(قالوا سبحانك لا علم لنا إلا ما علمتنا
إنك أنت العليم الحكيم)

صدق الله العظيم

NEONATAL DISORDERS

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LECTURE RULES

Smile

Interactive

Focusing Smile

Mobiles off/silent)

NEONATAL RESUSCITATION

Neonatal Resuscitation

1- Place the newborn under the radiant warmer.

2- Dry the newborn completely.

3- Suction the mouth, oropharynx and nares gently

-If mecenium stained amniotic fluid is present and the infant is not vigorous; suction the oropharynx and trachea as quickly as possible .

4- Rapid evaluation of the infant by Apgar scoring

- at 1 minute → decided the need and method for resuscitation.
- at 5 minutes → reflect adequacy of resuscitative efforts.
  → determine the need for further efforts.

APGAR SCORE

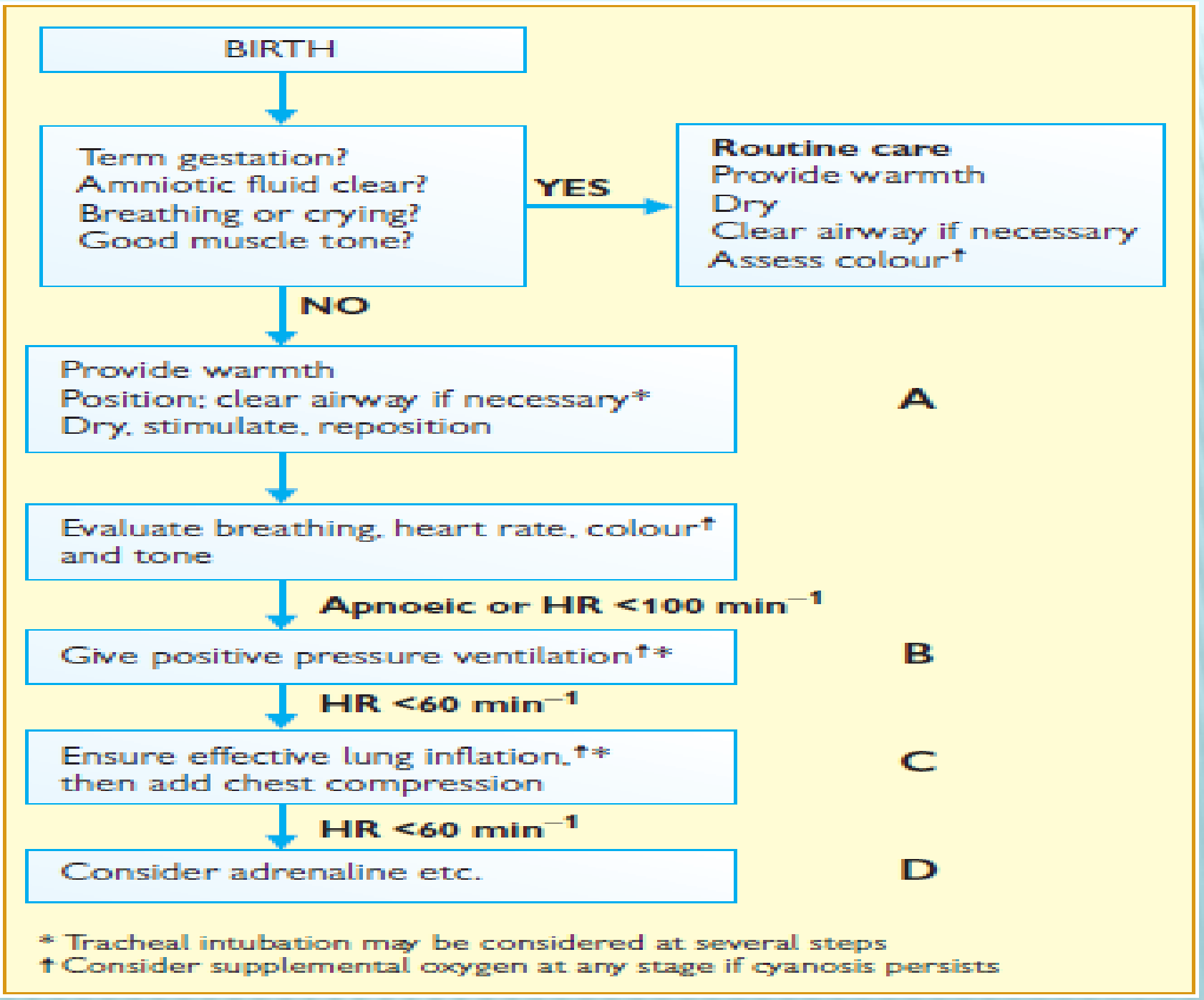
Sign	0	1	2
1- Colour (Appearance)	Blue or pale	Pink with blue extremities.	All pink.
2- Heart rate (Pulse)	Absent	under 100/min	over 100/min
3- Response to nasal catheter (Grimace)	No response	Grimace	Cough, sneezing.
4- Muscle tone (Activity)	Limp “flaccid”	Some flexion	Active motion
5- Respiration	Absent	Slow ± irregular	Normal and crying

