ACUTE WEAKNESS

Causes: (list not exhaustive)

Upper motor neurone lesion

- Central – stroke
- Spinal cord – acute cord compression
  transverse myelitis
  multiple sclerosis
  vascular event (can occur after AAA repair and angiogram)

Lower motor neurone lesion

- Anterior horn cells – poliomyelitis
- Root or plexus
- Motor neuropathy - Guillain Barre
  porphyria
  heavy metal poisoning
  critical illness polyneuropathy
  tick paralysis
- Neuromuscular junction – myasthenia gravis
  muscle relaxant
  organophosphate poisoning
  botulism
  tetradotoxin
- Myopathy - metabolic e.g. hypoPO_4, hypoK, hypoCa, hypoMg
  familial periodic paralysis
  endocrine myopathies
  critical illness polyneuropathy

Management

- General
  Intubation and ventilation may be required
  Look for bulbar weakness
  Monitor FVC (<15ml/kg) and watch out for respiratory weakness
  ABG may decompensate very late
  Haemodynamic instability can be due to a variety of causes
  e.g. autonomic neuropathy, cardiomyopathy, septic shock
  Treat accordingly

- Diagnosis
  Often difficult and need meticulous history, clinical examination and consultation with neurologists/neurosurgeons. You can read up on the different conditions in neurology textbooks. Some of the tests you may encounter:

CT brain,spine
MRI brain, spine
Lumbar puncture
Electrodiagnostic (Nerve conduction test, evoked potentials, nerve biopsy, EMG, muscle biopsy)
Tensilon test (for Myasthenia Gravis)
Electrolytes and metabolic screen
Toxicology/ heavy metal screen
Demonstration of toxin eg botulinal toxin in patient's serum or faeces
Acetylcholine receptor antibody (for MG), red cell/plasma cholinesterase (organophosphate poisoning)
Serology (HIV, syphilis etc)

- Specific management – depends on underlying cause
  Perform and document clearly the neurological findings in the notes.
  Further investigation – discuss with ICU senior and neurologists.
  Note in some cases, urgent diagnosis is important as the treatment is time dependent e.g. acute cord compression.
  Some diseases have specific treatment:
  - GBS – IVIG 0.4g/kg/day for 5 days, plasma exchange
  - MG - anticholinesterase (after excluding cholinergic crisis), steroids, plamapheresis
  - Botulism – botulism antitoxin for foodborne; antitoxin plus antibiotics for wound botulism and wound debridement
  - Organophosphate poisoning – atropine, pralidoxime
  - Muscle relaxant – Neostigmine (2.5 mg) and Atropine (1.2 mg) IV
  - Periodic paralysis – correct hypokalaemia
  - Endocrine paralysis – correct the appropriate endocrine abnormality eg thyrotoxicosis, hypothyroidism

- General principles apply:
  stress ulcer prophylaxis
  DVT prophylaxis
  nutrition
  pain management
  look out for pressure ulcers
  splints to avoid contracture
  bladder and bowel problems

Some of the more common conditions presenting as acute weakness to the ICU

Guillain-Barre Syndrome

- Autoimmune subacutely evolving paralytic disorder
- Aetiology include various viral infections (CMV, EBV, HIV, Hep b/C, Varicella zoster); bacterial (Campylobacter jejuni, Mycoplasma pneumoniae); post-vaccination
- S&S
  - Paraesthesia hands or feet/ weakness/neck pain/diplopia
  - Symmetric weakness, progressive usually over 3 weeks (some more rapid over days)
  - Ascending/descending/global
  - Bulbar dysfunction, resp insufficiency
  - Clinical findings: may have autonomic instability, areflexia or marked hyporeflexia
  - Lumbar puncture: CSF – can be normal in the first week; if normal early, repeat LP; findings of elevated protein < 10 mononuclear WBC/mm_3 ; >50 mononuclear cells or presence of PMNs against GBS diagnosis
  - EMG, nerve conduction studies may be diagnostic (neurologist)
- Specific treatment – see above

Myasthenia Gravis

- Autoimmune disease
- Aetiology – presence of antibodies to the nicotinic acetylcholine receptors (AchR)
- S&S
  - Fatiguability, resp insufficiency, bulbar involvement
  - Clinical findings – weakness esp demonstration of fatiguability, diplopia, ptosis, poor facial movement (myasthenic snarl)
  - Sensory normal
  - Reflexes normal
  - Positive tensilon test
  - Lab tests: Elevated titres of anti-AChR ntibodies in 85-50% of cases, EMG and nerve conduction studies (by neurologist)

You may be required to perform a tensilon test:

- Can be double blind (you and patient), or single blind (patient)
- Have atropine on standby
- Double blind study is preferable – fill one syringe with 10 mg of edrophonium (tensilon) and one with saline, make up to 10 mls in volume each. Get an assistant to label syringes such that you are blinded
- Perform baseline muscle testing eg FVC, cranial nerves, peripheral muscle power
- Inject 2 mls first then test muscle power. If worsens, repeat test with other syringe

If not, retest muscles and give the rest of the volume (8mls) and repeat muscle testing
Always test with both solutions and get assistant to break blind code at the end
Implications of a positive tensilon test (ie improvement in muscle power with edrophonium) – confirms diagnosis of myasthenia gravis (vs cholinergic crisis from overdose of pyridostigmine) as cause of acute weakness, patient may benefit by increasing dose of pyridostigmine

Specific treatment – see above

Critical Illness Polyneuropathy

- Seen in up to 70% critically ill patients
- Often presents as difficulty in weaning
- Risk factors
  - Length of stay in ICU (>5 days)
  - Severe sepsis with multiorgan failure
  - Use of steroids
  - Use of muscle relaxants, esp steroid relaxants
- Clinical features
  - Predominantly motor axonal polyneuropathy, distal muscle weakness, some sensory disturbances
  - Cranial nerves unaffected
- Investigations
  - EMG and nerve conduction studies (refer to neurologist)
  - CPK near normal
  - CSF at most mildly raised protein

Organophosphate poisoning

- Accidental or deliberate poisoning
- Highly lipid soluble agents – well absorbed from skin, oral mucosa, conjunctivae, GIT and respiratory routes
- Diagnosis
  - Based on history of exposure, symptoms of cholinergic overactivity and decrease RBC or plasma cholinesterase activity
  - Miosis, blurred vision, sweating, muscle paralysis, bradyarrhythmias, hypotension, bronchoconstriction, salivation, rhinorrhoea, dyspnoea
  - Lab – red cell or plasma cholinesterase. Do not correlate with clinical severity
- Management
  - Self protection
  - Atropine – note: reverses muscarinic effects only

Titrate 0.6-2.4 mg at 3-5 minute intervals until signs of successful atropinisation. Over 10-20mg or infusions up to 5 mg/h may be required

- Ventilatory and CVS support as indicated
- Pralidoxime iodide: 2 g IV over 30 minutes then 500 mg/h for 3 days