

Acute flaccid paralysis (AFP)

By

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Acute flaccid paralysis (AFP) is a clinical syndrome characterized by **rapid onset of weakness**, including (less frequently) weakness of the muscles of respiration and swallowing, **progressing to maximum severity** within **several days to weeks**.

The term "**flaccid**" indicates the absence of
spasticity or other signs of disordered central
nervous system motor tracts such as hyperreflexia,
clonus, or extensor plantar responses

AFP is a **complex clinical syndrome** with a broad array of **potential etiologies**.

☐ **Accurate diagnosis** of the cause of AFP has profound implications for **therapy** and prognosis.

☐ If **untreated**, AFP may not only **persist** but also lead to **death** due to **failure of respiratory muscles**.

Clinical Approach to AFP

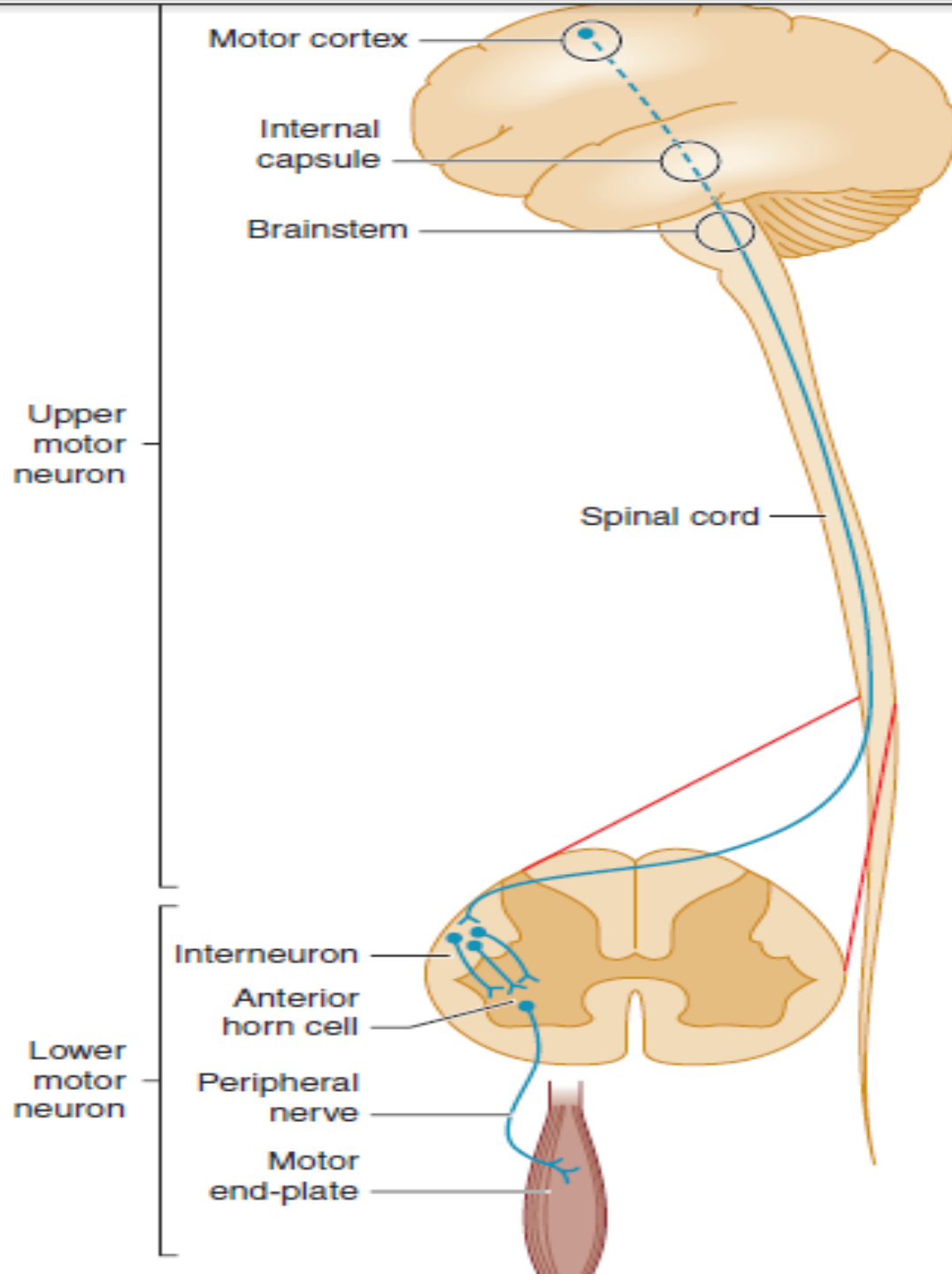
Each case of AFP is a **clinical emergency** and requires **immediate examination**.