Only 5–10% of all newborns require any resuscitation and <1% require intubation in the delivery room. Deliveries which should be attended by a paediatrician include:

gestation <36 weeks, instrumental or surgical deliveries, malpresentations, multiple pregnancies, fetal distress or meconium staining, antenatal diagnosis of malformation or Rhesus disease.

Factors other than intrapartum asphyxia may delay onset of respiration and may co-exist in a single baby.

They include drugs causing CNS depression, prematurity, birth trauma, sepsis, severe anaemia or congenital malformations such as congenital diaphragmatic hernia.


BIRTH
Term gestation?
Amniotic fluid clear?
Breathing or crying?
Good muscle tone?
YES
Routine care
Provide warmth
Dry
Clear airway if necessary
Assess colour†
NO
Provide warmth
Position: clear airway if necessary*
Dry, stimulate, reposition
A
Evaluate breathing, heart rate, colour† and tone
Apnoeic or HR <100 min−1
Give positive pressure ventilation†*
B
HR <60 min−1
Ensure effective lung inflation,†* then add chest compression
C
HR <60 min−1
Consider adrenaline etc.
D
* Tracheal intubation may be considered at several steps
† Consider supplemental oxygen at any stage if cyanosis persists

Before delivery introduce yourself to parents, review obstetric records and check all equipment is functional. At birth, start clock and assess the infant who is likely to fall into one of four main groups:

1-Fit, healthy term infant, crying lustily >90%

2-Irregular respirations, HR >100 bpm 5%

3-Pale, limp, apnoeic, HR <60 bpm 0.5%

4-Dead but rescuscitatable <0.1%.

Resuscitation follows the guidelines shown above

**If baby >2 kg with inadequate respiration, HR 80–100 bpm:* administer air by face mask, give peripheral stimulus to breathing.

**If poor response:* administer mask ventilation attached to a self-infl ating bag or a mechanical ventilator via a T-piece using 100% oxygen for term infants or air-oxygen mixture

*If <32 weeks gestation. Give five breaths at a pressure of 30–40 cmH2O for 2–3 s each to expand the lungs (check chest is moving) and continue at 40–60 breaths/min.

Resuscitative Drugs:

1-Epinephrine:

- Dose: 0.1 – 0.3 ml/kg of 1:10.000 solution.
- Route: intravenous or intra tracheal.
- Dose may be repeated every 5 minutes.

2- Naloxone (Narcan)

- Opiate antagonist
- Indication: if mother received narcotic analgesic within hours of delivery.
- Dose: 0.1 mg/kg I.V. or intratracheal.

3- Na bicarbonate:

- Indication: documented or suspected metabolic acidosis if 2 doses of epinephrine were ineffective.
- Dose: 2 meq/kg slow I.V.

4- Volume expanders

- Indication: hypovolemic shock due to intrapartum blood loss.
- Use:
  * Isotonic saline
  * O –ve fresh whole blood
  * Albumin 5%
  * Plasma.
- Dose: 10 ml/kg.

5- Dopamine:

- Indication: cardiogenic shock due to prolonged asphyxia.
- Dose: 5-20 μg/kg/min continuous I.V. infusion

If no improvement despite previous medications.

Always check:

1- Bag deliver 100% O_2.

2- Endotracheal tube is patent& well placed.

3- Head is not overflexed

4- Adequate ventilation pressure.

