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INTRAPARTUM ASPHYXIA

Intrapartum death

- Intrapartum asphyxia
- Intrapartum trauma
- Ascending infections

Intrapartum asphyxia: definition

- ⊙ Impaired gas exchange during labour
 - Hypoxemia and hypercapnia
 - Metabolic acidosis
 - BE < 12-16 mmol/l
 - pH < 7.2
- ⊙ Affects full term and pre term babies.
- ⊙ Full term infants can resist for a short time → expedite delivery

Physiology of birth

- ⦿ During uterine contraction: intermittent compression of blood vessels → brief hypoxia, normally rapidly reversed between contractions
- ⦿ Compressive forces induce increase in fetal venous pressure
- ⦿ Effects amplified in IUGR
- ⦿ Effects amplified in prolonged 2nd stage

The way it works:

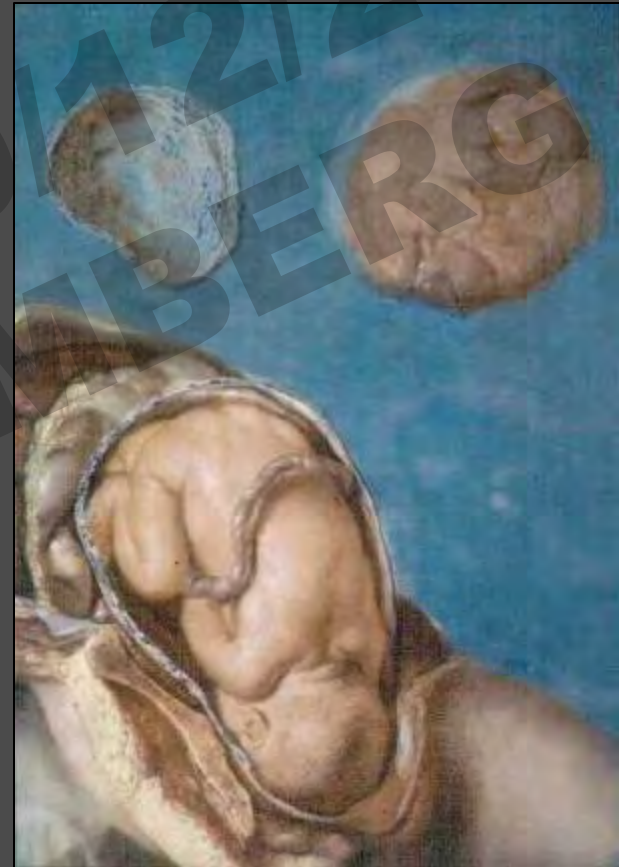
- Compression of blood vessels during uterine contraction
- Normally rapidly reversed between contractions.
- Excessive or prolonged head compression or lack of adaptation to the effects of contractions → fetal hypoxemia → acidosis → asphyxia

Effects on fetus

- Abnormal CTG
- Passage of meconium
- Reduced breathing movements followed by gasp → squames & meconium in the lungs
- Renal damage: ATN
- Coagulation problems
- Brain injury: loss of regulation with low pH

What causes intrapartum asphyxia

- ⦿ Maternal problems
- ⦿ Placental problems
- ⦿ Umbilical cord problems
- ⦿ Fetal problems
- ⦿ Mechanical problems

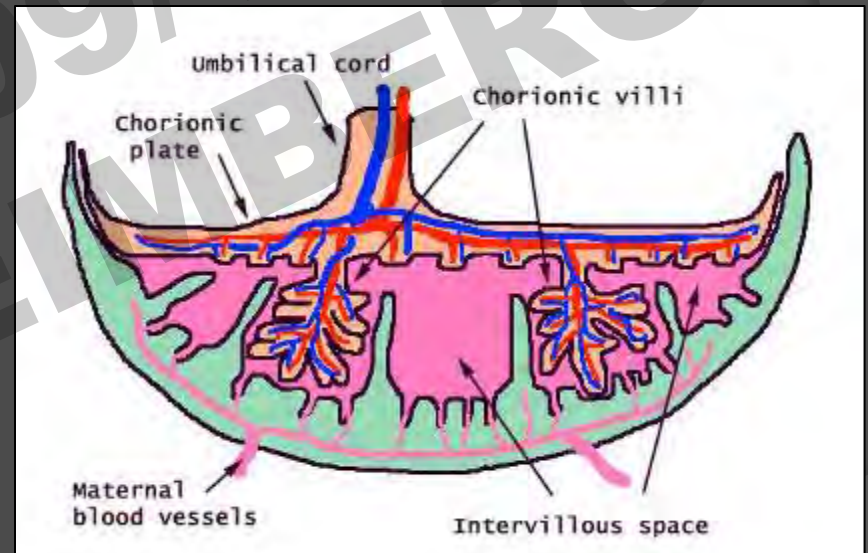


Mechanism

1. Inadequate gas exchange between maternal and fetal blood
2. Inadequate gas exchange between fetal blood and fetal tissues

