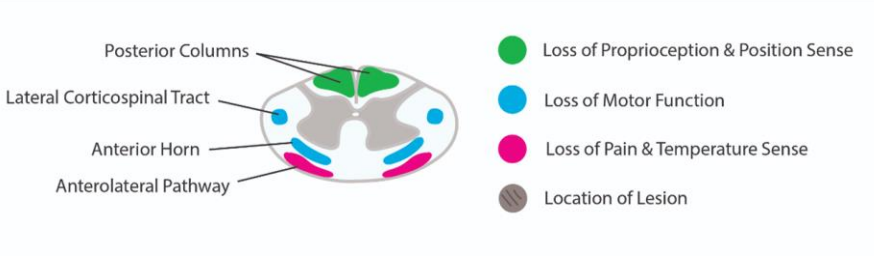
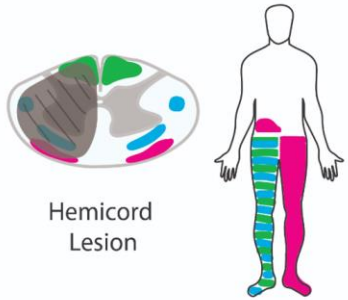
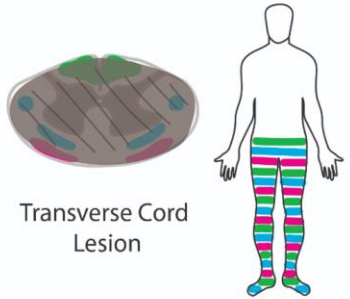
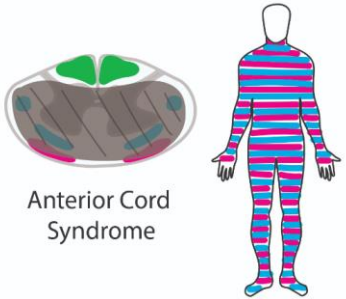
http://www.escif.org/spinal-cord-injury/

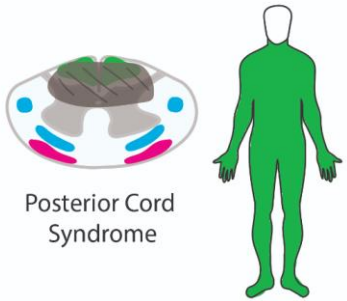
Cord vs. Peripheral Nerve:
Sensory Changes

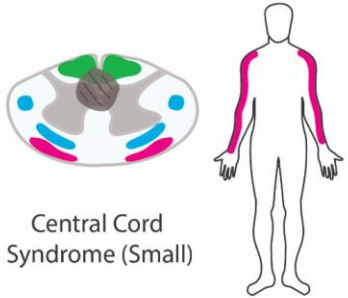


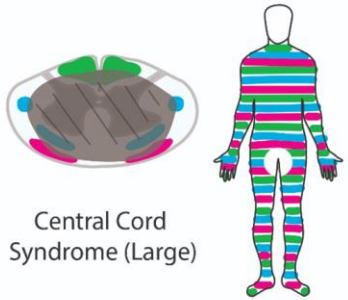












https://www.statpearls.com/ArticleLibrary/viewarticle/19123

Where is the lesion?

1. Brain
2. **Spinal cord**
3. **Peripheral nerve**
4. Neuromuscular junction
5. Muscle
6. Don’t know - need more information

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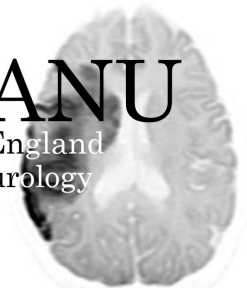

What is the lesion?

1. Vascular
2. Trauma
3. Inflammation / infective
4. Metabolic
5. Genetic
6. Degenerative
7. Toxic
8. Malignancy
9. Don’t know - need more information

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Mrs A presents to you complaining of new weakness and numbness in her limbs

60 year old nurse. Usually fit and well.

"Over the **past week** I have becomes weak and numb in my arms and legs."

On Examination:

Cranial nerves normal

Bilateral arm and leg weakness:

MRC 4 / 5 in a pyramidal pattern

Reduced pinprick sensation over legs and arms in a patchy distribution

Areflexic, plantars mute

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What is the lesion?

1. Vascular
2. Traumatic (cord compression)
3. Inflammation / infective
4. Metabolic
5. Genetic
6. Degenerative
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8. Malignancy
9. Don’t know - need more information

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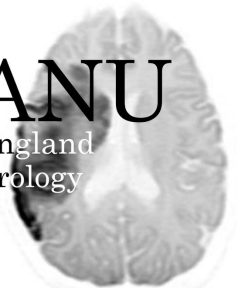

Mrs A presents to you complaining of new weakness and numbness in her limbs

We have deduced that she likely has:

- An inflammatory / infective process
- In the peripheral nerves
- However a spinal cord lesion is possible… (MRI C-spine should be considered)

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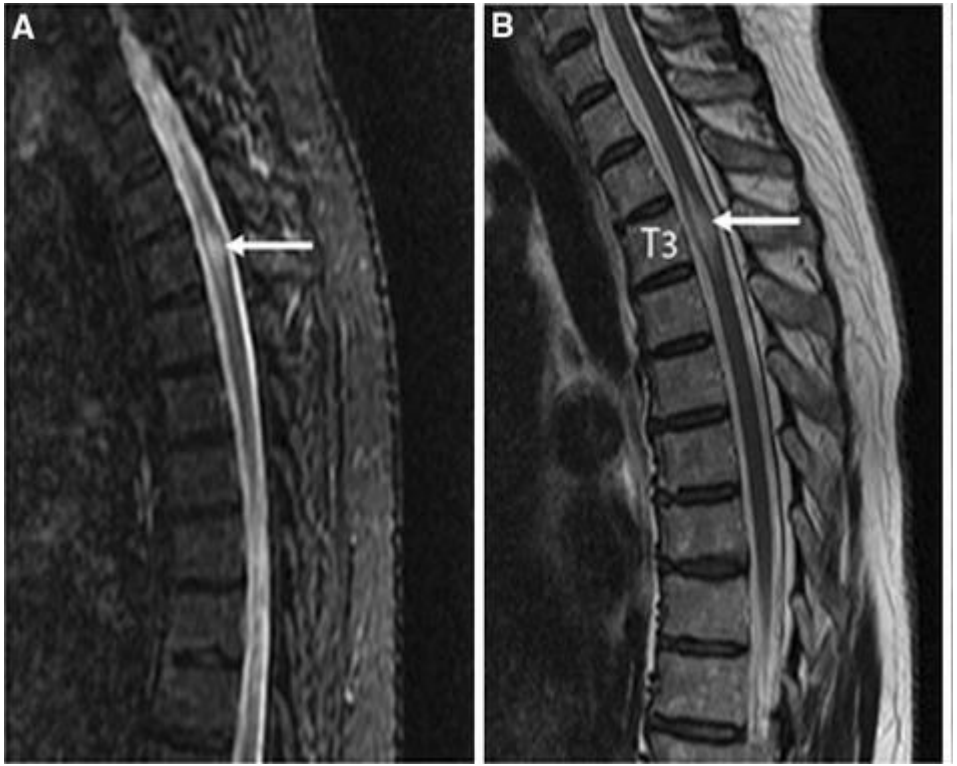
Transverse myelitis