5- No air leaks (e.g. pneumothorax)

6- Adequate cardiac massage.


DEVELOPMENTAL
REFLEXES
(Primitive Reflexes)

IDEA

- Cerebral cortex in newborn is not fully developed → subcortical centres (spinal cord or brain stem) mediate some primitive reflexes → with time → maturation of the cerebral cortex occur → successive disappearance of these reflexes.
- They appear prenatally at variable gestational ages and disappear posnatally during the first year of life as cerebral cortex matures.

General significance of the primitive reflexes:

1- **Absence** at the time they should present signify damage to the subcortical concerned areas.

2- **Presence** at the time they should disappear signify failure of development of the cortical area which suppress the reflex.

1- Moro reflex:

Time: Present at birth and disappear by 5-6 months

Stimulus:

i- Sudden dropping of the head from semisiting position in examiner hand.(avoided in preterm & suspected intra cranial hemrrhage)

ii- Making a loud noise near the ear

iii- Sudden withdrawal of the blankets from underneath the infant.

Response:

- Extension of the trunk.
- Extension and abduction then flexion and Adduction of upper limbs with little share of lower limbs (embracing movement).
- Loud crying follow.

Moro reflex

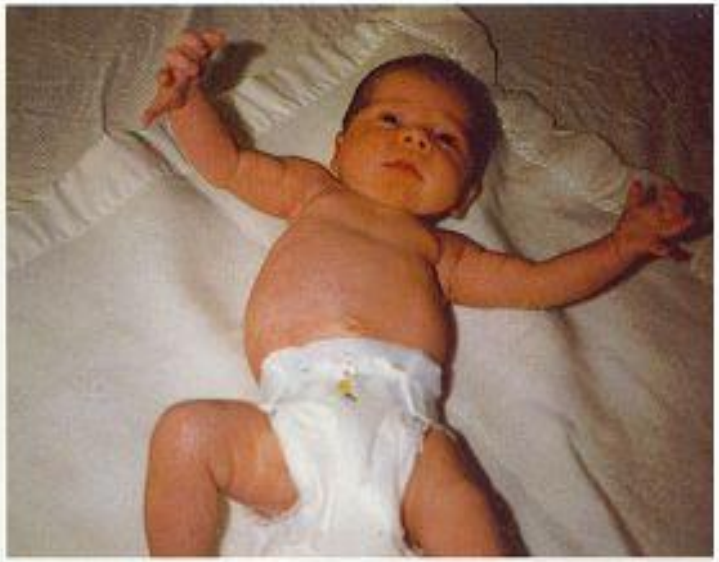


FIG. 2.50 The Moro reflex. To elicit the reflex, the head is supported and allowed to drop to the level of the bed (left). *The initial extension response to vestibular stimulation is shown at the right. Th plete response includes secondary flexion and cry.*

Value:

1- Normal reflex in normal time → normal CNS.

2- Absence:

a- Bilateral:

- Premature < 28 weeks
- CNS: - Depression by anoxia, narcotic or anaesthesia
  - Intra cranial hemorrhage
- Bilateral injury to: - Brachial plexus
  - Clavicles or humerus.

b- Unilateral (Asymmetrical):

- Erb's palsy
- Fracture clavicle or humerus
- Dislocated shoulder.

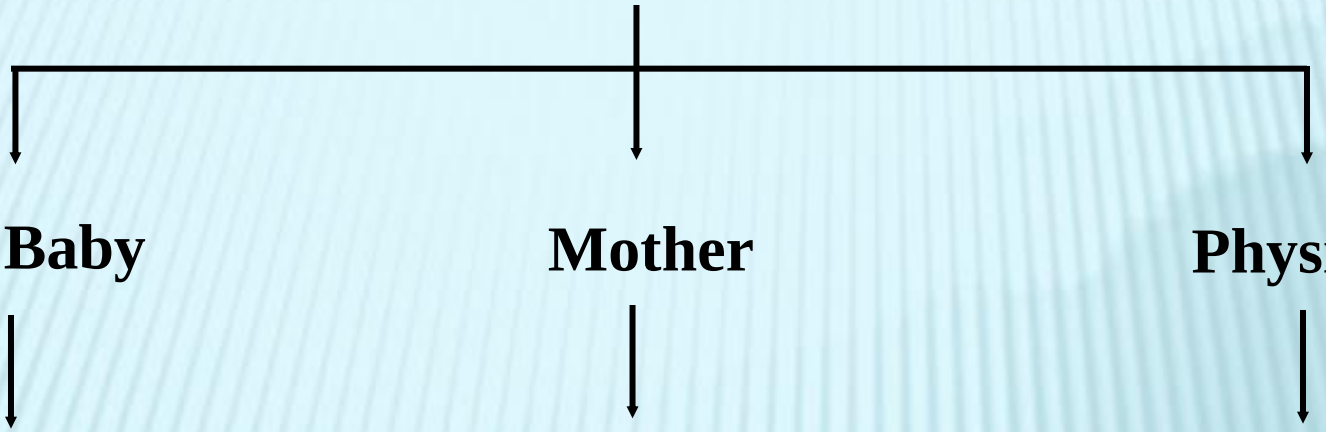
3- Sluggish response in:

