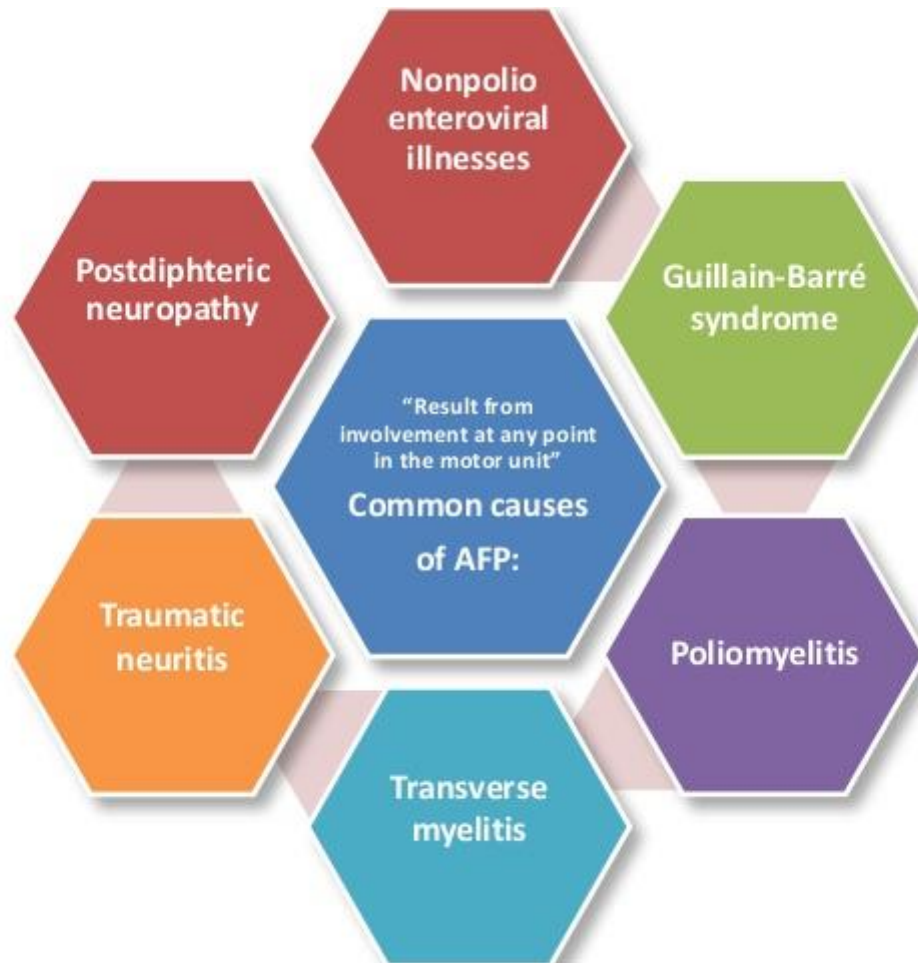


# *Approch to Flaccid paralysis*

By : Reza Rikhtegar (MD)



Nonpolio  
enteroviral  
illnesses

Postdiphtheric  
neuropathy

Guillain-Barré  
syndrome

"Result from  
involvement at any point  
in the motor unit"  
Common causes  
of AFP:

Traumatic  
neuritis

Poliomyelitis

Transverse  
myelitis

# Differential Diagnosis of AFP

- **SPINAL CORD**
- **Anterior horn cell disease**
- **PERIPHERAL NERVE**
- **Disorders of neuromuscular transmission**
- **MUSCLE**
- **Systemic disease**

# SPINAL CORD

- *Demyelinating diseases*
- transverse myelitis
- *Cord compression*
- tumour
- trauma
- paraspinal abscess
- haematoma
- vascular malformation with thrombosis/bleeding
- *Ischaemic cord damage*
- spinal cord stroke
- anterior spinal artery syndrome
- peri-operative complication

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# PERIPHERAL NERVE

- ***Unilateral:***
  - Enteroviral infection
  - local trauma
  - Focal mononeuropathy
- ***Bilateral:***
  - Guillain Barré syndrome
  - Acute toxic neuropathies (heavy metals, snake toxin)
  - Neuropathies of infectious diseases (diphtheria)

## Disorders of neuromuscular transmission

- Myasthenia gravis
- Botulism
- Insecticide (organophosphate poisoning)
- Tick bite paralysis
- Snake bite

# MUSCLE

- Polymyositis
- post viral myositis
- periodic paralysis
- toxic myositis (Corticosteroids and blocking agents)
- Mitochondrial diseases (infantile type)