1. Inadequate gas exchange between maternal and fetal blood

- a. Maternal disorders
- b. Placental disorders



a. Maternal disorders

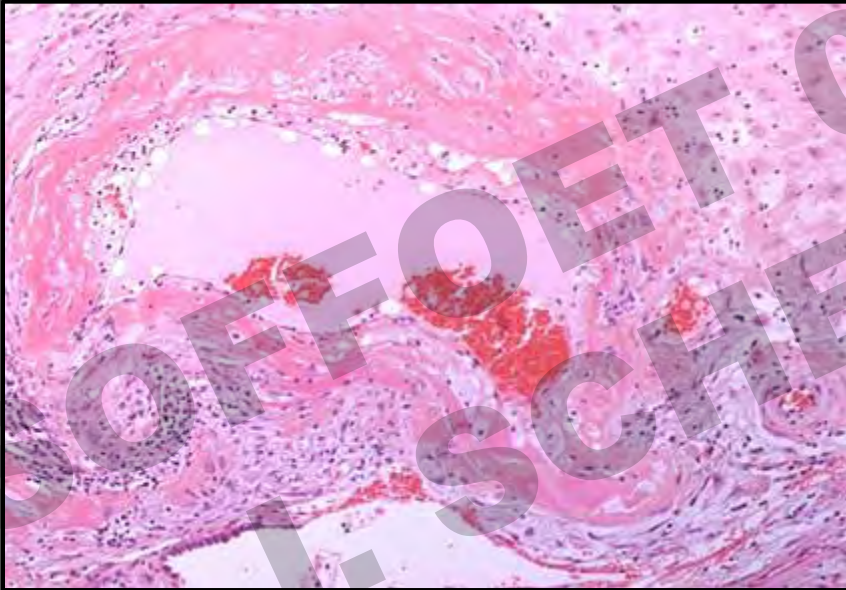
- ◉ Anaemia
- ◉ Malnutrition
- ◉ Chronic renal failure
- ◉ Heart disease
- ◉ Peripheral arterial disease
- ◉ Shock & hypoxia
- ◉ Epilepsy
- ◉ Aortic compression
- ◉ Drugs
- ◉ Pre-eclampsia
- ◉ Pregnancy cholestasis
- ◉ Uterine rupture

b. Placental disorders: limit gas exchange

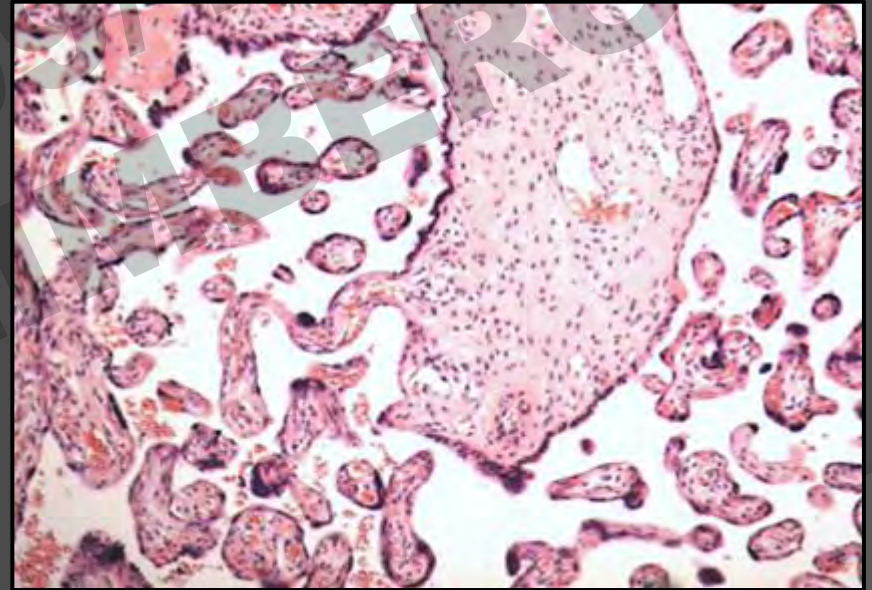
- Infarction (>10%)
- Widespread villitis
- Villous oedema
- MPVFD
- Abnl villous maturation
- Fetal vascular occlusion
- Prolonged pregnancy
- Placenta praevia
- Abruptio
- Circumvallate placenta