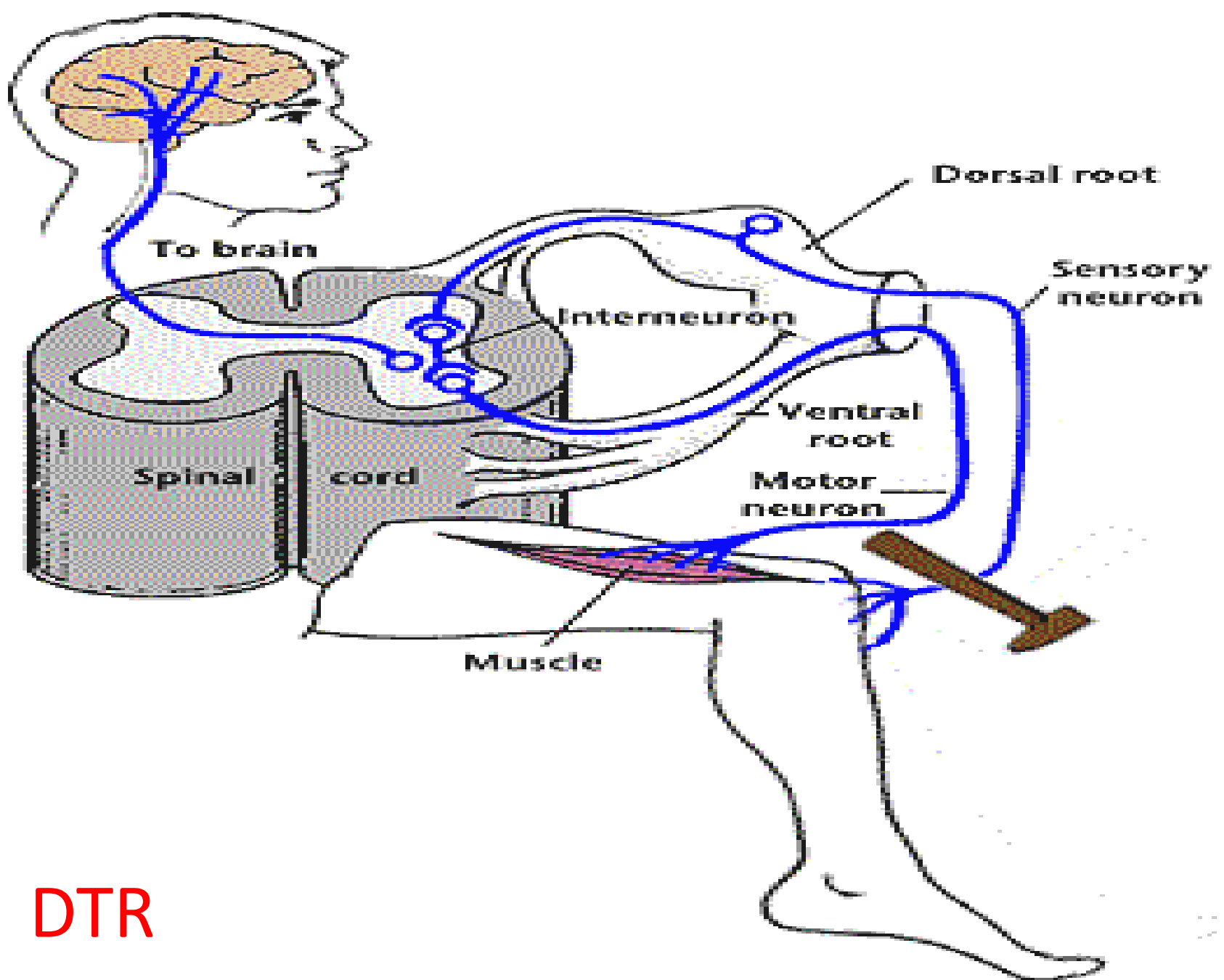
☐ For all cases, a **detailed clinical description** of the symptoms should be obtained, including **fever, myalgia and distribution, timing, and progression of paralysis**.