# Investigations

- Spinal cord MRI.
- CSF study.
- Nerve conduction study.
- OTHERS

# How to differentiate among

- Polio
- Guillain-Barré syndrome
- Traumatic neuritis
- Transverse Myelitis

	<b>Polio</b>	<b>Guillain-Barré syndrome</b>	<b>Traumatic neuritis</b>	<b>Transverse myelitis</b>
<b>Installation of paralysis</b>	24 to 48 hours onset to full paralysis	From hours to ten days	From hours to four days	from hours to four days
<b>Fever at onset</b>	High, always present at onset of flaccid paralysis, gone the following day	Not common	Commonly present before, during and after flaccid paralysis	rarely present
<b>Flaccid paralysis</b>	Acute, usually asymmetrical, principally proximal	Generally acute, symmetrical and distal	Asymmetrical, acute and affecting only one limb	acute, lower limbs, symmetrical
<b>Muscle tone</b>	Reduced or absent in affected limb	Global hypotonia	Reduced or absent in affected limb	Hypotonia in lower limbs
<b>Deep-tendon reflexes</b>	Decreased to absent	Globally absent	Decreased to absent	Absent in lower limbs early hyperreflexia late

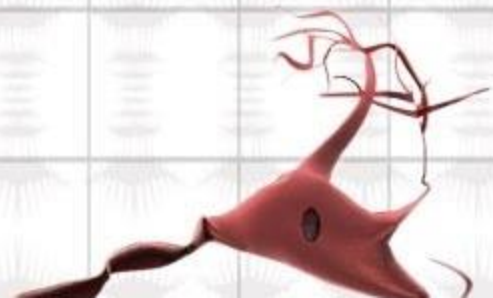
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	<b>Polio</b>	<b>Guillain-Barré syndrome</b>	<b>Traumatic neuritis</b>	<b>Transverse myelitis</b>
<b>Bladder dysfunction</b>	Absent	Transient	Never	Present
<b>Nerve conduction Velocity</b>	Abnormal: anterior horn cell disease (normal during the first 2 weeks)	Abnormal: slowed conduction, decreased motor amplitudes	Abnormal: axonal damage	normal or abnormal, no diagnostic value
<b>EMG</b>	Abnormal	Normal	Normal	Normal
<b>Sequel at three months and up to a year</b>	Severe, asymmetrical atrophy, skeletal deformities developing later	Symmetrical atrophy of distal muscles	Moderate atrophy, only in affected lower limb	flaccid diplegia atrophy after years



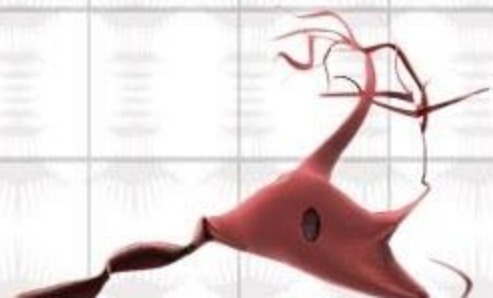
# Involvement

- AHC:
  - 1- acute poliomyelitis
  - 2- acute transverse myelitis
  
- Peripheral Nerves:
  - 1- roots: GBS (post-infectious)
  - 2- toxins: Diphtheria, porphyria



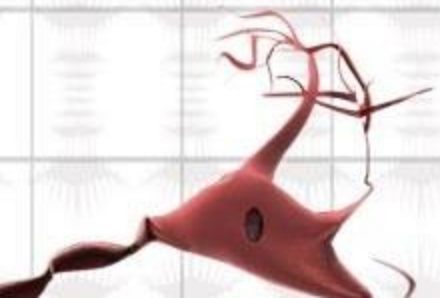
# Involvement

- N-M junction:  
botulinum toxin  
tick toxin
- Metabolic:  
Periodic paralysis
- Muscular:  
myositis



# Guillain Barre Syndrome GBS

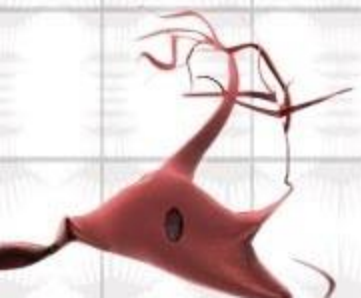
- The most common cause of acute flaccid paralysis (AFP) among infants.
- Age : any including newborn
- Sex : any ( male > female)