- Sedation
- Sepsis.
- Early kernicterus

4- Exaggerated reflex: → CNS irritation as in late kernicterus.

5- Persistence: beyond 6 months → cerebral palsy.

Risk Factors Of Birth Injuries



Baby

- Prematurity.
- Anomalies
- Malpresentation.
- Macrosomia.

Mother

- Oligohydramnios.
- Contracted pelvis.
- Prolonged labour

Physician

- Instrumental delivery

Cranial Injuries:

i– Scalp lesions

1- Caput succedaneum

Nature : Subcutaneous extraperiosteral fluid collection, occasionally hemorrhagic.

Onset : Immediate after birth.

Site : Over the presenting part.

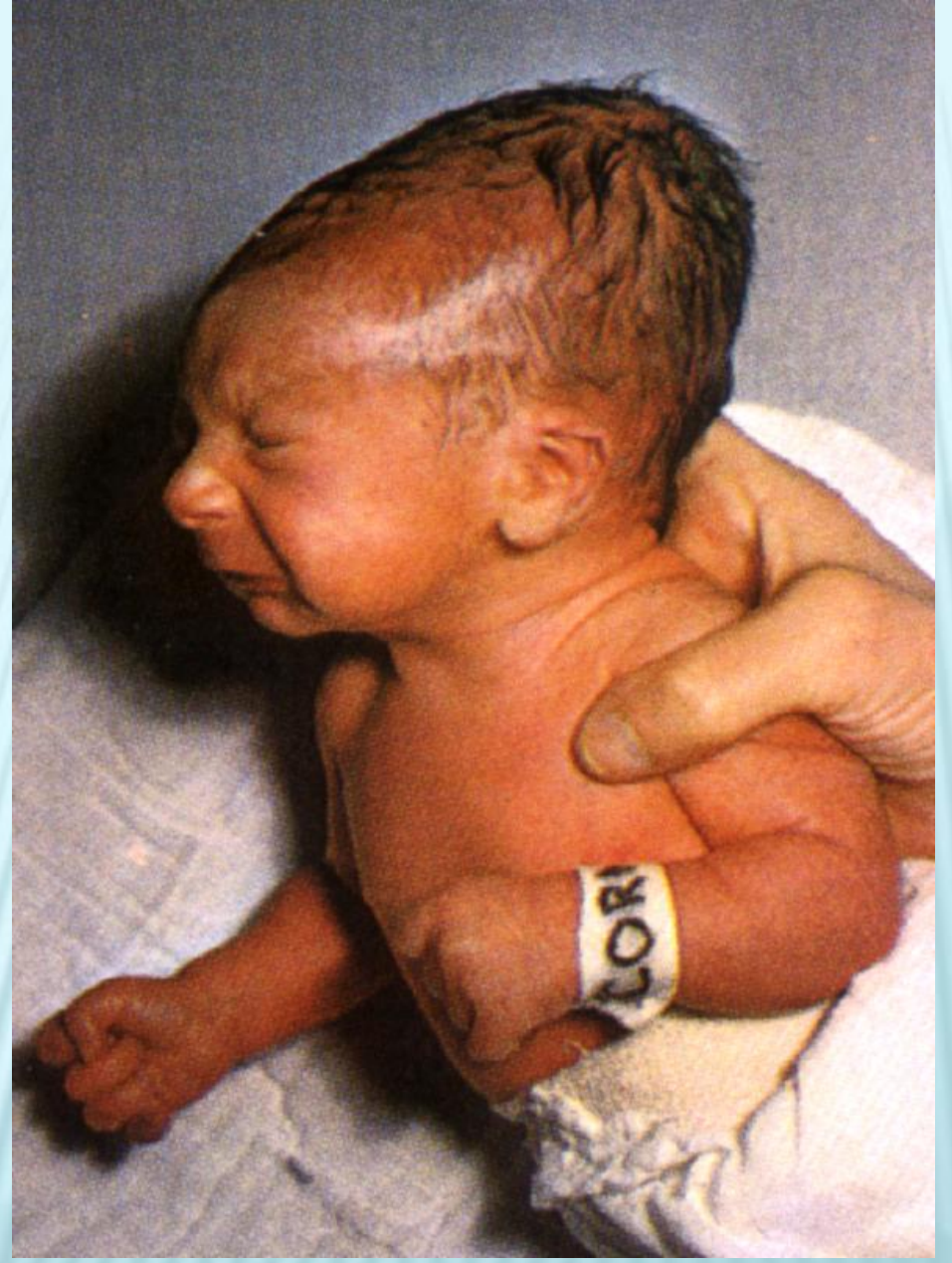
Extent : Diffuse (cross the suture lines)