☐ The **symptoms** of paralysis may include **gait disturbance or weakness in one or several extremities**

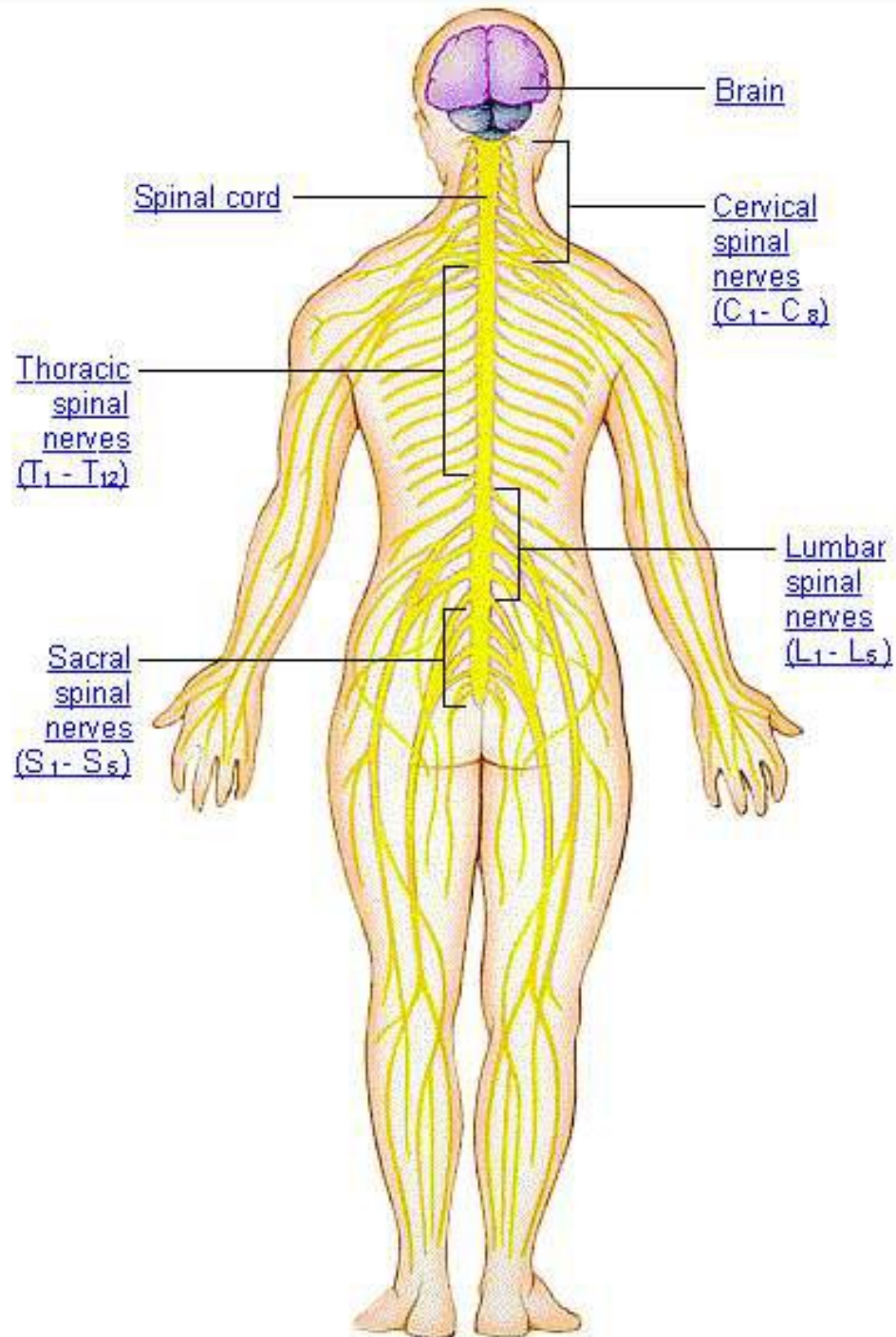
Electrophysiologic studies are **very important** for determining the **diagnosis** and **prognosis** of lower motor neuron disease