# Guillain-Barre' Syndrome

- Post-infectious polyneuropathy; ascending polyneuropathic paralysis
- An acute, rapidly progressing and potentially fatal form of polyneuritis



# Pathophysiology

**Autoimmune disorder (T cell sensitization)**

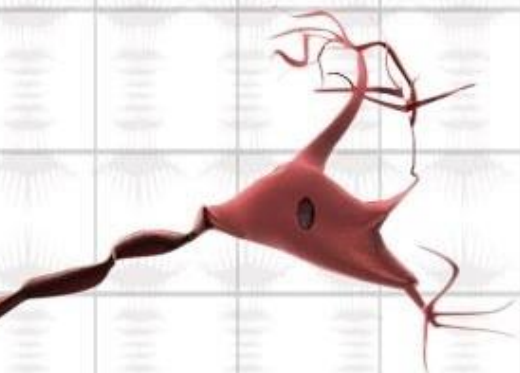
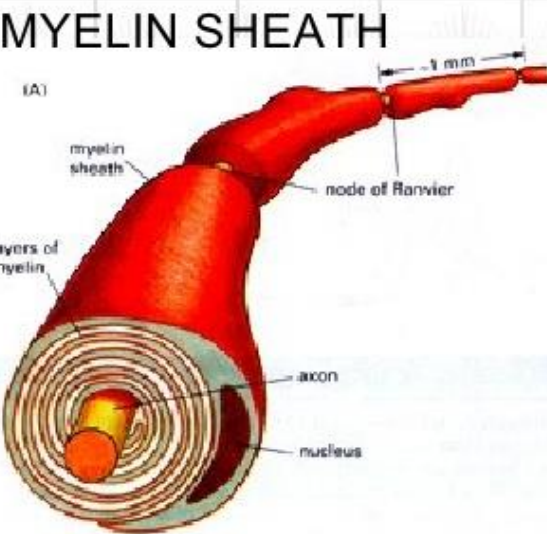
**cause of demyelination**

**Due to attack of the myelin sheath of nerves by:**

- **antibodies (Ig M, Ig G)**
- **white blood cells (macrophages)**
- **Complement activation** on the outer surface of myelinated fibers

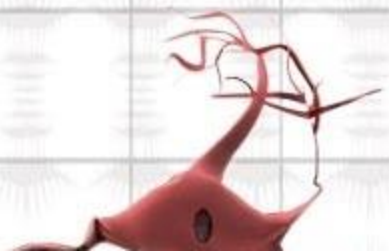
**Because (POST-)**

**Virus/Bacteria share antigenic sites with axons & peripheral nerve sheath or both**



## pathophysiology

- inflammation causes leakage of proteins into the CSF causing raised CSF proteins without pleocytosis
- Can involve the peripheral nerves, cranial nerves, dorsal roots, dorsal root ganglia & sympathetic chain





## Preceding Events : (1-3 WEEKS)

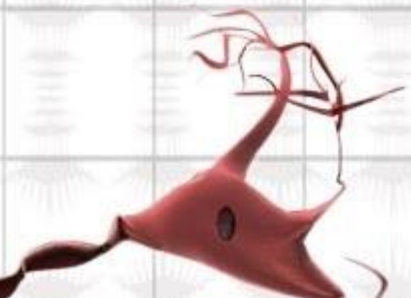
- **Respiratory infections :**
  - 1- Viral: CMV, EBV, Varicella virus , influenza virus
  - 2- Bacterial: Mycoplasma pneumoniae, H influenza
- **Gastrointestinal infections :** Campylobacter-jejuni (Bloody GE)

- **Vaccinations**
- Post surgery



# Aetiology

- Mycoplasma
- Hepatitis B
- CMV
- EBV
- Measles
- Mumps
- Echovirus
- Cocksakie virus
- Influenza virus
- Varicella virus
- Compylobacter jejuni

