Neonatal Birth Traumas or injuries:
These are avoidable and unavoidable injuries to the NB that occurs during the birth process. These are less common now because of more use of C/S. They can occur during prenatal, natal, or postnatal period.

Risk factors for birth injuries include:
prim-parity,
maternal pelvic anomalies,
prolonged or unusually rapid labor,
oligohydraminos,
malpresentation of fetus,
use of mid-forceps or vacuum extraction,
versions and extractions,
VLBWT or extreme prematurity,
fetal macrosomia or large fetal head, and fetal anomalies.

Evaluation of Birth injuries:
1. History
2. thorough examination, including neurologic examination
3. Special attention to symmetry of structure and function, cranial nerves, range of motion of joints, integrity of scalp and skin.

Types of birth injuries:
1. Soft tissues injuries:
   a. Caput succedaneum: it is subcutaneous, extra-periosteal fluid collection over the presenting part in vertex presentation. It extends beyond the suture lines, usually associated with molding of the skull bones, it appears at birth and resolve in few days, it requires no treatment.
   b. Cephal hematoma: it is sub-periosteal Hg due to rupture of vein of Galen between the skull and periostium. It is confined to suture lines, usually over parietal bones, the centre feel soft, it becomes visible on second or third day of life, it resolves over several weeks(8 weeks), when they are extensive can cause jaundice and anemia. Occasionally associated with linear fracture of skull (5-20%), it requires no treatment, but treat infection, anemia and jaundice. Aspiration of blood collection is contraindicated as it will induce infection.
c. **Chignon:** bruising edema from ventose extraction delivery.

d. **Bruising of face** after face presentation, and **Bruising of genitalia** after breech delivery due to repeated PV exam.

e. **Abrasión of the skin** from scalp electrodes applied during labor, or accidental scalpel incision at C/S.

f. **Subgaleal Hg:** it is sub-aponeurotic Hg (under skull aponeurosis), spread rapidly over head downward to the eye, very uncommon, may be associated with serious blood loss, may be associated with ICH, 90% associated with vacuum extraction. It requires no treatment unless there is shock or ICH that require blood transfusion.

g. **Severe or fatal injuries** (very rare) from tear of the tentorium cerebelli or fax cerebri.

h. **Subconjunctival or Retinal Hg:** frequent and common. It requires no treatment.

2. **Nerve injuries:**

A. **Brachial plexus nerve injuries:** this result from traction to the cervical nerve roots. They may occur after breech delivery or with shoulder dystocia.

   **Erbs’ palsy (waiters’ tip posture):** it is upper nerve roots injury (C5-C6). The moro, biceps reflexes are absent, but hand grasp is preserved.

   **Klumpks’ palsy (C7,8,T1):** the lower nerve roots are involved less frequently, resulting in weakness of long flexors of wrists and fingers and intrinsic muscles of the hand. The grasp reflex is absent.

   Immobilize for one week, then passive range of motion exercise of all joints of limb. It needs physiotherapy from second week of life. Most palsies resolve over few weeks. Occasionally following severe injury, paralysis is permanent. Surgical reconstruction of the nerve is attempted.

B. **Facial nerve injury:** most common peripheral nerve injury in neonates. May result from compression of the facial nerve by forceps blades or against mothers’ pelvis. It is usually transient, seen as asymmetrical crying face.

C. **Phrenic nerve injury:** it involves 3rd, 4th, 5th cervical nerves. There is ipsilateral diaphragm paralysis, 75% have brachial plexus injury, present with respiratory distress and cyanosis, diagnosed by U/S, fluoroscopy.

3. **Fractures:**

A. **Skull fractures:** They are rare, usually linear, usually in parietal bones, they require no treatment, just observation. **Depressed fracture** usually in parietal or frontal bones, they are unusual, but may be seen with complicated forceps delivery. If no neurologic deficit ---- no treatment. If neurologic deficit--- do CT scan –surgical elevation.
B. Fracture clavicle: usually from shoulder dystocia. a snap may be heard at delivery, or infant may have reduced movement of the arm on affected side, or a lump from callus formation over clavicle may be felt. The prognosis is excellent.

C. Fracture of humerus and femur: usually mid-shaft, occurring at breech delivery, they present with loss of spontaneous arm or leg movement, pain and swelling in passive movement if displaced. Diagnosis by x ray, treatment by splinting, close reduction and casting.