☐ **Nerve conduction velocity** and **electromyographic studies** are used to **differentiate** demyelinating neuropathies from axonal neuropathies



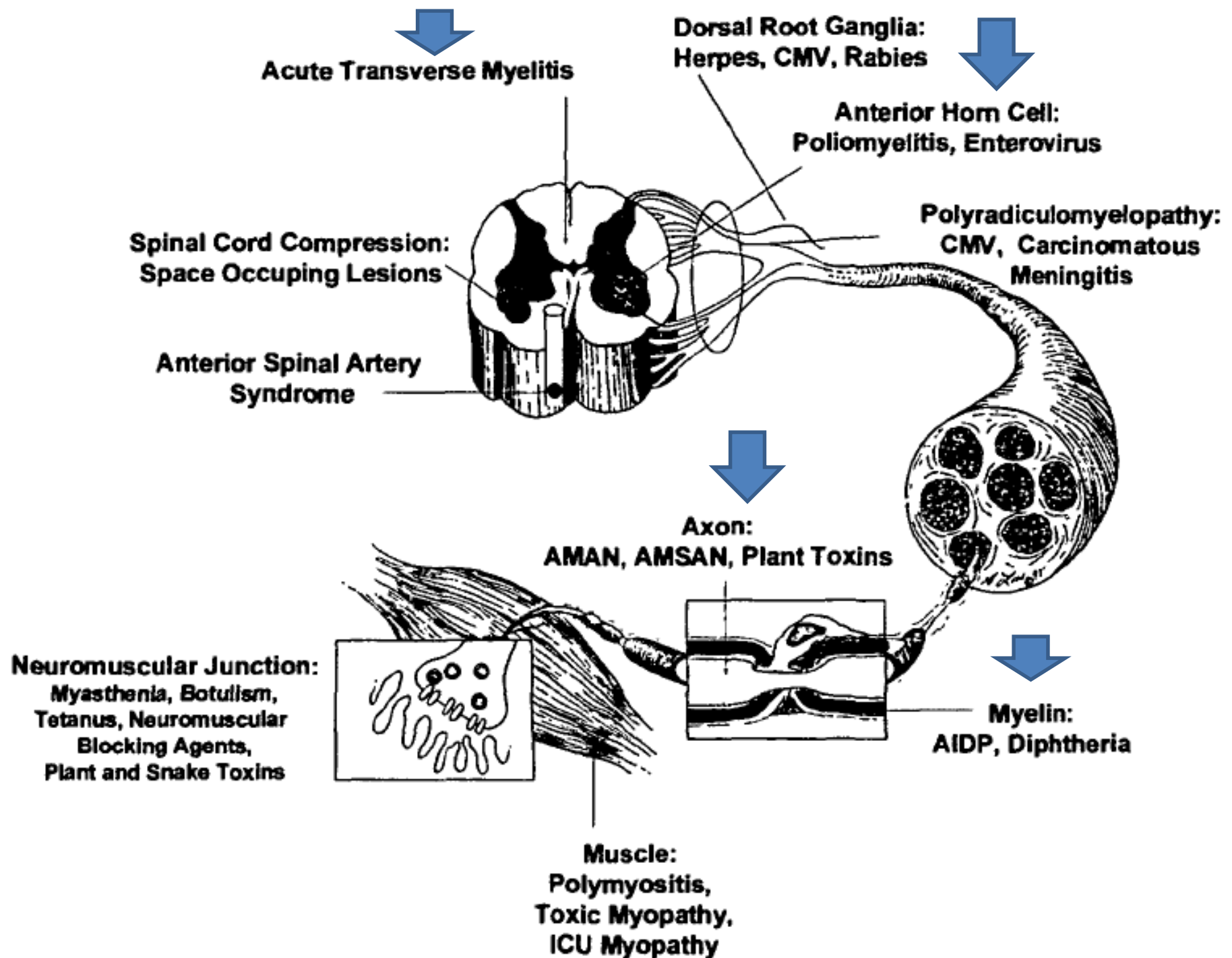


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Differential Diagnosis

- Guillain –Barré Syndrome
- Acute Transverse Myelitis
- Poliomyelitis
- Hypokalemic Periodic Paralysis



Guillain-Barre syndrome(GBS)

the commonest cause of acute flaccid paralysis
(Acute inflammatory demyelinating
polyradiculoneuropathy (AIDP))

**Symmetrical progressive ascending weakness,
areflexia, variable sensory complaints, and
elevated CSF protein without pleocytosis .**

Pathophysiology of GBS

- Acquired, monophasic
- Immune mediated disease
- No known genetic factor
- Two thirds of cases follow a **respiratory or gastrointestinal infection**
- Campylobacter infection is the most common, but other organisms include CMV, EBV, HSV, enteroviruses, ...