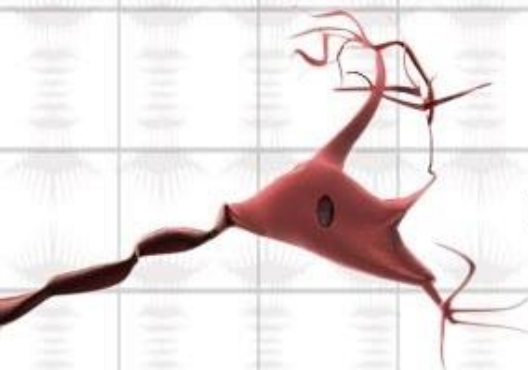


# classification of GBS

## (Clinical, Pathological & neurophysiological)

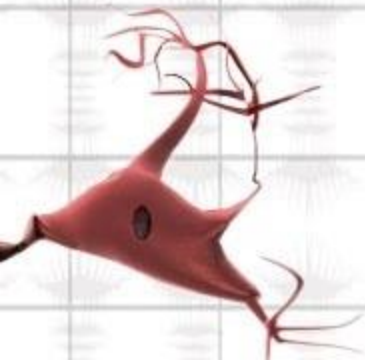
- **1-Classic type (mixed): Acute inflammatory demyelinating polyradiculo-neuropathy (AIDP)**
- **2- Pure motor GBS\***
- 3- Pure sensory GBS
- 4- Pure pandysautonomia
- 5-- Miller-Fisher syndrome ( hypotonia, ophthalmoplegia, ataxia)

NB., Pure Motor GBS\*  
Usually Post Campylobacter-jejuni infection



## C/P:

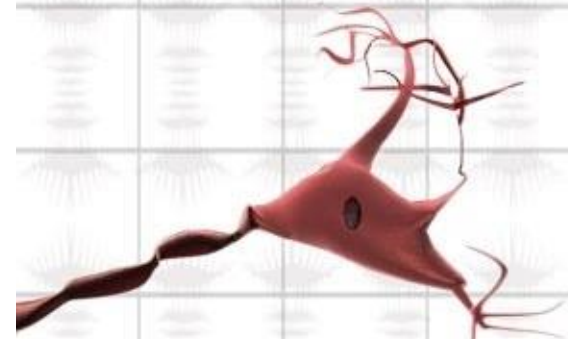
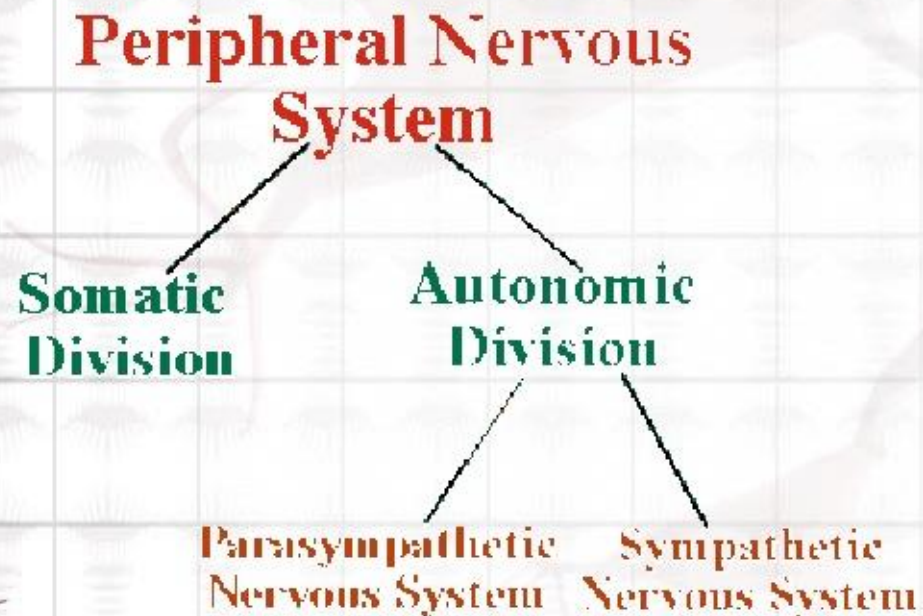
- 1- IP ( afrebrile ) : 1-3 weeks
- 2- CP: motor , sensory , autonomic
- 3- Serious Association





# Guillain-Barre' Syndrome

- Affects the peripheral nervous system
- 



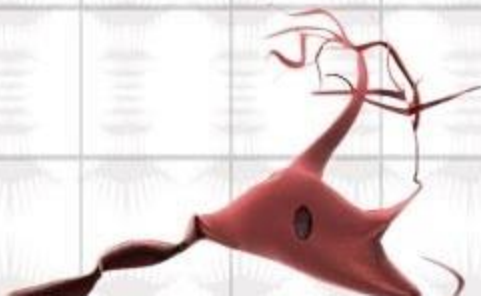


# Symptoms and Signs (Typical GBS)

**Motor :** 1- Symmetric acute progressive ascending weakness <4 wks,  
starting in LL

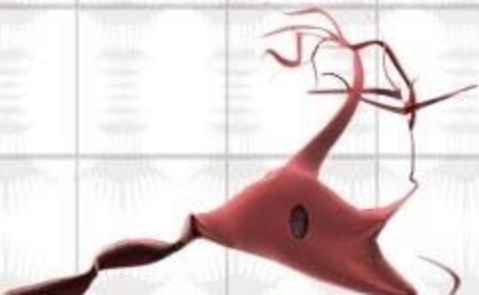
2- Areflexia or hyporeflexia

3- Atonia or hypotonia



# Symptoms and Signs (Typical GBS)

- Sensation :**
- 1- C/O pain as hyperthesia or cramps
  - 2- O/E loss of pain sensation (hypothesia) in feet/hands
  - 3- Both C/O, and O/E