Inflammation in spinal cord

Autoimmune (e.g. MS) or post-viral

Comes on over a few days

Usually affects younger people

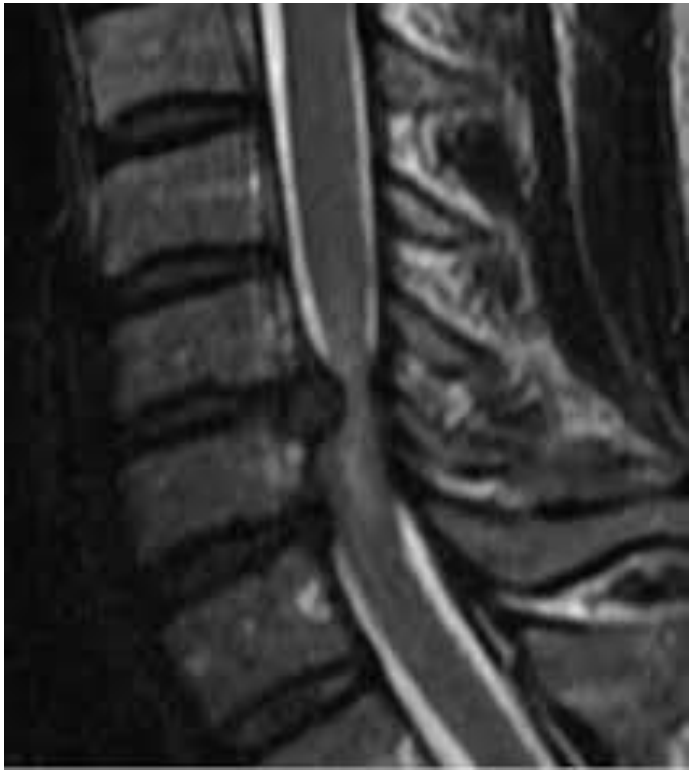


Spinal cord compression

May be sudden or slow

May have history of trauma / pain

Often in older people



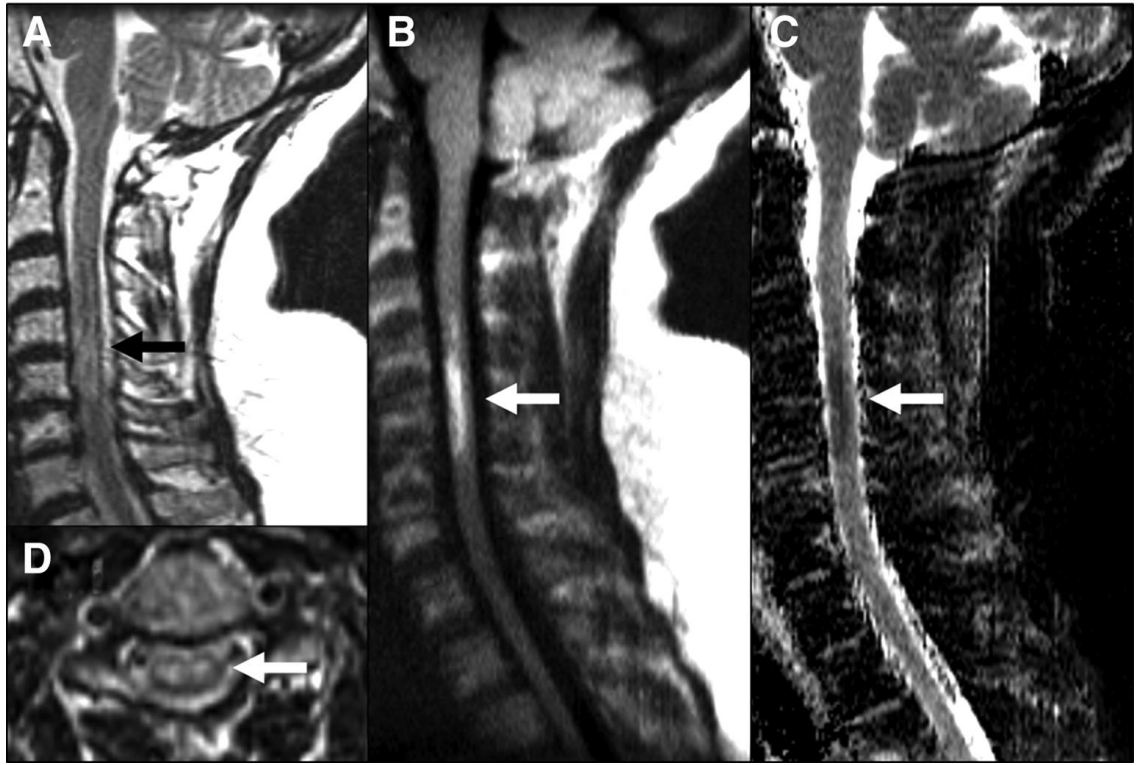
Spinal cord infarct

Sudden onset

Anterior spinal artery - proprioception and vibration preserved

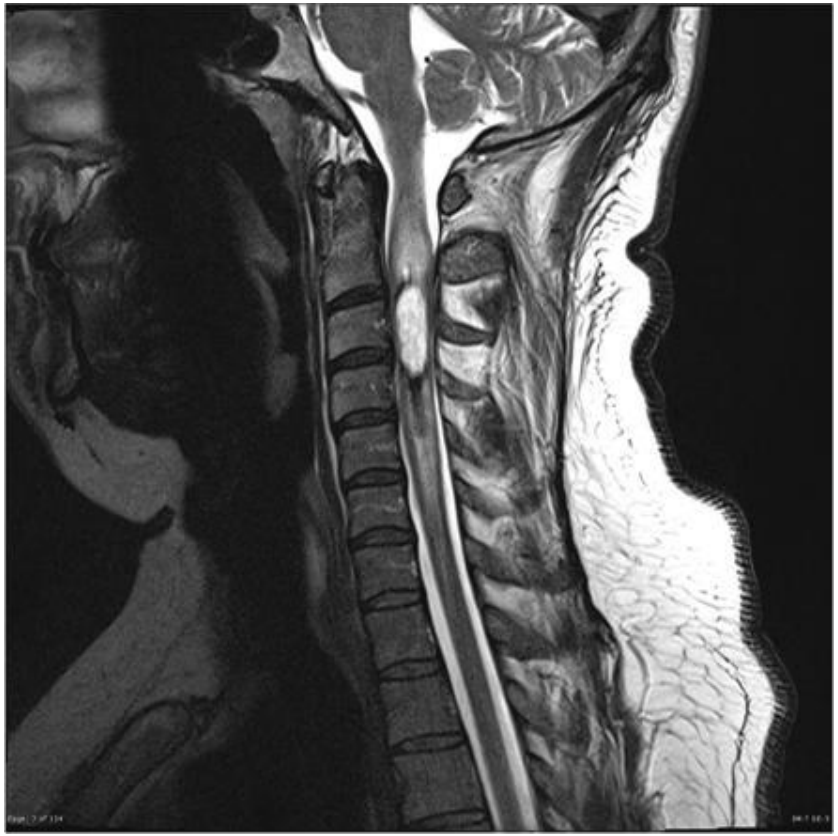
Vascular risk factors /embolic

Poor prognosis



Spinal cord tumour

Slowly progressive symptoms



Metabolic Myelopathy

Posterior cord - vibration and proprioception

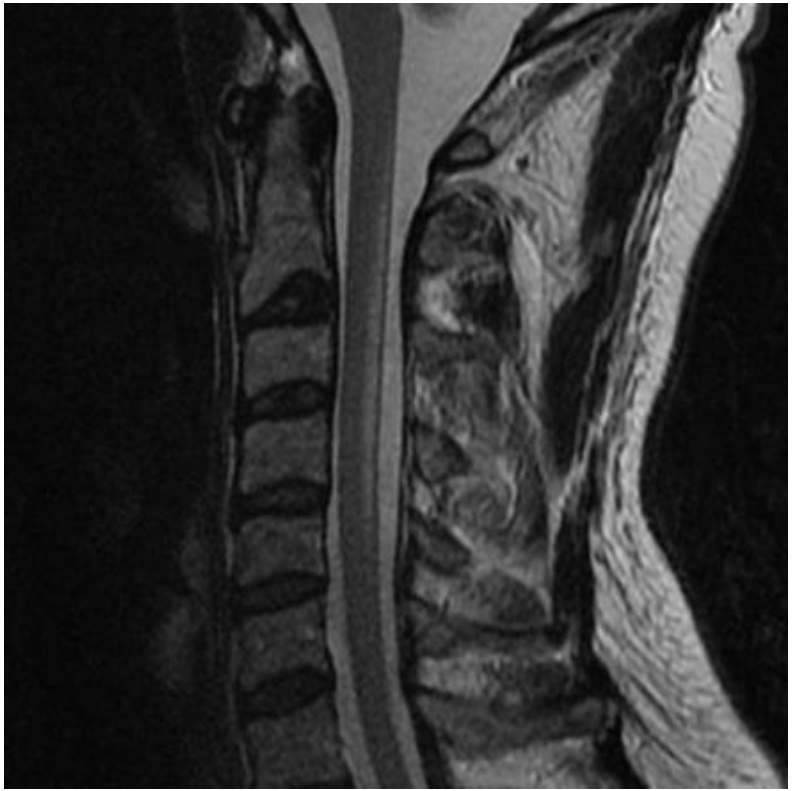
E.g. low B12 (inc. N_2O abuse) / low copper

Onset over weeks-months

Vegan?

Nitrous oxide use?

Zinc supplements?



Where is the lesion?

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3. Peripheral nerve
4. Neuromuscular junction
5. Muscle
6. Don’t know - need more information

MRI cervical spine is normal

What is the lesion?

- Inflammatory / infective

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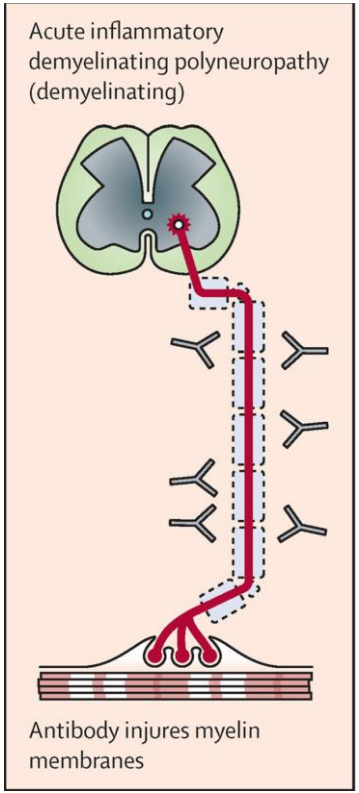
Diagnosis: Probable AIDP - Acute Inflammatory Demyelinating Poly(radiculo)neuropathy (Guillain-Barre Syndrome)