Clinical Features of GBS

- Two to four weeks after a **benign febrile illness**
- Common presentations are **paresthesias** in the **fingers and toes**, **pain** is a common presentation in **children** (79%), particularly low back pain
- Symmetrical weakness in the lower extremities, that **ascends over hours to days** to involve the **arms**, and in **severe** cases **respiratory** muscles

Cranial nerves are affected in **30%** of the cases, **most commonly** the **facial nerve** with **bilateral** facial weakness

More than 90% of patients reach the nadir of their function within **2-4 weeks**

Physical Examination in GBS

Symmetrical weakness with diminished or absent reflexes

Vibration and position sensation are affected in **40%** of cases

50% of patients have evidence of **autonomic dysfunction** :

- ☐ Cardiac dysrhythmias

- ☐ Orthostatic hypotension, hypertension

- ☐ Paralytic ileus

- ☐ Bladder dysfunction

Diagnosis of GBS

Cerebrospinal fluid:

- ☐ After the first week of symptoms, CSF typically reveals **normal pressure, normal cell count, and elevated protein**

Electrophysiologic studies:

- ☐ **Most specific and sensitive tests** for diagnosis
- ☐ Evidence of evolving **multifocal demyelination**
- ☐ **Normal studies after 10 days of illness make the diagnosis of GBS unlikely**

GBS Management

- ☐ Expectant with **mild cases**
- ☐ Immune modulatory therapy for **rapidly progressive cases**, (most effective the first 10 days) :
 - **Plasmapheresis**
 - **IVIG**
 - *** **Steroids** are **not** effective and **not** indicated
 - **Critical care monitoring**
- ☐ Most common cause of death is **autonomic dysfunction**
- ☐ Second most common cause of death is **respiratory failure**

Risk factors for **respiratory failure** in GBS :

☐ ***Cranial nerve involvement***

☐ ***Short time from preceding respiratory illness***

☐ ***Rapid progression over less than 7 days***

☐ ***Elevated CSF protein in the first week***

☐ ***Severe weakness :***

- Unable to lift elbows above the bed

- Unable to lift head above the bed

- Unable to stand

Transverse Myelitis

Acute demyelinating disorder of the **spinal cord** that **evolves** over **days** usually but **may** have a **hyperacute presentation**

- ☐ **May** be associated with demyelination in other parts of the central nervous system
- ☐ **Commonly preceded by a viral infection or immunization**

❑ **Commonly** presents with **an ascending weakness**

❑ **Initially** **reflexes may be depressed or absent**
because of *spinal shock* or involvement of the
nerve roots

Symptoms **progress rapidly, peaking within 2 days**

- ☐ Usually level of myelitis is **thoracic**
- ☐ **Sensory level, asymmetrical leg weakness, and early bladder involvement.**
- ☐ **Back pain** is **common at the onset**
- ☐ **Tendon reflexes** may be **decreased or increased**
- ☐ **Recovery** usually **begins after a week of onset**



Transvers Myelitis

Treatment of TM

High doses of IV steroids (methylprednisolone)
followed by **tapering** doses of prednisone

❑ **Prognosis:** **50%** make a **full recovery**
40% recover incompletely
10% do not recover

Poliomyelitis

Enteroviruses (*Poliovirus*, *coxsackievirus*, and the *echovirus group*) are **RNA viruses** that inhabit the **GI tract of humans**

☐ They are **neurotropic**, and produce **paralytic disease** by **destroying the motor neurons of the brainstem and spinal cord**

☐ **Poliovirus** causes the **most severe paralysis**, **coxsackie and echoviruses** are more likely to cause an **aseptic meningitis**

Nonpolio enteroviruses have been associated with **polio-like paralytic disease**, frequently accompanied by **other clinical syndromes**, such as aseptic meningitis, hand-foot-and mouth disease, and acute hemorrhagic conjunctivitis.