## Characteristic “3A” triad:

- **ascending weakness** : a- bilateral symmetrical weakness  
b- usually start in LL, then UL  
c-then, might be affected :
  - i- **cranial nerves (Brain stem)** :  
including glossopharyngeal and vagus nerves ( difficulty of swallowing even of fluid and water) and III, IV, VI cranial nerves ( eye muscles in Miller Fisher variety), VII Facial nerve ( unilateral or bilateral), and the respiratory muscles
  - ii- **respiratory muscles**
  - iii- **phrenic nerves ( diaphragm )**
- **areflexia ( Hallmark)**
- **atonia ( hypotonia)**



# CLINICAL VARIANTS

1–Polyneuritis cranialis

Cranial nerve involvement

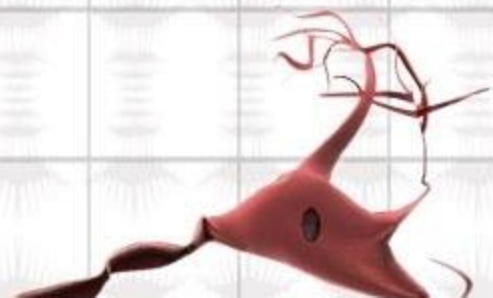
2–Miller fisher syndrome

Ophthalmoplegia, ataxia, areflexia

3–Chronic progressive GBS

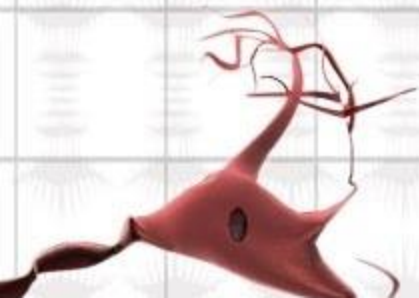
Symptoms persisting more than 6 weeks

4- Chronic relapsing GB



## Differentiation from spinal cord syndrome

- ♦ Loss of arm reflexes
- ♦ Absence of sensory level
- ♦ Lack of spinal tenderness
- ♦ Normal bowel and bladder function





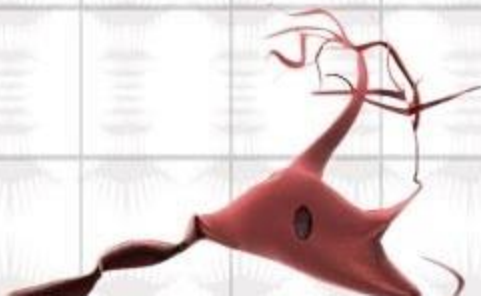
# Investigations

Early

Nerve Conduction Velocity (NCV) abnormality

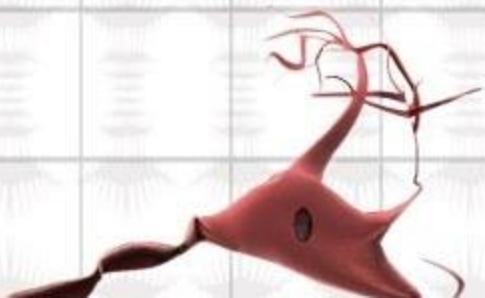
**AFTER 1<sup>ST</sup> WEEK**

- Late : CSF study : albuminocytogenic dissociation



# 1- NCV/EMG:

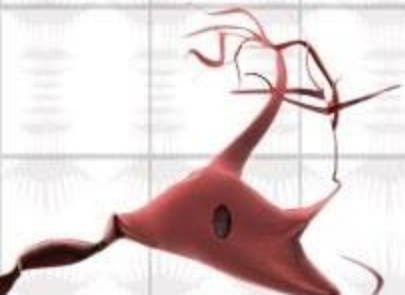
- i- **Early** :Delayed or absent F waves or H reflexes
- ii- slow or block of Nerve conduction velocity
- iii- normal EMG/ extensive fibrillation showing denervation