Consistency: Soft

Associations: Ecchymotic skin pathches

Fate : Usually disappear gradually within few days

Treatment : Nothing.



Caput succedaneum

2- Cephalhematoma:

Nature : Sub-periosteal blood collection.

Onset : Few hours after birth.

Site : Any bone (commonly parietal or occipital)

Extent : Localized (the sutures prevent its spread).

Consistency: Firm.

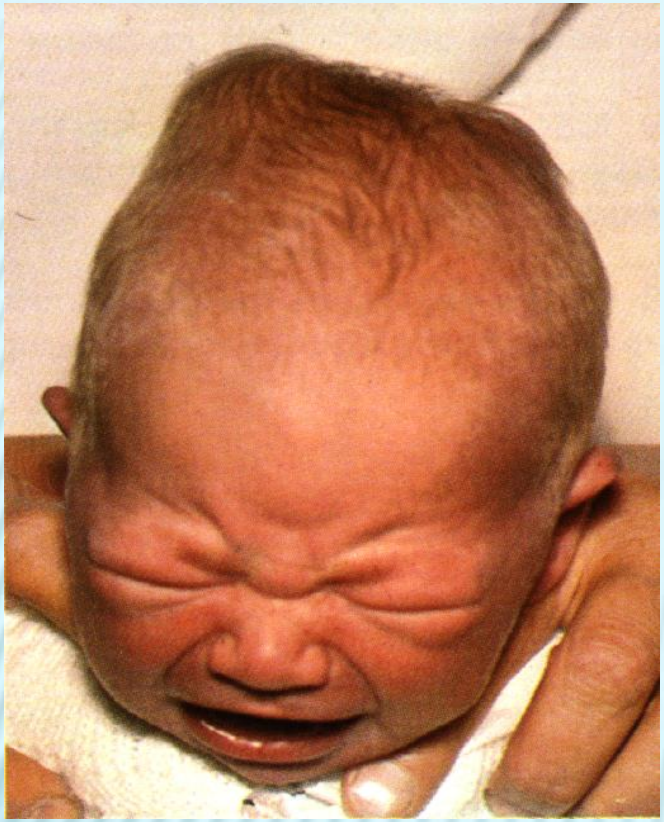
Associations: - Skull fractures in 5-20 %
- Anemia and jaundice (if large).

Fate : Resolve spontaneously over 8 weeks (infection, calcification or ossification may rarely occur).

Treatment : 1- Observation

2- treat
- anemia (blood transfusion),
- jaundice (± phototherapy)
- infection (antibiotics)

3- Incision and drainage are contraindicated. (Except if infected)



Cephalhematoma

(unilateral)

Cephalhematoma

(Bilateral)

3- Subgaleal hematoma:

- Bloody collection in subgaleal space (potential space between periosteum and aponeurosis). It extends from orbital ridges anteriorly to the occiput posteriorly and up to ears laterally.
- Very soft
- May lead to anemia, jaundice, shock.