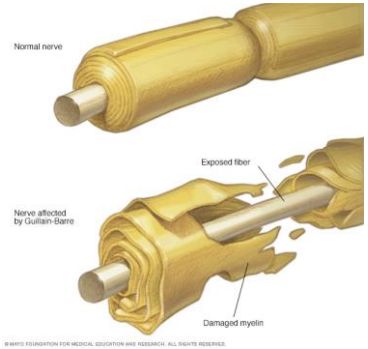
Management

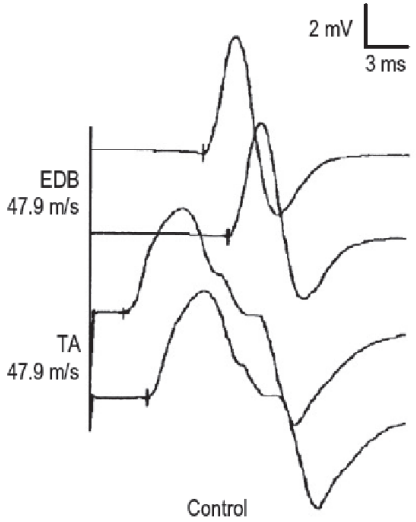
- Lumbar puncture: raised protein, exclude infection (CSF WCC, EBV + CMV PCR)
- Serum HIV
- Monitor respiratory function:
  - 4 hrly FVC or breath count
- Screen for autonomic disturbance
  - History, L/S BP, ECG
- DVT prophylaxis
- Assess swallow
- Physio/OT
- IVIG / PLEX
- NCS - 2 weeks after onset

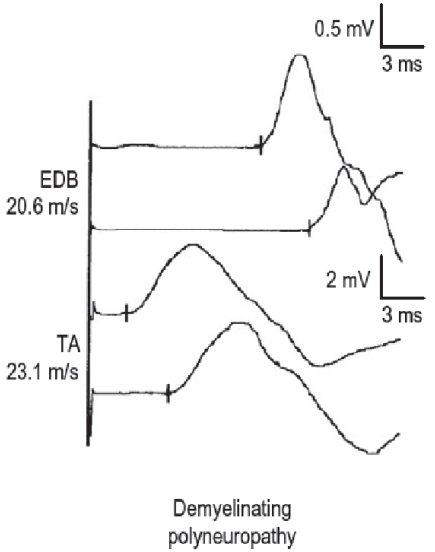


Neurophysiology in GBS

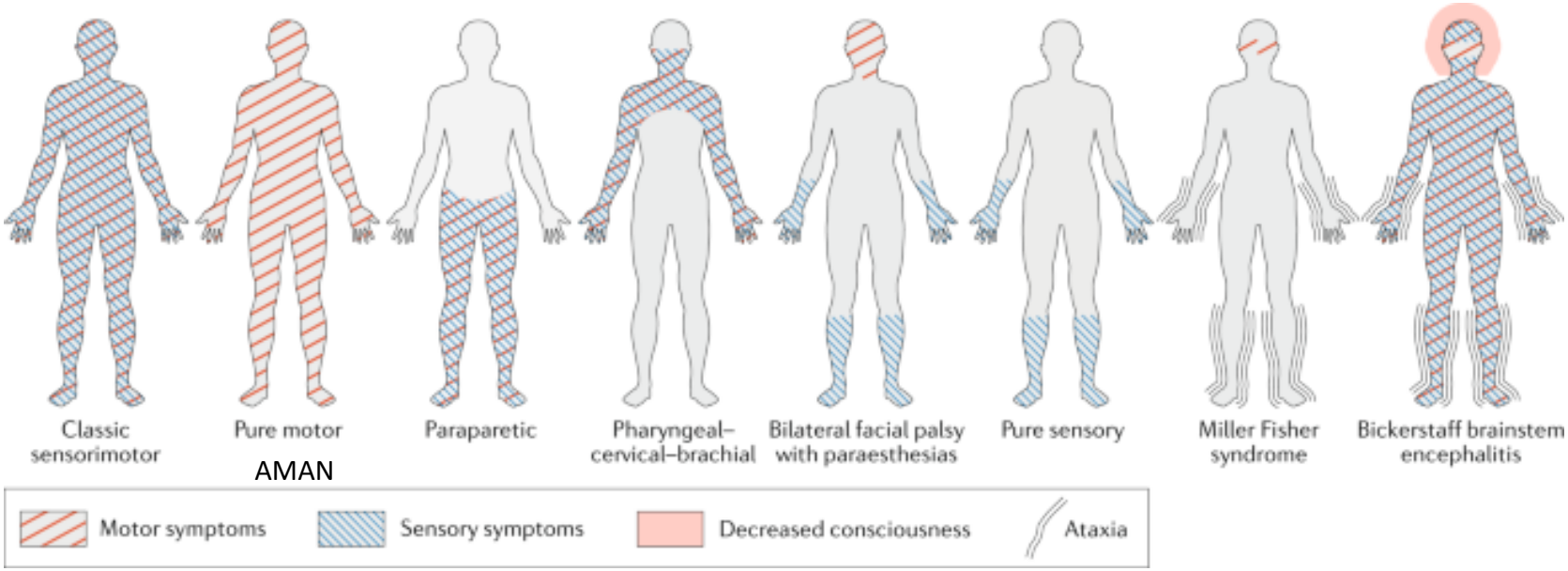
- Delayed F-waves
- Prolonged distal latency
- Reduced velocities







GBS phenotypes



Ganglioside Antibodies:

GM1
GD1b
GD1a
GQ1b
GT1a
GQ1b
GT1a
GT1a

Peripheral Nerve Differential Diagnosis of GBS

Vascular	**Mononeuritis multiplex**	Asymmetrical, multiple individual nerves, painful, renal involvement
Compressive	**Compressive neuropathies / HNPP**	Asymmetrical, individual nerves, history of unusual positioning
Inflammation	**CIDP**	Progression of symptoms over 3 months
	Multifocal motor neuropathy	Asymmetrical, motor only, typically finger drop
Metabolic	**Critical care neuropathy / myopathy**	After ICU stay
	Nutritional / toxic neuropathy	Alcohol excess / poor nutrition or medications / chemo
	Subacute combined degeneration of the cord	B12 deficiency, Nitrous oxide use (functional B12 deficiency)
Infiltrative	**Amyloid**	Enlarged tongue, diarrhoea, heart failure, autonomic dysfunction
	Lymphoma	Weight loss, night sweats, lymphadenopathy
Genetic	**Charcot Marie Tooth**	Family history, high arches, hammer toes

IVIG / Plasma Exchange on a medical ward

IV Immunoglobulins

- Serum immunoglobulins - low IgA - anaphylactoid reaction
- Thrombosis risk - review vascular risk factors
- Allergic risk - careful up titration of rate of infusion
- Approval required
- 0.4mg/kg / day for 5 days
- Consent

Plasma Exchange

- Requires central line (usually)
- Need to prescribe / order albumin
- 200-250ml/kg for 5 sessions

General Principles

Blood products (HIV, Hep B+C)

Not required in mild illness

Takes weeks to work

Equal efficacy, different risks

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GBS: *Prognosis*

- ~25% require artificial ventilation
- Mortality ~5% in high income countries
- ~20% unable to walk unaided at 6 months
- Predictors of poor outcome: advanced age, need for ventilation, preceding campylobacter, axonal sub-type
- Second doses of IVIG/PLEX not thought to be beneficial

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Mrs B is admitted complaining of swallowing difficulty…

60 year old vet, usually fit and well

"I've been **struggling with my swallowing** for a few months, but it is getting worse. When I try to swallow I choke and liquid can come **out of my nose.**"

Examination reported to you:

Speech sounds slurred

Thin arms and legs

Reflexes brisk

Sensation normal

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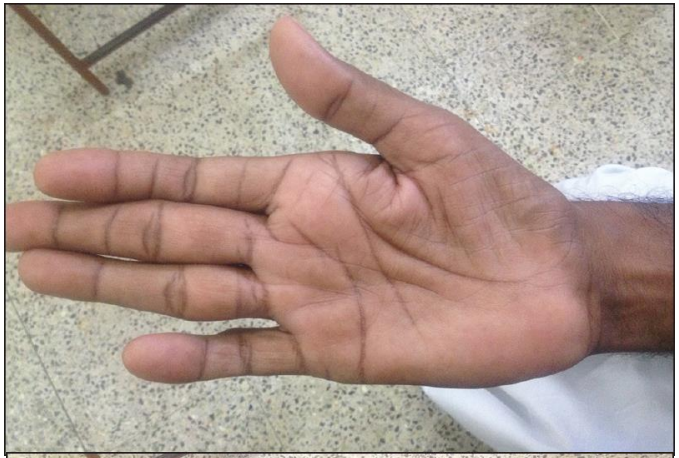
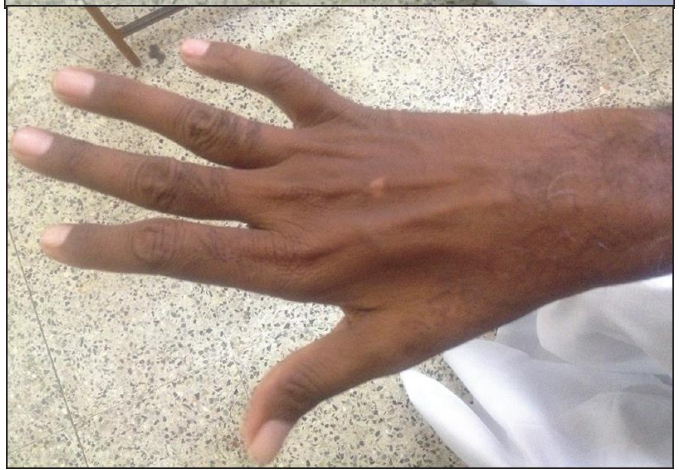
You re-examine Mrs B