4. Visceral trauma: Trauma to liver, spleen, and adrenals: they are uncommon, seen in macrosomic infants, very premature infants, with or without breech or vaginal deliveries. They may be asymptomatic, or present with shock, pallor, jaundice. They are diagnosed by ultrasound. Sub-capsular hematoma of liver is the most common, may fellow macrosomia, hepatomegaly, breech presentation. It is suspected if infant had anemia, hypovolemia, shock, but no evidence of IVH. It is usually seen in 1-3 days after birth. Serial PCV is needed suggesting blood loss. Management by restoring blood volume, correct coagulation disorder, and surgical consultation for possible laprotomy.

5. Intracranial Hg: subdural Hg, subarachnoid Hg, per-ventricular Hg, intraventricular Hg, Intra-parenchymal Hg or cerebral Hg.

Subdural Hemorrhage (SDH): usually seen in association with birth trauma, cephalo-pelvic disproportion, LGA babies, forceps deliveries, skull fracture, and postnatal head trauma. It may be asymptomatic initially as Rbc's undergoes hemolysis; the water is drawn into the Hg resulting in expanding symptomatic lesion. Anemia, vomiting, seizure, macrocephaly may occur in infants who is 1-2 months old with SDH. Occasionally massive SDH in neonate by rupture of the vein of Galen or by inherited coagulation disorder as hemophilia. Treatment of all symptomatic SDH is surgical evacuation.

6. Neonatal Cold injury: LBWT neonates and full term neonates with CNS disorders are risk groups, usually in inadequately heated homes and damp cold spells. Neonate presents with apathy, refusal to feed, oliguria, coldness to touch, body temperature of 29.5- 35C. They had immobility, edema, redness of extremities, bradycardia, apnea, facial erythema (due to failure of oxyHb to dissociate at low temp.), and local hardening over areas of edema. Serious metabolic disturbances especially hypoglycemia, acidosis. Hemorrhages especially pulmonary Hg may occur. Management includes gradual re-warming and correct hypotension by normal saline 10-20 mls/kg, correct hypoglycemia, and metabolic disturbances by NAHCO3 and treating infection and bleeding. Prevention by providing adequate environmental temperature. Outcome 10% mortality, 10% of survivors got brain damage.
NEONATAL SEIZURES

**Definition.** A seizure is defined clinically as a paroxysmal alteration in neurologic function (i.e. behavioral, motor, or autonomic function).

**Incidence.** Neonatal seizures are not uncommon. The incidence ranges from 1.5 - 14 in 1000 live births.

**Causes of neonatal seizures**

A. Perinatal asphyxia
B. Intracranial hemorrhage (Subarachnoid hemorrhage, Periventricular or intraventricular hemorrhage, Subdural hemorrhage
C. Metabolic abnormalities: Hypoglycemia, Hypocalcaemia, Electrolyte disturbances: hypo- and hypernatremia, Amino acid disorders
D. Infections: Meningitis, Encephalitis, Syphilis, cytomegalovirus infections, Toxoplasmosis, Cerebral abscess
E. Drug withdrawal
F. Toxin exposure (particular local anesthetics)
H. Congenital malformations

**Clinical presentation.**

It is important to understand that seizures in the neonate are different from those seen in older children. The differences are perhaps due to the neuroanatomic and neurophysiologic developmental status of the newborn infant. In the neonatal brain, glial proliferation, neuronal migration, establishment of axonal and dendritic contacts, and myelin deposition are incomplete.

**Four types of seizures,** based on clinical presentation, are recognized:

**subtle, clonic, tonic, and myoclonic.**

A. **Subtle seizures.** These seizures are not clearly clonic, tonic, or myoclonic and are more common in premature than in full-term infants. Subtle seizures are more commonly associated with an electroencephalographic seizure in premature infants than in full-term infants.

**apnea.** It is differentiated from nonconvulsive apnea (which is due to sepsis, lung disease, or metabolic abnormalities) by the absence of electroencephalographic abnormalities. Apnea as a manifestation of seizures is usually accompanied or preceded by other subtle manifestations. In premature infants, apnea is less likely to be a manifestation of seizures.

B. **Clonic seizures** are more common in full-term infants than in premature infants and are commonly associated with an electroencephalographic seizure. There are two types of clonic seizures.

1. **Focal seizures.**
2. Multifocal seizures. Several body parts seize in a sequential, nonjacksonian fashion (eg, left arm jerking followed by right leg jerking).

C. Tonic seizures occur primarily in premature infants. Two types of tonic seizures are seen.
   1. Focal seizures.
   2. Generalized seizures.

D. Myoclonic seizures are seen in both full-term and premature infants and are characterized by single or multiple synchronous jerks. Three types of myoclonic seizures are seen.
   1. Focal seizures
   2. Multifocal seizures
   3. Generalized seizures

Note: It is important to distinguish jitteriness from seizures. Jitteriness is not accompanied by abnormal eye movements, and movements cease on application of passive flexion, movements are stimulus sensitive and are not jerky.