Poliomyelitis Clinical presentation

Epidemics usually occur in the spring and summer

- ☐ Usually a **brief illness** characterized by **fever, malaise and GI symptoms** precedes the **paralytic illness**
- ☐ **After the febrile illness**, there is a **brief period of apparent well being, after which** the **fever recurs**, with **headache, vomiting and meningeal irritation**

☐ **Pain** in the *limbs and spine* is followed rapidly by limb weakness

☐ **Pattern of limb weakness** is variable, but is generally asymmetric

☐ **Weakness, diminished reflexes and muscle atrophy** are seen

☐ **Paralysis**

❑ **Bulbar polio** may occur with or without spinal polio and is life threatening

❑ Affected children have prolonged periods of apnea and require mechanical ventilation

❑ **Extraocular muscles** are spared

❑ **Paralytic polio** is rarely seen after the introduction of the polio vaccine

Diagnosis:

- ❑ **Clinical suspicion**
- ❑ **CSF leukocytosis** is seen the **acute phase**, **elevated protein** may also be seen
- ❑ **CBC** shows **leukocytosis**
- ❑ **Virus** recovery from **stool** is **essential**
- ❑ Obtain ***stool, blood and throat samples*** for **viral serology**, demonstrating a **four fold rise in IgG** is *helpful but not always easy*.
- ❑ **Positive IgM antibodies** is **diagnostic**

Treatment: mainly supportive

- ❑ **Mechanical ventilation** may be needed in **bulbar** involvement
- ❑ **Pain management** for paresthesias
- ❑ **Physical therapy**

Polio Vaccine

A **single dose** of Sabin's **oral polio vaccine** produces immunity to **all three poliovirus serotypes** in approximately **50%** of recipients. **Three doses** of live-attenuated OPV produce protective antibody to **all three poliovirus** types in **more than 95%** of recipients.

After **two doses** of **IPV** (given by injection), **90% or more** of individuals develop protective antibody to **all three serotypes** of poliovirus, and **at least 99%** are immune to poliovirus following **three doses**.

Acute Periodic Paralysis

CALCIUM CHANNEL DISEASE

This disease has become clinically apparent **after adolescence** and has been **much more severe in males**.

The usual pattern of inheritance is **autosomal dominant with reduced penetrance in women** (**male-to-female ratio** of **3** or **4:1**)

The **typical attack** comes on **during the second half of the night or the early morning hours**, after a day of unusually strenuous exercise; a meal rich in **carbohydrates** favors its development.

Excessive hunger or thirst, dry mouth, palpitation, sweating, diarrhea, nervousness, and a sense of fatigue are mentioned as **prodromata** but *do not necessarily* precede an attack.

the weakness lasts a few hours if **mild** or several days if **severe**.

Limbs are affected earlier and often more severely than trunk muscles, and proximal muscles are possibly more susceptible than distal ones.

The muscles most likely to **escape** are those of the eyes, face, tongue , pharynx, larynx, diaphragm, and sphincters,

As the attack subsides, strength generally returns first to the muscles that were last to be affected.

Laboratory Findings

- Reduction in serum K levels, as low as 1.8 mEq/L
(The serum K levels return to normal during recovery)
- The fall in serum K is associated with little or no increase in urinary K excretion (large quantities of K enter the muscle fibers during an attack)

Diagnosis at a time **when the patient is normal** may be facilitated by **provocative tests** with the carefully **monitored**, including the use of **ECG**, the **oral** administration of **50 to 100 g of glucose** or **loading with 2 g of NaCl every hour for 7 doses**, followed by **vigorous exercise**, brings on an attack, which then can be **terminated** by **2 to 4 g of oral KCl**

Treatment

A low-sodium diet (less than 160 mEq/d), avoidance of large meals and of exposure to cold, and acetazolamide 250 mg tid may be helpful in **preventing attacks**.

For an **acute attack**, 0.25 mEq KCl/kg should be given orally or, if this is not tolerated, some other K salt may be tried. This dose may be insufficient and if there is no improvement in 1 or 2 h, KCl may have to be given intravenously: 0.05 to 0.1 mEq/kg initially in a bolus at a safe rate, followed by 20 to 40 mEq KCl in 5 percent mannitol, avoiding glucose or NaCl as the carrier solution



END