Examination

No ptosis or double vision

Tongue not wasted / fasciculating, moves slowly

Speech abnormality

No fatiguability

Muscle wasting in the hands

Fasciculations in periscapular muscles

Weakness of ankle dorsiflexion on the right

Reflexes brisk, plantars upgoing

Sensation normal

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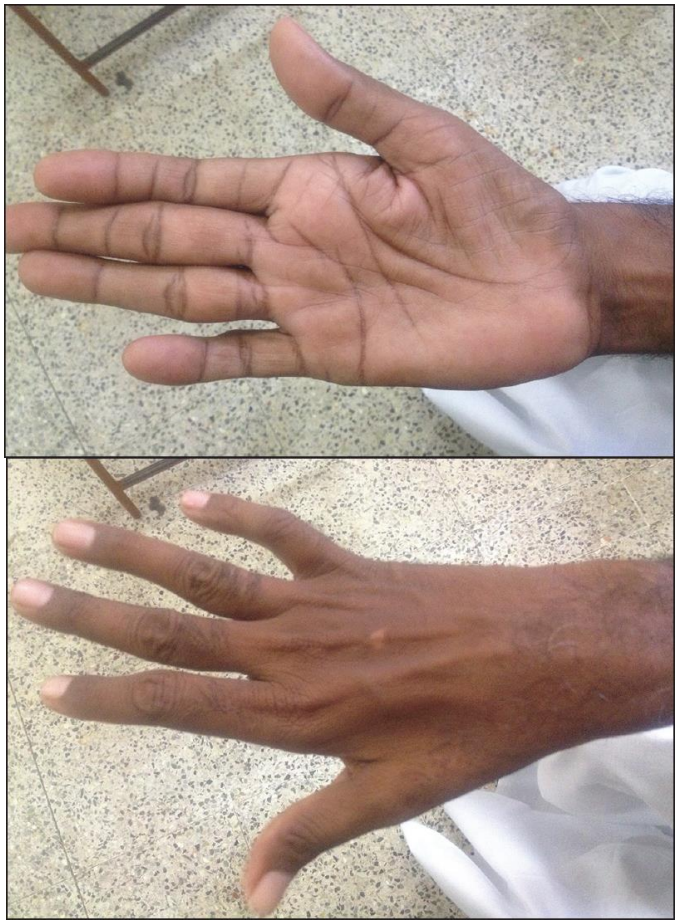
Mixed UMN and LMN, no sensory:



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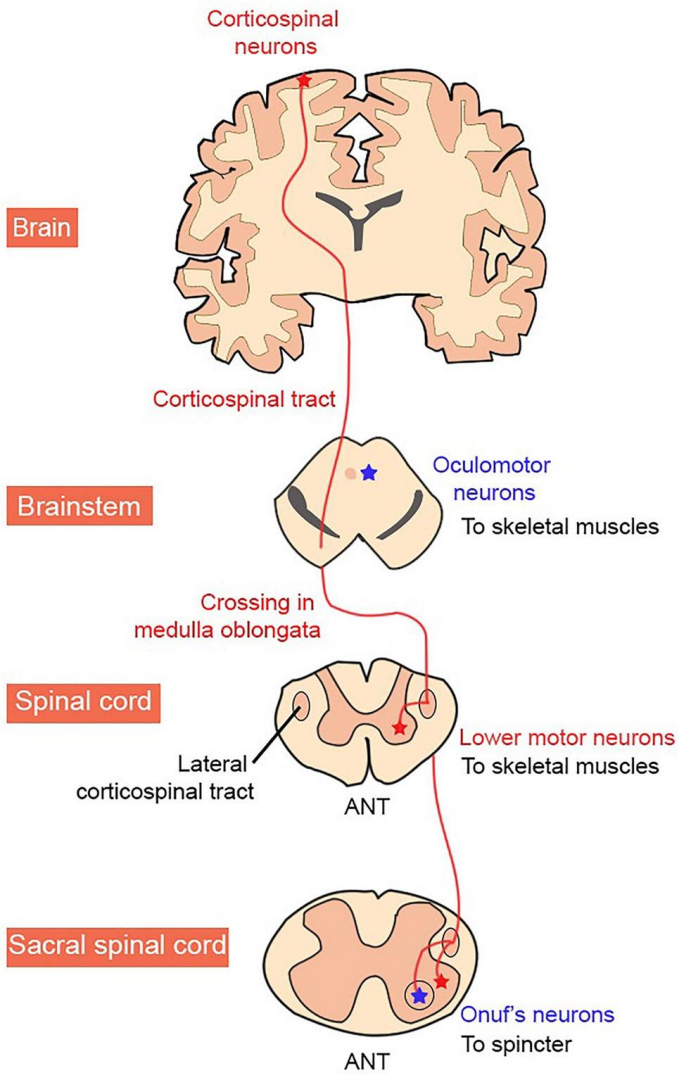
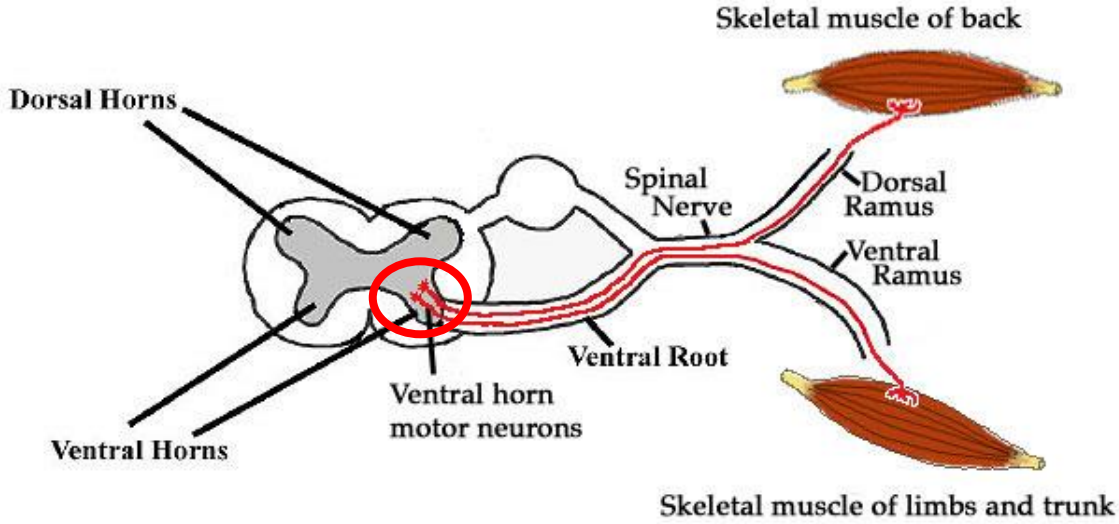
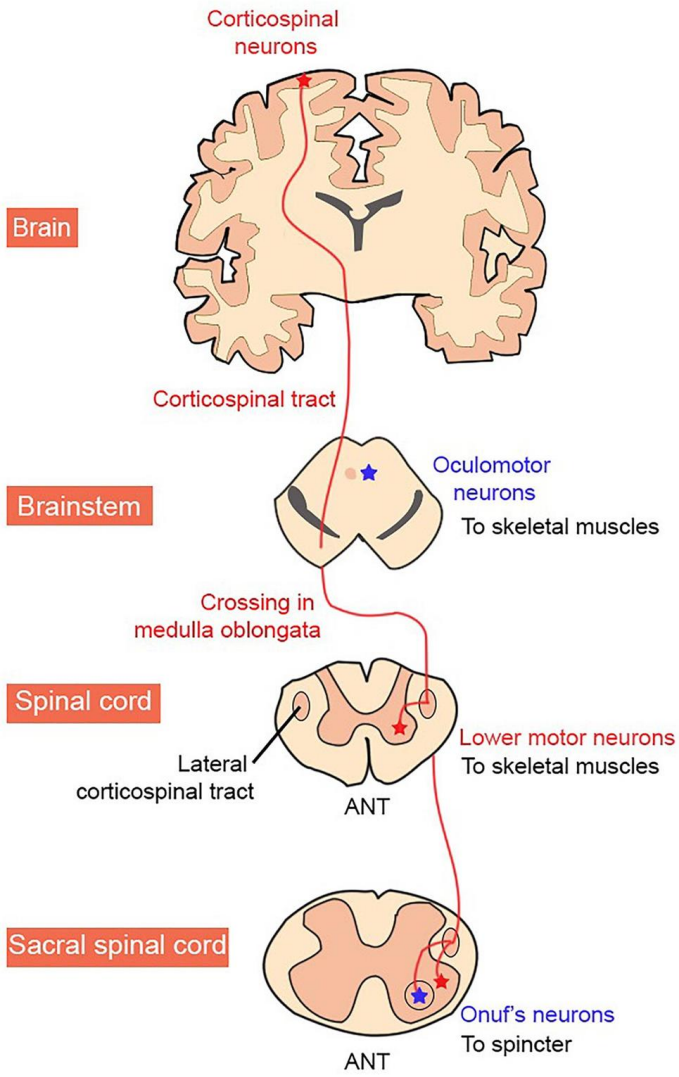


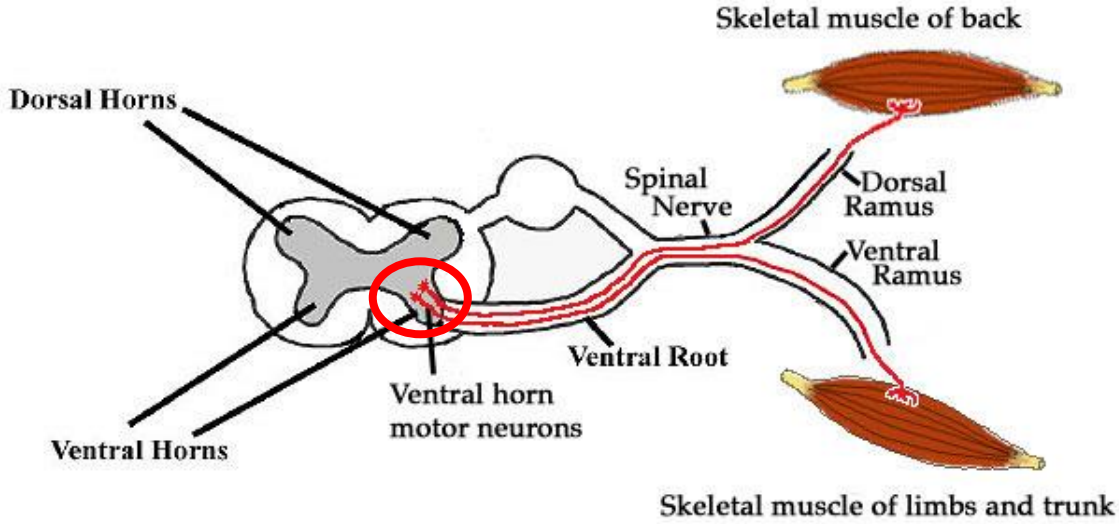
Where is the lesion?