**Diagnosis**

A. History
   1. Family history. A positive family history of neonatal seizures is usually obtained in cases of metabolic errors and benign familial neonatal convulsions.
   2. Maternal drug history is critical in cases of narcotic withdrawal syndrome.
   3. Delivery. Details of the delivery provide information regarding maternal analgesia, the mode and nature of delivery, the fetal intrapartum status, and the resuscitative measures used. Information regarding maternal infections during pregnancy points toward an infectious basis for seizures in an infant.

B. Physical examination
   2. Neurologic evaluation
   3. Notation of the seizure pattern. When seizures are noted, they should be described in detail, including the site of onset, spread, nature, duration, and level of consciousness. Recognition of subtle seizures requires special attention.

C. Laboratory studies. We must use the information obtained by history taking and physical examination and look for common and treatable causes.
   1. Serum chemistries. Estimations of serum glucose, calcium, sodium, blood urea nitrogen, and magnesium and blood gas levels.
   2. Spinal fluid examination.
3. **Metabolic disorders.** Blood ammonia levels, urine and plasma Amino acids and urine for reducing substances, urea cycle disorders, maple syrup urine disease.

D. **Radiologic studies**
1. **Ultrasonography of the head** is performed to rule out IVH or periventricular hemorrhage.
2. **CT scanning of the head** provides detailed information regarding intracranial disease. CT scanning is helpful in looking for evidence of infarction, hemorrhage, calcification, and cerebral malformations. Experience with this technique suggests that valuable information is obtained in term infants with seizures, especially when seizures are asymmetric.

E. **Other studies**
1. **Electroencephalography (EEG).** EEGs obtained during a seizure will be abnormal. Interictal EEGs may be normal. The diagnostic value of an EEG is greater when it is obtained in the first few days because diagnostic patterns indicative of unfavorable prognosis disappear thereafter. EEG is valuable in confirming the presence of seizures when manifestations are subtle or when neuromuscular paralyzing agents have been given. EEGs are of prognostic significance in full-term infants with recognized seizures. For proper interpretation of EEGs, it is important to know the clinical status of the infant (including the sleep state) and any medications given.

**Management.**
Because repeated seizures may lead to brain injury, **urgent treatment is indicated.**

The method of treatment depends on the cause.

common and treatable causes.

A. **Hypoglycemia.** Hypoglycemic infants with seizures should receive 10% dextrose in water, 2-4 mL/kg intravenously, followed by 6-8 mg/kg/min by continuous intravenous infusion.

B. **Hypocalcemia** is treated with slow intravenous infusion of calcium gluconate. If serum magnesium levels are low (<1.52 mEq/ L), magnesium should be given.

C. **Anticonvulsant therapy.** Conventional anticonvulsant treatment is used when no underlying metabolic cause is found.

Loading doses of phenobarbital and phenytoin control 70% of neonatal seizures.

1. **Phenobarbital** is usually given first. When phenobarbital alone fails to control seizures, another agent is used. If seizures are not controlled at a serum phenobarbital level of 40 mcg/ mL, it is recommended to administer a second agent (eg, phenytoin).
2. **Phenytoin** is used next by many practitioners. Fosphenytoin may be a preferred form.

3. **Pyridoxine** trial with EEG monitoring is recommended.

4. **Diazepam (Valium)** has not been used extensively in the control of neonatal seizures.

5. **Lorazepam**, given intravenously, has been quite effective and safe, even when repeated 4-6 times in a 24-h period

6. **Intravenous midazolam and oral carbamazepine** have been found to be effective.

7. **Paraldehyde**, given rectally, has been used as an effective anticonvulsant.

D. **Duration of anticonvulsant therapy.** The optimal duration of anticonvulsant therapy has not been established. Although some clinicians recommend continuation of Phenobarbital for a prolonged period, others recommend stopping it after seizures have been absent for 2 weeks.

**Prognosis.**

As a result of improved obstetric management and modern neonatal intensive care, the outcome of infants experiencing seizures has improved. The mortality rate has decreased from 40 to 20% but neurologic sequelae are still seen in 25-35% of cases.

As would be expected, **the prognosis varies with the cause.**

Infants with hypocalcaemic convulsions have an excellent prognosis.

Seizures secondary to congenital malformations have a poor prognosis.

Symptomatic hypoglycemia has a 50% risk of death or complications, CNS infection carries a risk of 70%.

Asphyxiated infants with seizures have a 50% chance of a poor outcome.

17% of patients with neonatal seizures have recurrent seizures later in life.