Nerve Injuries:

1– Facial nerve injury:

Due to : Compression during delivery by forceps but may be unrelated to trauma

C/P : Mainly peripheral facial nerve injury result in paralysis of whole face on the same side:

- No nasolabial fold.
- Asymmetric cry.
- Deviation of the mouth to healthy side.
- Inability to close the eye firmly.

ttt:

- Care of the eyes with → eye drops & ointment.
- Care of feeding
- Physiotherapy → if persist more than 3 months → neuroplasty.

FACIAL PALSY (RIGHT)

2– Brachial plexus injury:

Due to: Traction over the head or the shoulders during delivery.

C/P :

1- Injury to upper nerve roots (C_5, C_6) ⇨ Duchenne-Erb's palsy

2- Injury to lower nerve roots (C_7, C_8, T_1) ⇨ Klumpke's palsy.

3- Entire brachial plexus injury → the affected limb is flaccid with loss of all reflexes.

Duchenne-Erb's palsy:

Affected muscles:

- Deltoid → loss of abduction
- Supra and infraspinatus → loss of external rotation.
- Biceps and supinator → loss of supination.

⇓

The net result will be adduction, internal rotation and pronation

→ **Waiter's tip posture**

right Duchenne-Erb's palsy (Waiter's tip posture)

Reflexes:

- Absent Moro and preserved Grasp reflex on the affected side

Association:

- Impaired sensation over the external surface of the upper limb.
- May be associated with phrenic nerve palsy (C_3, C_4, C_5).

Treatment:

1- Partial intermittent immobilization in position opposite to the lesion i.e. abduction, external rotation and supination (statue of liberty splint).

2- Physiotherapy after one week (after resolution of nerve oedema) to prevent muscles contractures.

Prognosis:

- If nerve root are intact, full recovery will occur in more than
  - 90% by 3 months
- If no improvement within 3 months, consult neurosurgery for nerve grafting or neuroplasty.

Klumpke's palsy:

Affected muscles:

- Paralysis of all intrinsic
- muscles of the hand.

Reflexes:

- Absent Grasp and preserved
- Moro reflex on the affected side

Association:

- If injury to sympathetic fibres of $T_1 \rightarrow$ Horner syndrome (ptosis, meiosis, enophthaloms and anhydrosis).

Left facial palsy &right klumpeks palsy

Treatment:

1- Hand is kept in neutral position with pad of cotton in the fist → hand writing position.

2- Physiotherapy.

Prognosis:

- If nerve root are intact, full recovery will occur in more than 90% by 3 months
- If no improvement within 3 months, consult neurosurgery for nerve grafting or neuroplasty.

NEONATAL SEPTICEMIA

Physical defences: skin is thin and easily damaged, the umbilical stump becomes necrotic and acts as a locus for infection, insertion of tubes provides a route of entry for pathogens, and there is no protection from resident flora.

Cellular immunity: phagocytic activity of polymorphs from newborn infants is reduced in neonatal serum defi cient in immunoglobulins and complement.

Humoral immunity: the normal newborn has virtually no circulating IgA, IgD, IgE, or IgM. IgG is acquired from the mother and in the normal term baby levels are high, falling following delivery with a half-life of 3 weeks to cause transient postnatal hypogammaglobulinaemia which is more severe in preterm infants. Complement levels are low, and even lower in preterm infants.

Definition:

Clinical syndrome characterized by systemic illness with decumentation of infection (multiplication of bacteria with their toxins in the blood)

Pathogenesis:

. *Early-onset:* within 72 h of birth

Late-onset: after 3–7 days.

The risk factors and aetiology differ for each category.

Early-onset sepsis

This results from vertical infection during birth. Common pathogens include GBS and *E. coli*.

Risk factors include:

Preterm delivery

Prolonged rupture of membranes

Maternal pyrexia or infection

Chorioamnionitis

Previous infected infant

Late-onset sepsis

Infections presenting late may be vertically acquired but transmission is more often horizontal by transmission from person to person.

Risk factors include:

Preterm delivery

Indwelling catheters (intravascular) or endotracheal tube

Prolonged antibiotic treatment

Damage to skin.

CONS is often recovered from the blood stream in infants with indwelling catheters.