Aetiology?



Where is the lesion? Anterior Horn Cells



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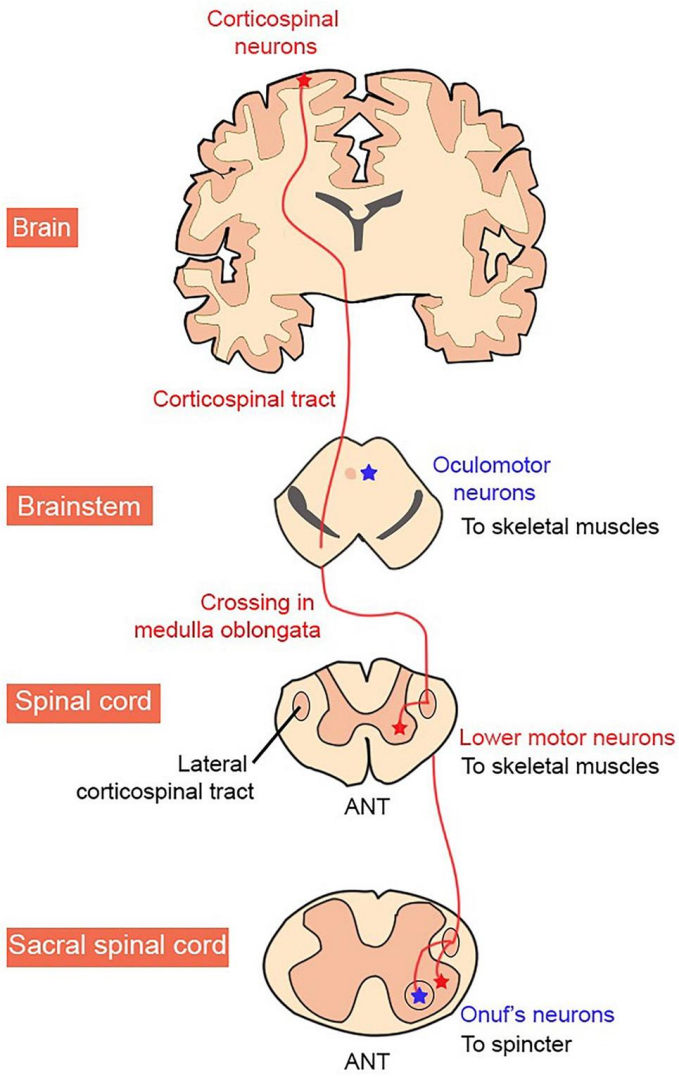
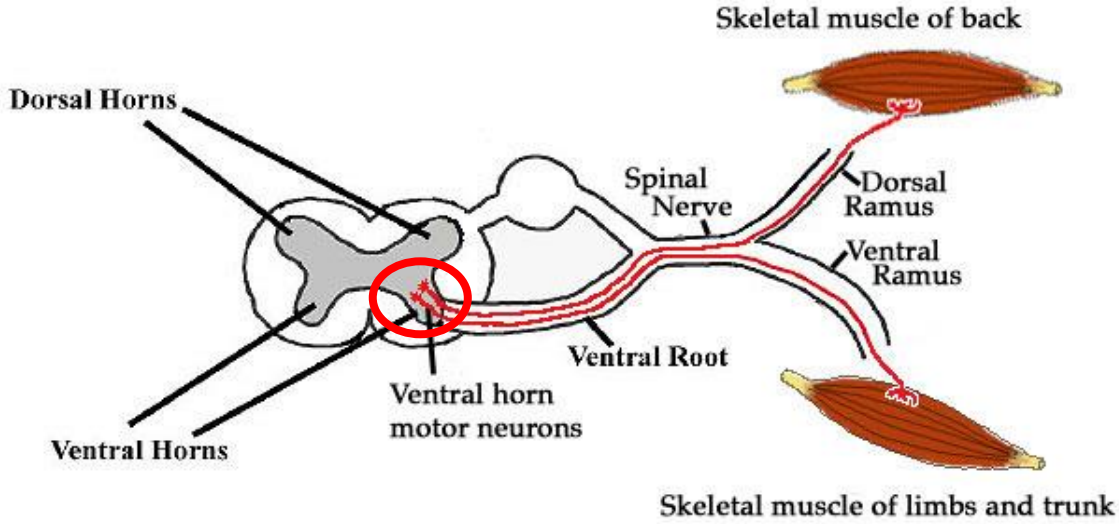


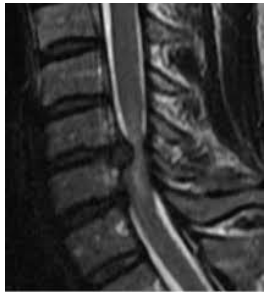
Where is the lesion? **Anterior Horn Cells**



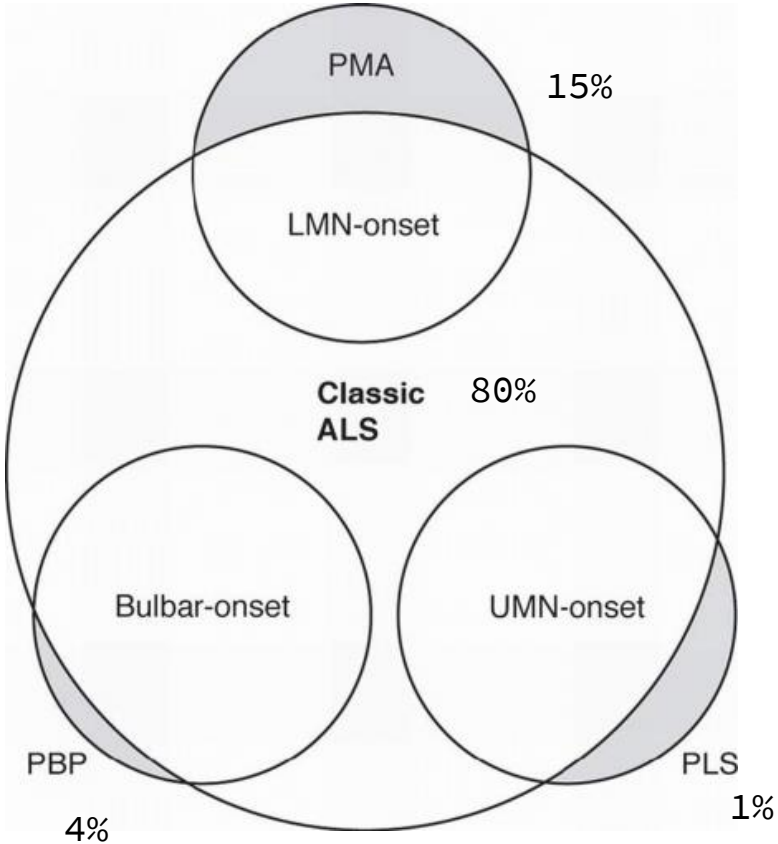
Aetiology? **MND**

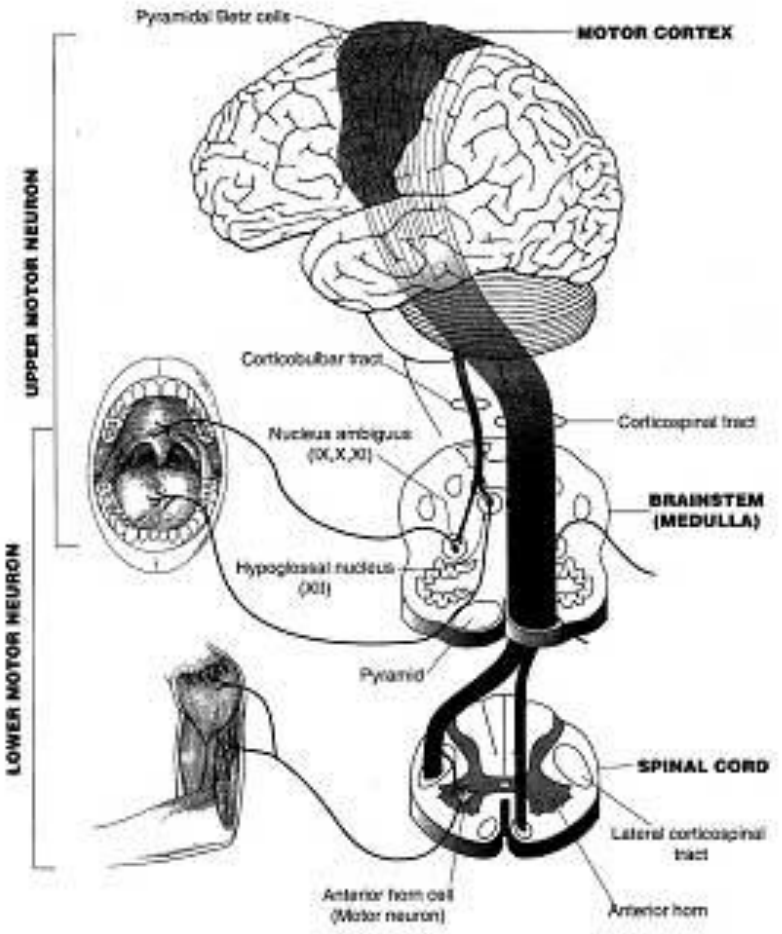
Differential diagnosis of MND:
Don't miss something treatable!