## 2- CSF: Albuminocytologic dissociation (Froin Syndrome)

- i- Increased CSF protein with normal cells
- ii- might be normal CSF during 1st week
- ii- usually +ve after 2 weeks of onset

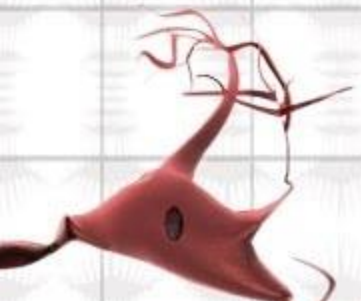
**Differential Diagnosis of cytoalbuminous dissociation**





# DD

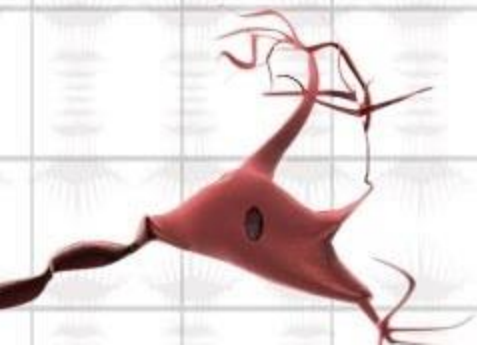
- **1- GBS**
- **2- poliomyelitis**
- **3- Diphtheria polyneuritis**
- **4- spinal cord compression**
- **5- transverse myelitis**
- **6- infratentorial tumor**
- **7- venous sinus thrombosis**
- **8- lead poison**
- **9- botulism**



# Treatment

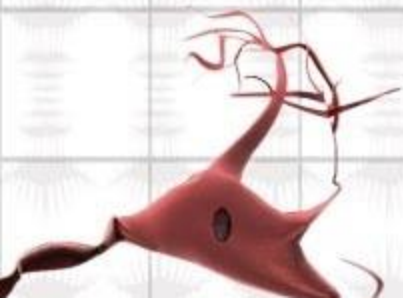
## Hospitalization

- 1- **General care**
- 2-- **Specific treatment**
- 3- **complication treatment**



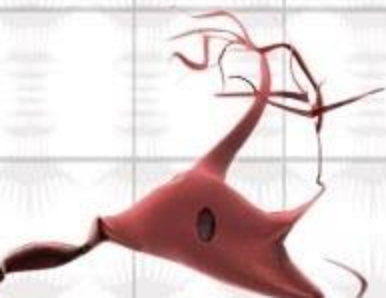
# Specific treatment

- **i- IV immunoglobulin: 2 gm/kg treatment**
  - \* at a dose of 0.4 g/kg/day for 5 consecutive days or
  - \* 1gm/kg/day for 2 days
- **ii- plasmapheresis: 5 exchanges of 50 ml plasma/ kg on alternate days ( 10 days course).**
- **iii- both i and ii**



# Transverse Myelitis:

- ? of immunological disorder
- C/P : of AFP ( acute onset of flaccid hypotonic weakness ) with the following characters:
  - LL paralysis : paraplegia with areflexia
  - with sensory level of loss of sensation
  - Later : hyperreflexia



