

Neonatal neurology



Developmental process of the human brain

Neuronal proliferation	-----	--	--	--	--	--
Neuronal migration	--	-----	--	--	--	--
Outgrowth of axons	--	--	-----	-----	-----	-----
Synapse formation	--	--	-----	-----	-----	-----
Glia cell proliferation	--	--	--	-----	--	--
Myelination	--	-----	-----	-----	-----	-----
Neuronal death	--	-----	--	--	--	--
Axon retraction	--	--	-----	--	--	--
Synapse elimination	--	--	-----	-----	-----	-----
	C	3M	6M	B	1 y	10y 30 y

Birth injury

Caput succedaneum

edematous swelling
 extends the sutures lines
 some days after disappears



Birth injury

Cephalhematoma

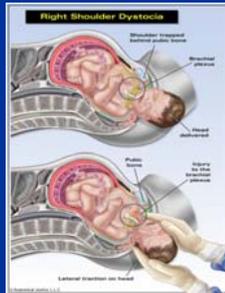
subperiosteal hemorrhage
 does not extend the suture lines
 skull fracture may occur



no therapy is needed

Birth injury

- Brachial plexus palsy
- Risk factors:
 - macrosomia
 - narrow pelvic outlet



Birth injury

Erb-Duchenne paralysis

- excessive traction of the neck
- injury of the brachial plexus (C5,C6)
- Can not abduct, externally rotate and supinate
- Biceps and Moro reflex are absent



Birth Injury

Klumpke paralysis

Injury of the brachial plexus (C7,C8)
Paralysed hand

1st th nerve injury may occur → Horner trias (ptosis, myosis, enophthalmus)



Birth injury

- Complete arm and hand paralysis (C5-C8)



Birth injury

Erb-Duchenne and Klumpke paralysis

Therapy:

Positioning to prevent contractures
Motion exercises
Electrotherapy
Surgery (neuroplasty)

Birth injury

Erb-Duchenne and Klumpke paralysis

Prognosis: complete recovery 70-90 percent

Nerve was not lacerated → good, the function will return within a few months

Nerve was lacerated → permanent damage

Birth injury

Phrenic nerve palsy

diaphragmatic paralysis → respiratory distress

Breath sounds are diminished

Fractures skull, clavicle, extremities

Spinal cord injury, subdural hemorrhage

Hypoxic-ischemic encephalopathy Etiology

Intrauterin (uteroplacental insufficiency, abruptio placentae)

Intrapartum (umbilical cord compression)

Postpartum (infections, ptx, NRDS)



Asphyxia

(hypoxia, hypercapnia, acidosis)

Hypoxic-ischemic encephalopathy

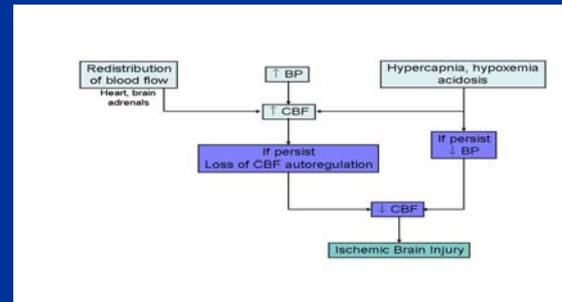
Asphyxia

↓
bradycardia → reduced tissue blood flow → diminished blood flow to the brain → ischemia

↓
increased capillary permeability → cerebral edema, petechiae

Intracranial hemorrhage may be present

Hypoxic-ischemic encephalopathy



Hypoxic-ischemic encephalopathy Symptoms

Depends on:

the gestational age

time, severity and/or duration of asphyxia



Hypoxic-ischemic encephalopathy Symptoms in term infants

Mild asphyxia (stI)

irritability, hyperalertness

muscle tone is normal

deep tendon reflexes are brisk

mydriasis

Hypoxic-ischemic encephalopathy Symptoms in term infants

Severe asphyxia (stIII-III)

letargy, stupor

hypotonia

deep tendon reflexes may be absent

myosis

seizures



Hypoxic-ischemic encephalopathy Symptoms in term infants

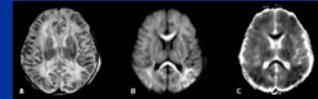
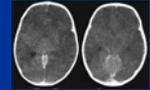
Ultrasound /MRI

- cerebral oedema

- cortical necrosis

- lesions os basal ganglias

„bright thalamus“



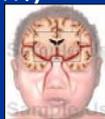
Hypoxic-ischemic encephalopathy in preterms

Circulation system and autoregulation is vulnerable, vessels have poor structural support

CBF autoregulation is narrow



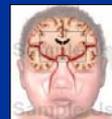
CBF become pressure-passive



Hypoxic-ischemic encephalopathy in preterms

Blood pressure's changes have a deep impact on border zone, vessels may rupture easier

- Hypercapnia, hypoxia → vasodilatation → increased cerebral blood flow
- Hypocapnia, hyperoxia → vasoconstriction → decreased cerebral blood flow



Hypoxic-ischemic encephalopathy in preterms Consequence I



Periventricular leukomalacia

symmetrical ischemic infarction of the white matter adjacent to the lateral ventricles



Hypoxic-ischemic encephalopathy in preterms Consequence II

Periventricular intraventricular hemorrhage

Vulnerable region! - periventricular, subependymal germinal matrix is highly vascularized



Hypoxic-ischemic encephalopathy Therapy

- Maintain adequate ventilation
- Maintain perfusion (dopamine, dobutamine)
- Fluid and glucose homeostasis
- Treatment of seizures (phenobarbital, lorazepam, phenytoin)

Hypoxic-ischemic encephalopathy Therapy

Hypothermia treatment (selective head or whole body cooling)

- 3-4°C below the baseline temperature
- no later than 6 hour

Possible mechanism:

- reduced metabolic rate and energy depletion
- decreased excitatory transmitter release
- reduced apoptosis and vascular permeability

Hydrocephalus internus Early complication of intracranial hemorrhage

- Head circumference grows rapidly
- Bulging fontanel
- „Setting sun phenomen“
- spasticity



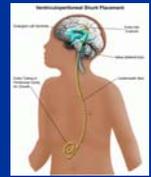
Intracranial hemorrhage Therapy

-Supportive care (ventillation therapy, transfusion, drugs)

In a case of threatening hydrocephalus:

-Serial daily lumbal puncture, external ventriculostomy

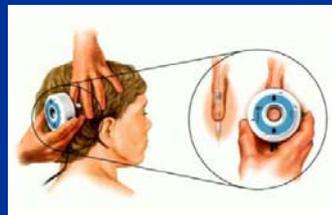
-Ventriculoperitoneal shunt



Hydrocephalus -VP-shunt



Programmable shunt with magnet



Complications in later ages

- Cerebral palsy
- Mental retardation
- Hydrocephalus
- Microcephaly
- Visual and hearing problems
- Epilepsy
- ADHD



Thank you for attention !