Domain	Diagnosis	Why not?
Genetic	Spinal muscular atrophy	Too rapid, Too old
	Kennedy's disease	Female, UMN features
	Hereditary spastic paraparesis	Upper limb and bulbar involvement
Cord disease	**Compressive cervical myelopathy + lumbar radiculopathies**	No sensory involvement
Myopathy	Inclusion body myositis	UMN signs Wrong pattern of weakness
	Immune mediated necrotising myopathy	UMN signs Wrong pattern of weakness
Nerve	**Multifocal motor neuropathy**	UMN signs



MND subtypes



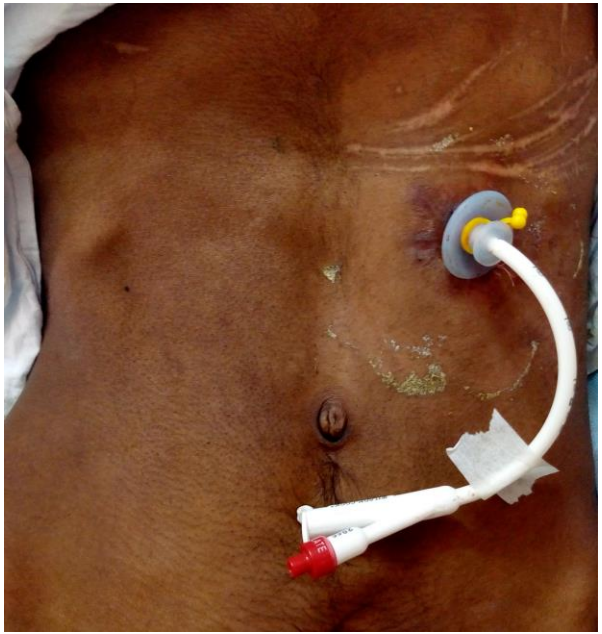


What shall we do?

1. **Confirm the diagnosis:**
    a. Electrodiagnostic testing
    b. MRI brain and cervical spine?
    c. Genetics?
2. **Involve the MND team early**
3. **Manage the problems:**
    a. Swallowing - SALT and dietician review ? NG / PEG
    b. Respiratory function - morning blood gas, overnight oximetry - ?NIV
    c. Therapist review
    d. Riluzole - check LFTs
    e. Secretions - hyoscine patch / atropine drops

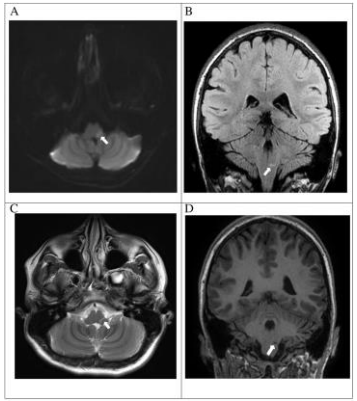
Nutrition and Respiratory Function

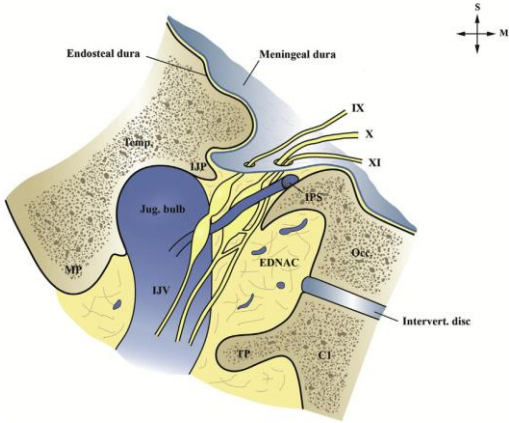
- **Supportive care** has a big impact on life expectancy
- Needs to be **personalised**
- Co-existing **FTD** can make decisions complex
- **Nutrition**
  - Predict bulbar failure
  - PEG / RIG insertion
  - Can be risky, esp if respiratory failure
- **Respiratory** function
  - Secretion management and aspiration risk
  - Screen for respiratory failure
  - Consider trial of NIV
  - When to stop?



Approaching dysphagia

- History - *is it neurological?*
  - Cadence of onset
  - Solids vs. liquids, Nasal regurgitation
  - Speech change, Fatiguability
  - H/O Parkinsons / stroke / small vessel disease
- Examination:
  - Additional cranial nerve involvement:
    - CN III, IV, VI, ptosis - ?Myasthenia
    - CN IX, X, XI - Jugular foramen syndrome
  - Limbs:
    - Mixed UMN / LMN signs - MND
    - Proximal weakness, rash - Myositis
    - Grip weakness - IBM





BREAK

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Update

Mrs C presents feeling tired and drowsy

30 year old nurse. Usually fit and well.

“I’ve been feeling generally weak over the past few weeks, and now I’m more sleepy than usual.”

On Examination:

Bilateral ptosis

Right eye appears to look downwards and outwards, double vision

Slurred speech which fatigues

Limb strength normal

Reflexes normal

Sensation normal

No ataxia

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Where is the lesion?

1. Brain
2. Spinal cord
3. Peripheral nerve
4. Neuromuscular junction
5. Muscle
6. Don’t know - need more information

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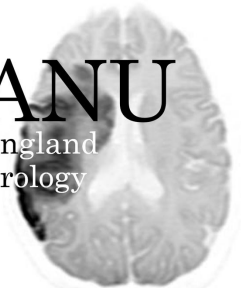
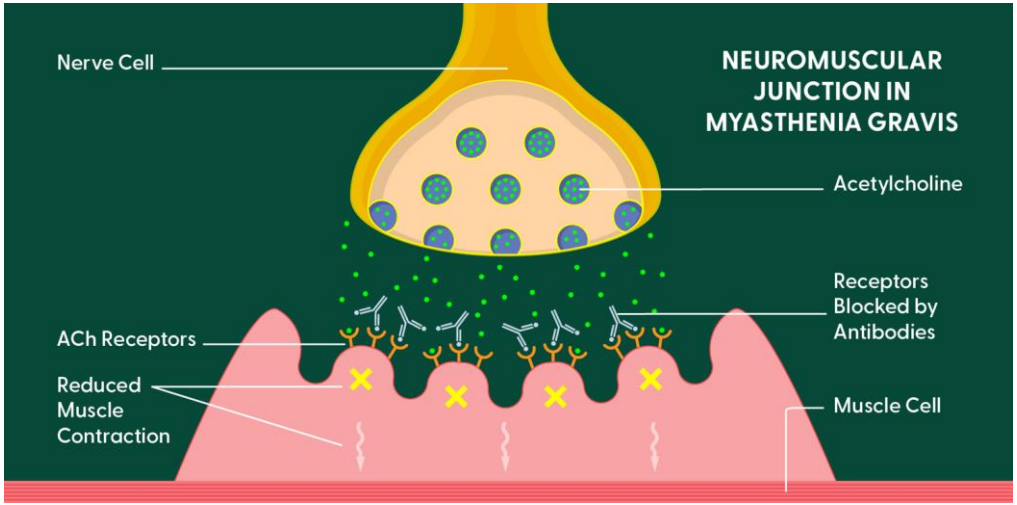


Myasthenia Gravis

Fatigable weakness of some or all of:

- Eye movement (double vision)
- Ptosis
- Neck muscles (head drop)
- Bulbar muscles (speech / swallowing)
- Proximal limb muscles
- Respiratory muscles



https://www.mg-united.com/disease-and-treatment/what-is-myasthenia-gravis/

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Ice pack test

https://www.youtube.com/watch?v=oetVi29_qbE

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Acute Neuromuscular Junction Disorders

MG	LEMS	Botulism
AChR / MUSK antibodies	Voltage gated Ca channel Abs	Botulism toxin (serum / stool)
Usually starts in the eyes before generalising	Usually starts in the limbs and moves up	Acute descending paralysis starting in face
No autonomic dysfunction	Increased thirst and impotence	Dry mouth, dilated pupils, postural hypotension, ileus
Diplopia and dysphagia common	Diplopia and dysphagia uncommon	Diplopia and dysphagia common
Weakness worsens with activity	Weakness improves with activity	Not fatiguable
Thymoma (10%)	Associated with SCLC	Canned food / wounds
Immune modulating treatment, mestinon	Immune modulating treatment, mestinon, 3,4-DAP	Antibiotics and anti-toxin

Management of Myasthenic Crisis

- Respiratory monitoring
- Assess swallow
- Call neurology SpR for guidance on treatment
    - Pyridostigmine (mestinon)
    - Steroids (prednisolone) - start low - risk of steroid dip
    - IVIG / PLEX
- Check not on contraindicated medications - https://www.myaware.org/drugs-to-avoid
- Try to avoid fatiguing patient

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When would you refer to Critical Care?