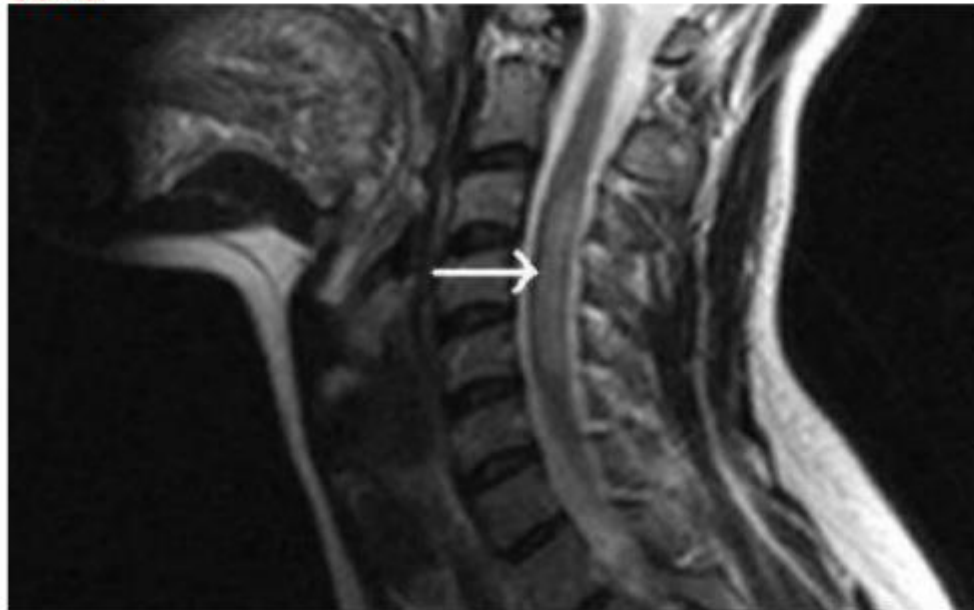


# Transverse Myelitis

- Transverse myelitis is a condition characterized by rapid development of both motor and sensory deficits .

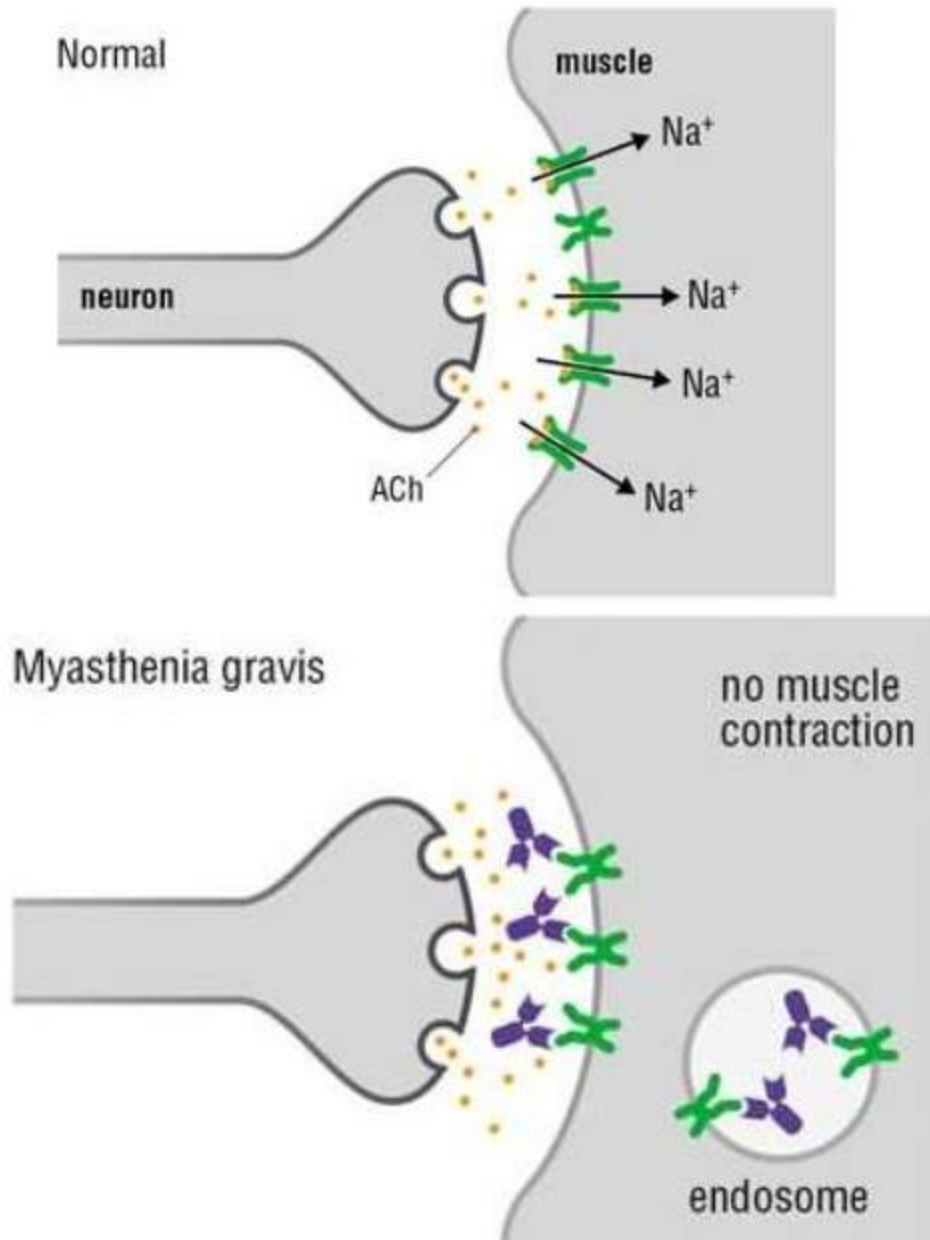
# Transverse Myelitis

- disorder caused by **inflammation of the spinal cord**



- An autoimmune of neuromuscular junction.
- Weakness of skeletal muscles.
- Fatigability on exertion.

