- **Guillain-Barre' syndrom**

- **Seizure disorder**

Guillain-Barre' syndrom, or acute idiopathic polyneuritis, is also known as infectious polyneuritis. It's an acute, rapidly progressive, and fatal form of polyneuritis that causes muscle weakness and mild distal sensory loss.

-Most commonly between ages 30 and 50.

-Affects both sexes equally.

-Recovery is spontaneous and complete in about 95% of patients; however mild motor or reflex deficits may persist in the feet and legs.

-Prognosis is best when symptoms resolve sooner than 15 to 20 days after onset.

Three phase syndrome

-The acute phase begins with the - onset of the first definitive symptom and ends 1 to 3 weeks later. Further deterioration doesn't occur after the acute phase.

-The plateau phase lasts several days to 2 weeks.

- The recovery phase coincides with remyelinization and growth of axonal processes. Recovery commonly takes 4 to 6 months, but may take as long as 2 to 3 years in severe cases.

Complications:

==========

-Thrombophlebitis

-Pressure ulcers

-Muscle wasting

-Sepsis

-Joint contractures

-Respiratory tract infections

-Respiratory failure

-Loss of bladder and bowel control

What causes it?

- The exact cause is un-known

But it may be cell-mediated response to a virus.

About 50% have recent history of upper respiratory tract infection or less commonly, gastroenteritis.

When infection precedes the onset of Guillain-Barre' syndrome, signs of infection subside before neurological features appear

- Precipitating factors:

.Surgery

.Rabies or swine influenza vaccination

.Hodgkin's or other malignant disease

.Systemic lupus erythromatosis

- Pathology:

  The major pathological feature of Guillain Barr'e syndrome is segmental demyelination of the peripheral nerves, which prevents the normal transmission of electrical impulses along the sensorimotor nerve roots.

Assessment:

-Look for progressive symmetrical muscle weakness (the major neurological sign), appearing first in the legs and then extending to the arms and facial nerves within 24 to 72 hours.

-Muscle weakness developing in brain stem, in cranial nerves and progressing downward in the arms first or in the arms and legs simultaneously.

-Normal muscle strength or weakness affecting only the cranial nerves.

-Paresthesia, sometimes preceding muscle weakness but vanishing quickly.

-Diplegia, possibly with ophthalmoplegia (ocular paralysis), from impaired motor nerve root transmission and involvment of cranial nerves III, IV and VI.

-Dysphagia or dysarthria and, less commonly, weakness of the muscles supplied by CN XI

-Hypotonia and areflexia from interruption of the reflex arc.

Investigations:

-CSF analysis (protein levels increases several days after onset of symptoms and peak at 4 to 6 weeks).

-CBC early in illness shows leukocytosis and immature WBCs called bands).

-EMG may show repeated firing of the same motor unit instead of the widespread sectional stimulation.

-Nerve conduction velocities slow soon after paralysis develops.

-Serum immunoglobulin are elevated due to an inflammatory response.

Treatment:

-Primarily Supportive(ET intubation, tracheostomy)

-Trial dose(7 days) of prednisone to reduce inflammatory response if the disease is relentlessly progressive.

-Plasmapheresis in the initial phase (no benefit if started 2 weeks after onset).

-Continuous ECG monitoring for possible arrythmias due to autonomic dysfunction. Inderal is used to reduce tachycardia. Atropine for bradycardia. Volume replacement for severe hypotension .

Nursing Care:

-Watch for ascending sensory loss, which procedes motor loss.

-Monitor vital signs and LOC.

-Assess and treat patients with respiratory dysfunction.

-Auscultate breath sounds, turn and position the patient, encourage coughing and deep breathing. BEGIN respiratory support at the first sign of respiratory failure (including ETE and mechanical ventilation).

-Provide meticulous skin care.

-Perform passive ROM excercises within the patients pain limits.

-To prevent aspiration, test the gag reflex and elevate the head of the bed before the patients eats (if gag reflex lost give NG tube feeding).

-Change position slowly and be alert of hypotension.

-Apply antiembolism stockings and a sequential compression device to the legs.

-Provide eye and mouth care every 4 hours for people with facial paralysis.

-Watch for urine retention, use undwelling catheter if necessary.

-To prevent constipation, provide high fiber diet, administer a suppository or enema as ordered.

-Refer the patient to physical therapy.

Seizure disorder

- **Seizure disorder**, or **epilipsy,** is a condition of the brain characterized by recurrent seizures (paraxysmol events associated with abnormal electrical discharges of neurons in the brain)

.Primary seizure disorder or epilipsy is idiopathic without apparent changes in the brain.

Secondary epilipsy, characterized by structural changes or metabolic alterations of the neuronal membranes, causes increased automaticity.

Epilipsy affects **1% to 2%** of the population; approximately 2 million people live with epilipsy.

This incidence is highest in childhood and old age.

The prognosis is good if the patient adheres strictly to the prescribed treatment.

Complications may include hypoxia or anoxia due to airway occlusion, traumatic injury, brain damage, and depression and anxiety.

Causes:

- In about one-half cases, the cause is unknown.

Possible causes:

1. Birth trauma (like inadequate oxygen supply to the brain, blood incompatibility, or hemmorrhage).
2. Perinatal infection.
3. Anoxia.
4. Infectious diseases(meningitis, encephalitis or brain abscess)
5. Head injury or trauma.

Mechanism:

- Some neurons in the brain may depolarize easily or be hyperexcitable, firing more readily than normal when stimulated.

On stimulation, the electrical current spreads to the surrounding cells, which fire in turn. The impulse thus cascades to:

.One side of the brain (a partial seizure).

.Both sides of brain(generalized seizure).

.Cortical, subcortical and brain stem areas.

The brain metabolic demand for oxygen increases dramatically during seizure. If this demand isn't met, hypoxia and brain damage results.

Firing of inhibitory neurons causes the excited neurons to slow their firing and eventually stop. Without this inhibitory action, the result is status epilepticus (seizures occurring one right after another).

Without treatment, resulting anoxia is fatal.