1. When Forced Vital Capacity has fallen by >30% from baseline
2. When Forced vital capacity has fallen to <20ml/kg body weight
3. When CO2 rising on ABG
4. When acidotic
5. When hypoxic
6. When unable to swallow their secretions
7. All of the above

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Acute Neuromuscular Respiratory Failure: Escalation to ITU

- Monitor FVC or SBC every 4 hours
- Bulbar weakness or neck flexion weakness?
- Rapidly worsening weakness?
- Call ITU if:
    - FVC <20ml/kg body weight (e.g. <1.4L in a 70kg man : SBC<14)
    - or if falling (>30% from baseline within 24 hours)
- Rise in CO2 or acidosis are late signs
- ? Managing secretions
- Dysautonomia

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The weak patient: *Problems assessing on the ward*

- Pulmonary function “tests” often unreliable
  - Poor effort (or poor coaching)
  - Inadequate mouth seal
  - Unreliable / lack of machinery
  - Can miss fatigability
- Classic signs appear only late
  - Paradoxical breathing
  - Hypercapnia
- Degree of limb weakness may not always with diaphragmatic function
- Delays in getting neurophys (or a

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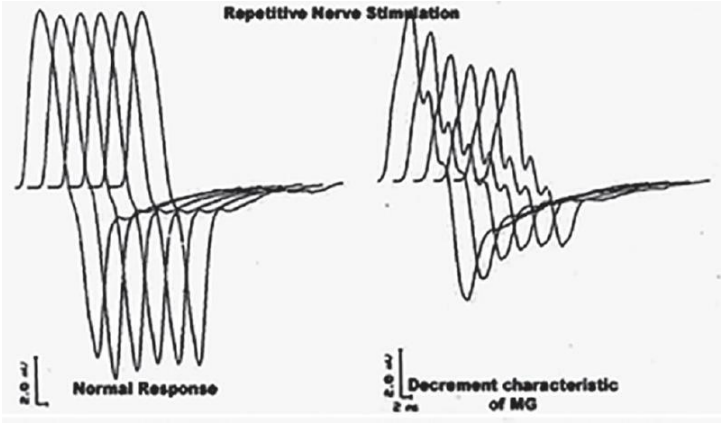
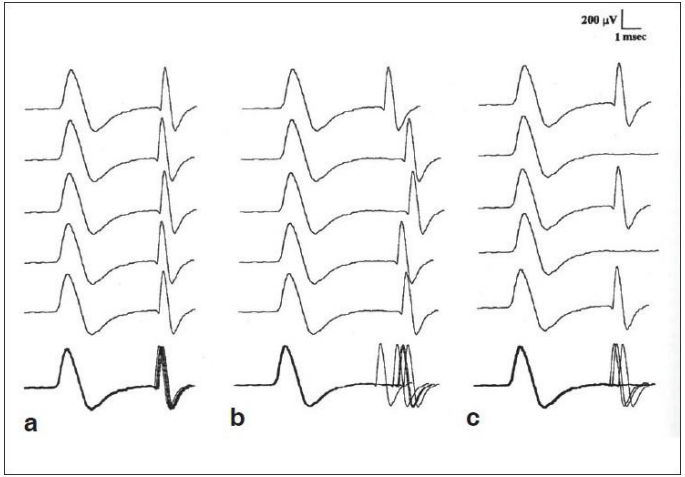
Further Management of Myasthenia

- Confirm diagnosis:
  - AChR Antibodies, MuSK antibodies
  - NCS with repetitive nerve stimulation
  - Single fibre EMG
- Screen for thymoma
- Long term management
  - Pyridostigmine
  - Steroids
  - Azathioprine / mycophenolate





Selvan, V.A., 2011. Single-fiber EMG: A review. *Annals of Indian Academy of Neurology*, *14*(1), p.64.

Is this breathlessness due to neuromuscular weakness?

- Other neuromuscular symptoms / signs:
  - dysarthria,
  - dysphagia,
  - neck flexion weakness,
  - breathlessness lying flat
- Spirometry:
  - restrictive pattern,
  - lying / sitting FVC,
  - MEPs / MIPS / SNIPs

Consider other causes / co-morbidity / decompensation

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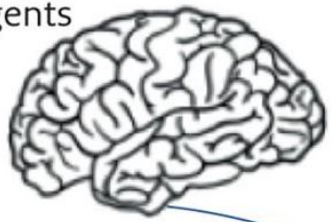
Other Neurological Causes of Respiratory Failure

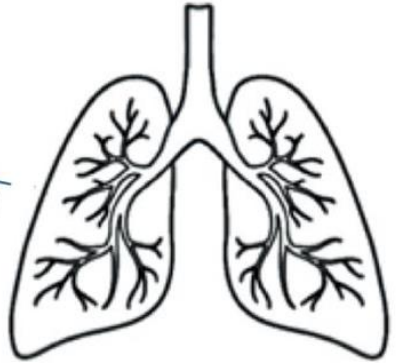
Drive
- Opioids/neuropathic agents
- Congenital central hypoventilation syndrome



Transmission
- Motor neuron disorders
- Guillain–Barré syndrome
- Critical illness neuromyopathy
- Spinal cord injury

Action
- Muscular dystrophies
- Inflammatory myopathies
- Metabolic myopathies
- Critical illness myopathy

Neuromuscular junction
- Myasthenia gravis



Neeraj M. Shah et al. Breathe 2020;16:200121

55 year old

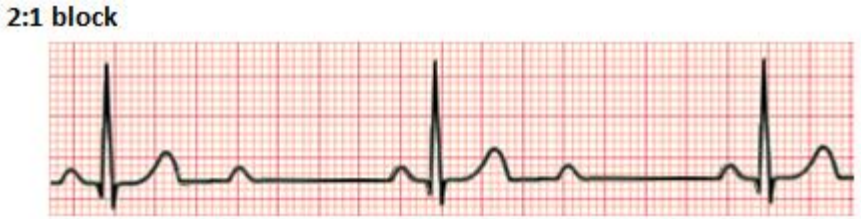
Background: depression, anxiety, personality disorder, diabetes

On Examination:

Distal weakness (grip, foot drop)

Unusual finger movements

Mrs D is admitted with dizzy episodes. ECG shows second degree heart block



Thoughts?

Myotonic Dystrophy I

A multisystem disorder:

Skeletal muscle
- *Distal* limb weakness
- Myotonia
- Facial weakness

Respiratory
- Diaphragmatic and chest wall weakness
- Ventilatory failure

Cardiac
- SAN fibrosis
- Cardiac arrythmia

Cataracts

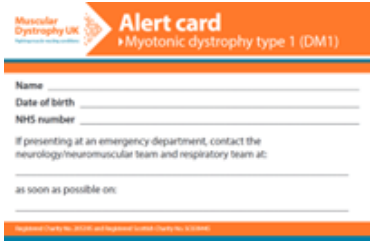
CNS
- Intellectual deficits
- Passive aggressive traits
- Central sleep apnoea
- Daytime somnolence

Gastrointestinal
- Gallstones
- Cholecystitis
- Dysphagia
- Dysmotility

Endocrine
- Testicular atrophy
- Insulin insensitivity

Skin
- Balding
- Pilomatrixomata and epitheliomas

Anaesthetic intolerance


Muscular Dystrophy UK

Alert card

Myotonic dystrophy type 1 (DM1)

Name

Date of birth

NHS number

If presenting at an emergency department, contact the neurology/neuromuscular team and respiratory team at:

as soon as possible on:

Myotonic Dystrophy I: *Myotonia*

When to think of a myopathy…

- History:
  - Floppy baby
  - Delayed motor milestones
  - Sports at school
  - Family history
  - Previous rhabdomyolysis
  - Unexplained respiratory problems
- Examination:
  - Inspection of muscles
  - Myopathic face
  - Ptosis
  - Cataracts
  - Contractures, scapular winging

Idiopathic Inflammatory Myopathies

Dermatomyositis: proximal weakness, heliotrope rash, gottron's papules, cancer

Anti-synthetase syndrome: Jo-1 Abs,mechanic hands, ILD, arthritis

Necrotising myopathy: severe weakness, v. high CK, SRP Abs (cancer) / HMGCR Abs (statins)

Inclusion Body Myositis: long finger flexors and quadriceps, chronic

Metabolic / Endocrine

Hypokalaemia / magnesaemia

Hypothyroid

Toxic

Steroids
Statins

Genetic

Muscular Dystrophies: Myotonic dystrophy 1+2, Duchenne, Becker, Limb girdle, FSH, Oculopharyngeal

Glycogen Storage: McArdles, Pompe

Fatty Acid Oxidation defects: CPTII

Channelopathies: Periodic paralysis, Myotonia / paramyotonia congenita

Mitochondrial: MELAS

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Mrs E is admitted with generalised weakness and a raised troponin I

60 year old nurse. Recent treatment for melanoma.

“I’m feeling really weak over the past few days. I’ve got double vision, and I’m starting to feel breathless.”