Clinical features

The history may reveal risk factors such as maternal pyrexia. A host of symptoms, signs or laboratory findings may be early warnings of sepsis:

Temperature change: temperature <36°C or >37.5°C sustained for >1 h in an appropriate environmental temperature

Respiratory: apnoea or tachypnoea with respiratory distress or increased ventilatory requirements

Cardiovascular: tachycardia, prolonged capillary return (>3 s), pallor, mottled skin

Gastrointestinal: slow feeding, vomiting, abdominal distension, ileus, intestinal obstruction

CNS: lethargy, irritability, hypotonia, seizures

Local signs: pseudoparalysis due to limb pain, tense fontanelle, head retraction.

2- Early manifestations ⇒ Non specific = not doing well baby

- Respiratory distress and apneic attacks.
- Lethargy
- Poor feeding and vomiting
- Unstable temperature (mainly hypothermia)
- Poor Moro and suckling reflexes

3- Late manifestations = focal infections

- Pneumonia: respiratory distress
- Meningitis: seizures, tense bulging fontanelle.
- Hepatitis: direct hyperbilirubinemia
- Pyelonephritis
- Arthritis.

4- Complications:

- Septic shock :- hypotension
  - decreased peripheral perfusion (cold mottled skin)
  - metabolic acidosis
  - oliguria,.
- Necrotizing enterocolitis (NEC).
- DIC → Bleeding from puncture sites.
  → Purpura, may be skin gangrene
- Sclerema = hardening of the skin → poor prognosis.

Investigations:

1- Sepsis screen: Septicemia is suggested when:

i- CBC: show:

- Leucopenia < 5000/mm^3 & neutrophil count < 1000/mm^3 (with severe sepsis)
- Toxic granulations in neutrophils.
- Bandemia: Band cells (immature)>20% of total neutrophil count.
- Less commonly leucocytosis (> 25.000 / mm^3)

ii- Serial determination of C-reactive protein (CRP)

2- Identification of the causative organism:

- Blood cultures over aerobic & unaerobic media.
- Lumbar puncture: CSF analysis, culture and gram stain.
- Cultures of urine, stool and endotracheal aspirate.
- Detections of GBS and E-Coli antigens in urine and CSF by latex agglutination.

Differential diagnosis:

1- Other causes of respiratory distress.

2- Other causes of lethargic neonate

3- Other causes of neonatal vomiting.

Management:

Prophylaxis:

Maternal intrapartum ampicillin if there is risk factors.

Curative:

1- **Incubator care in neonatal intensive care unit (NICU):** for

- Warming.
- Support respiration: → O_2 inhalation
  → Mechanical ventilation support.

Diagnosis should be madebefore late signs emerge such as cyanosis, bilious vomiting, bulging fontanelle, shock, and DIC. Careful clinical examination is mandatory.

Management

Investigations

The standard 'septic screen' includes:

Full blood count, differential white count

C-reactive protein (CRP)

Microbial samples for culture: blood, urine, ear and throat swabs, endotracheal tube aspirate (if applicable)

Lumbar puncture: CSF

CXR: unless there is an obvious extrapulmonary focus.

Additional investigations to consider

Additional investigations to consider

Blood gases, U&E, calcium, albumin, coagulation screen

Maternal vaginal swab culture

Placental tissue for *Listeria monocytogenes*

Viral studies.

Results that mandate early antibiotic treatment include:

Neutrophilia or neutropaenia

Thrombocytopaenia: platelets <100 × 10 9/L

Raised CRP (may be normal initially)

Urine white cells

CSF: >21 × 10 9/L white cells, protein >2 g/L in term infants, glucose <40% blood glucose, organisms seen.

. An aminoglycoside such as gentamicin, tobramycin, amikacin, or netilmicin is usually indicated to cover coliforms. A second or third antibiotic is given depending on which organisms are common:

GBS: penicillin G

CONS: fl ucloxacillin, vancomycin

Pseudomonas aeruginosa: ceftazidime, piperacillin

Anaerobes: metronidazole.

Once culture and sensitivity results are available changes can be made. If blood cultures are negative, CRP remains normal, and signs of sepsis resolve antibiotics are stopped after 48 h. Proven infection is treated for 7–10 days, rising to 14 days in gram-negative infection, 21 days in meningitis, and 3–6 weeks in septic arthritis/osteomyelitis.

Thank You

Thank You