🟢 PATHOPHYSIOLOGY;

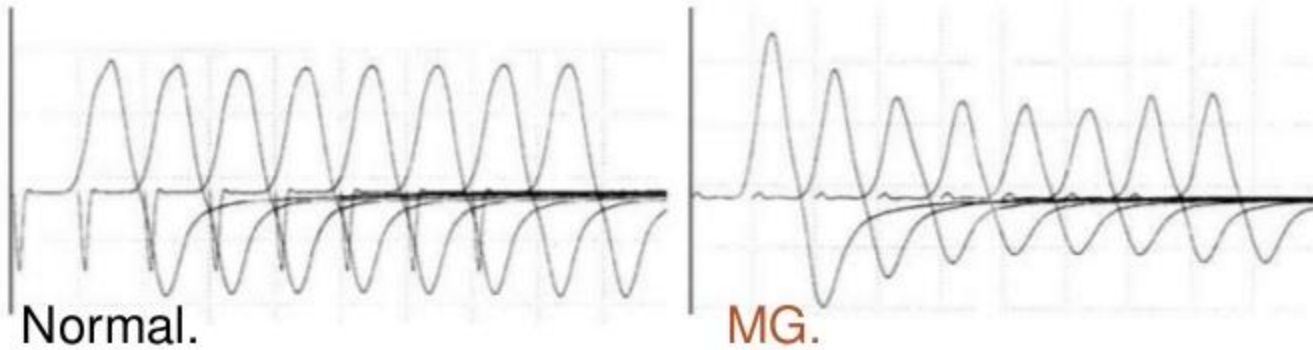


# MYASTHENIA GRAVIS



- ◆ Myasthenia Gravis is often associated with:
  - ◆ Hashimoto thyroiditis.
  - ◆ Some collagen vascular diseases.
  - ◆ Thymoma (*mostly with adults; rarely in children*).
  
- ◆ **Post-infectious myasthenia:**
  - ◆ Affects children.
  - ◆ Follows infection with varicella zoster.
  - ◆ Transient.

- ◆ DIAGNOSTIC STUDIES;
- ◆ EMG.
  - ◆ *More diagnostic than Bx.*



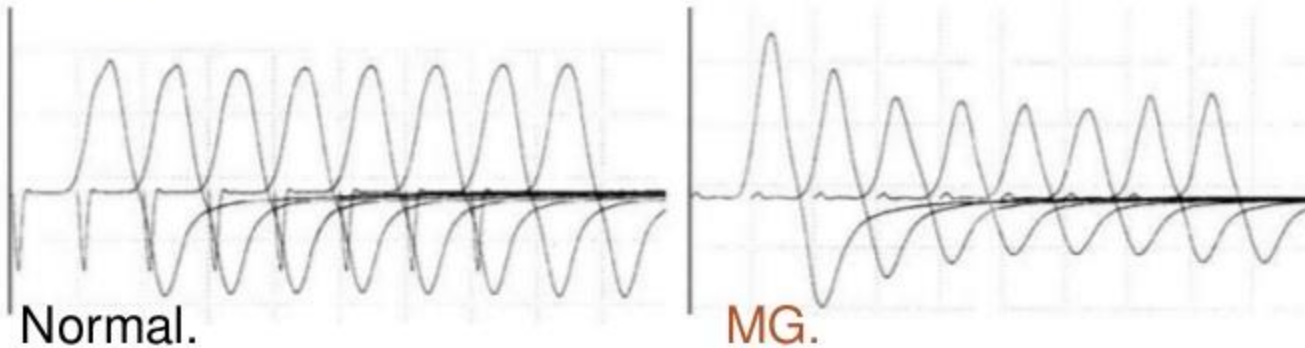
- ◆ Anti-Ach Abs.



- ◆ Tensilon test (Edrophonium Test)
  - ◆ *Ptosis and ophthalmoplegia improve within a few seconds, and fatigability of other muscles decreases.*

# MYASTHENIA GRAVIS DIAGNOSIS

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- EMG.
  - More diagnostic than Bx.*



- Anti-Ach Abs.



- Tensilon test (Edrophonium Test)
  - Ptosis and ophthalmoplegia improve within a few seconds, and fatigability of other muscles decreases.*

# Hypokalemic Periodic Paralysis