On Examination:

Bilateral ptosis

Double vision with complex eye movement abnormalities

Proximal weakness

Normal reflexes

Normal sensation

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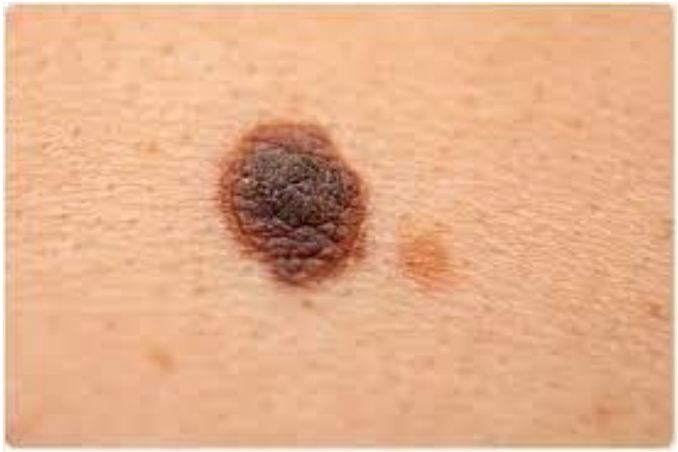
Plan

1. **Find details of cancer treatment**
2. **Check ECG, FVC / breath count**
3. **Routine bloods, including CK**
4. **Cardiac monitoring**



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Plan

1. **Find details of cancer treatment** -

   Ipilimumab and nivolumab for malignant melanoma - second infusion 2 weeks ago

1. **Check ECG, FVC / breath count**

   ECG - prolonged PR interval and diffuse T wave inversion

   FVC 1.5L

1. **Routine bloods including CK**

   CK 8000 IU/L

4. **Cardiac monitoring**

   Occasional ventricular ectopics

Diagnosis:
Checkpoint Inhibitor associated Myasthenia/Myositis/Myocarditis

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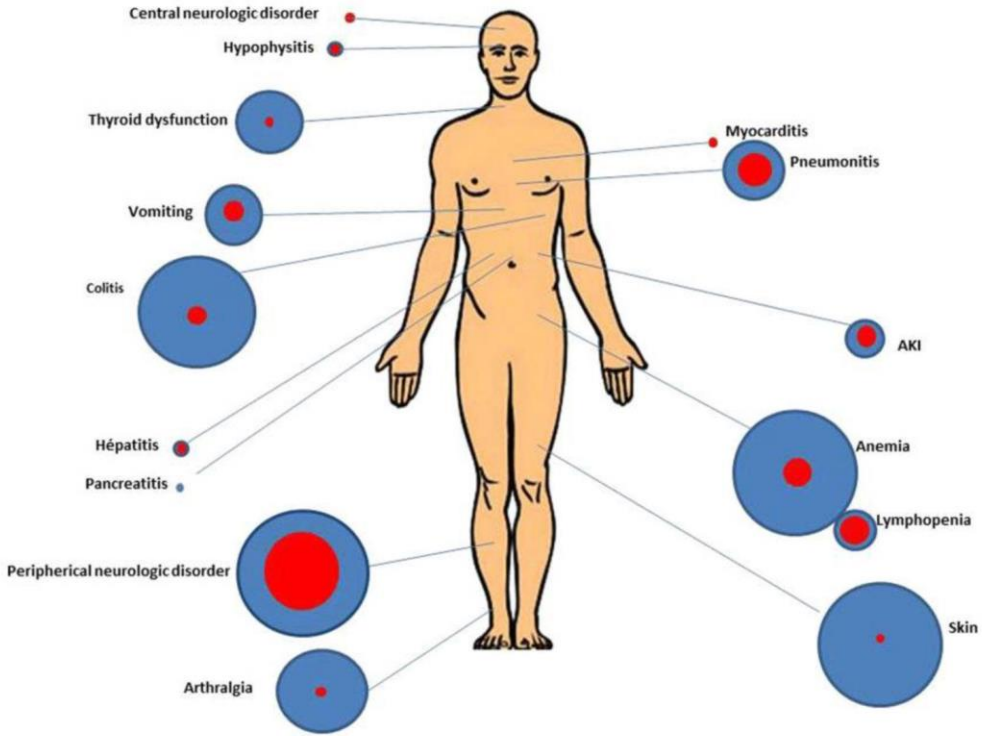
Checkpoint Inhibitor Toxicity

- Block immune checkpoints
- Encourages T cell attack
- Revolutionised treatment of many cancers
- Autoimmune side effects common
- Neurological complications have high mortality (irMG = 30%)
- Aggressive treatment



Ann Intensive Care. Feb. 2019. doi: 10.1186/s13613-019-0487-x.

58 years old

Diagnosed with CIDP 8 years ago

Initially treated with steroids and MTX

Now on IVIG

Recently feeling more unsteady on feet

Had a fall yesterday - banged head

Mrs F is admitted “off legs”

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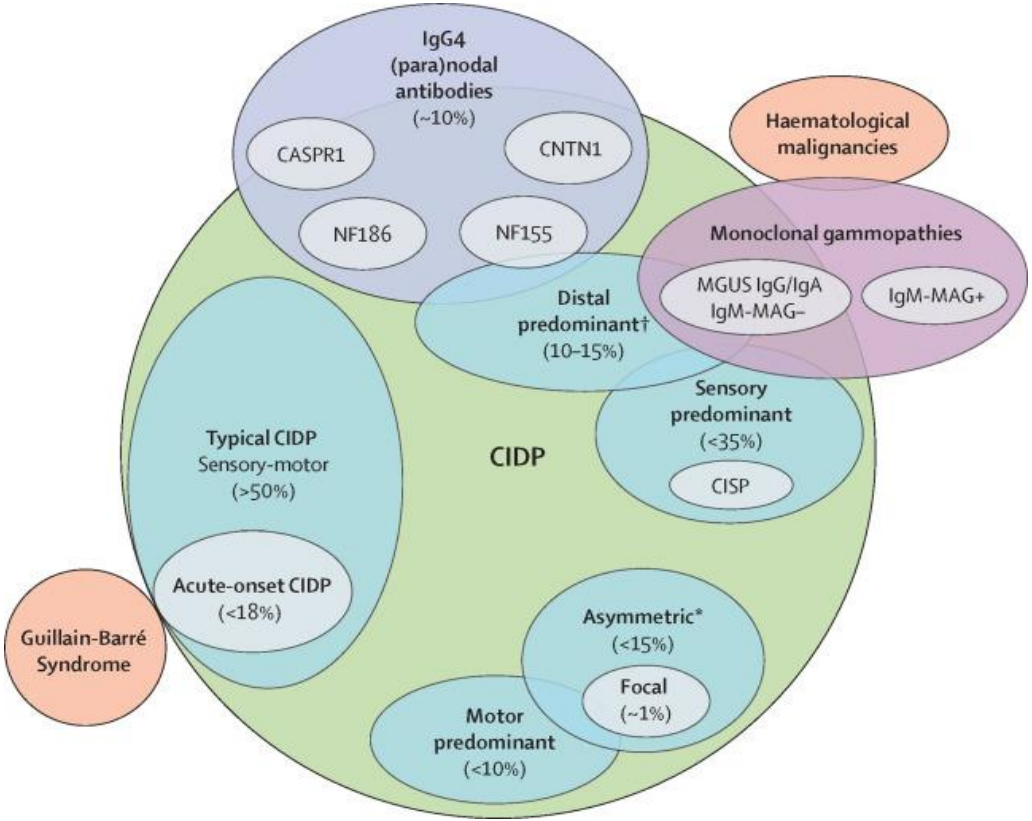
- Thoughts?
- Do you want to know more?

Key principles in the “flaring” patient

- Decompensation related to systemic illness?
  - Infection / metabolic screen
- Compliance?
  - Steroids / DMARDS / IVIG / Rituximab
- Missed treatments?
  - Holidays etc.
- Recent changes in treatment?
  - Attempts to wean down IVIG…
- Is the diagnosis correct?
- → Liaise with neurology

Chronic inflammatory demyelinating neuropathy

- “Chronic version of GBS”
- Sensory - motor syndrome
- Proximal and distal weakness
- No central signs
- Associated with diabetes
- Can complicate haematological malignancy
- Raised CSF protein and demyelinating NCS
- Often requires intensive immunosuppression / immunomodulation (especially paranodapathies)



Primer in Acute Neuromuscular Disease: *Summary*

- **Localise** the lesion
  - Nerve / NMJ / Muscle diseases can have very different causes
- **Avoid harm** from preventable causes
  - Respiratory failure and aspiration
  - Cardiac arrhythmia and cardiomyopathy
  - DVT / PE
- Timely commencement of effective **treatment**

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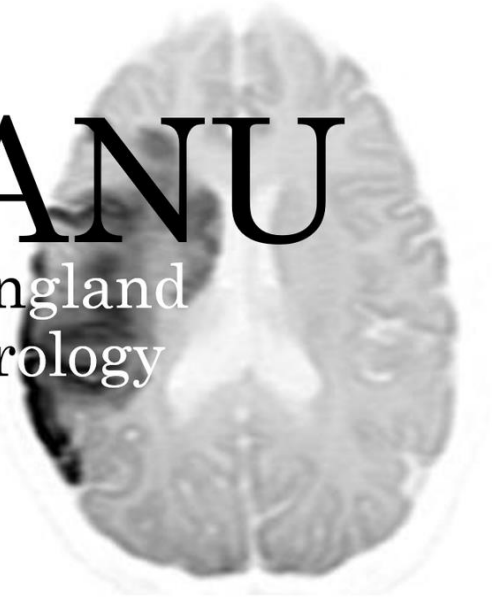

Thank you