Pre-eclampsia

A maternal problem that can be diagnosed on the placenta



Atherosclerosis & fibrinoid necrosis

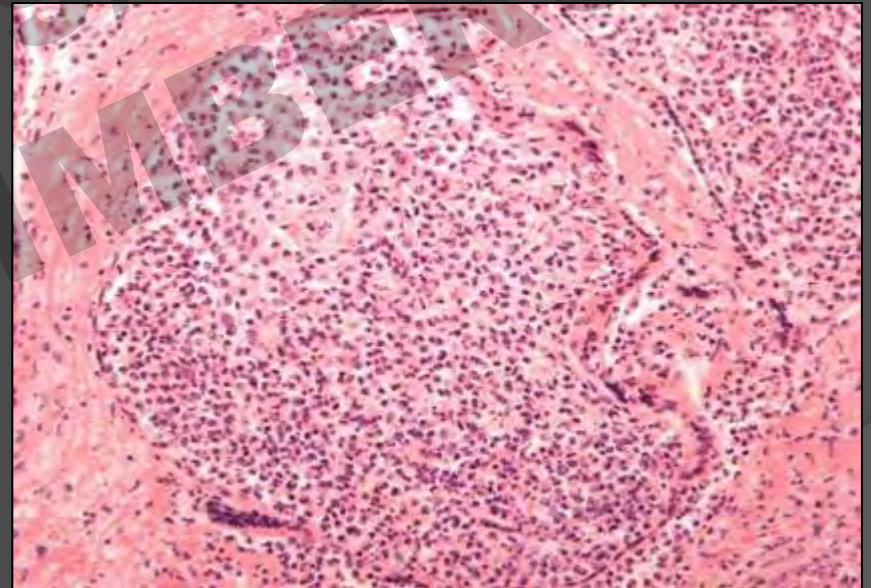


Accelerated villous maturation

Placental problems



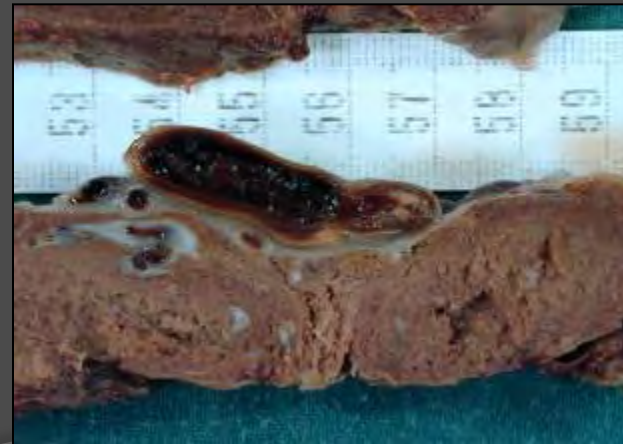
MPVFD



Acute villitis

a. Cord abnormalities (reduced fetoplacental circulation)

- Abnormal length
- Cord entanglement
- Cord knots
- Cord prolapse
- Cord compression
- Necrotizing funisitis
- Vessel thrombosis
- Vessel aneurysm
- Velamentous insertion



b. Fetal conditions

- Fetal cardiac failure
- Chronic fetal anaemia (hydropic)
- Acute fetal haemorrhage (pale)



Mechanical causes

- Fetal malpresentation
- Excess fetal size
- Maternal pelvic abnormalities
- Reduced distensibility of the birth canal
- Prolonged labour
- Excessive uterine contractions

INTRAPARTUM ASPHYXIA AUTOPSY FINDINGS

Early deaths

Late deaths

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Early deaths

External examination

- Meconium staining
- No maceration if < 8h
- IUGR/macrosomia
- Hydrops
- Fixed flexion deformities
- Pale/congested body



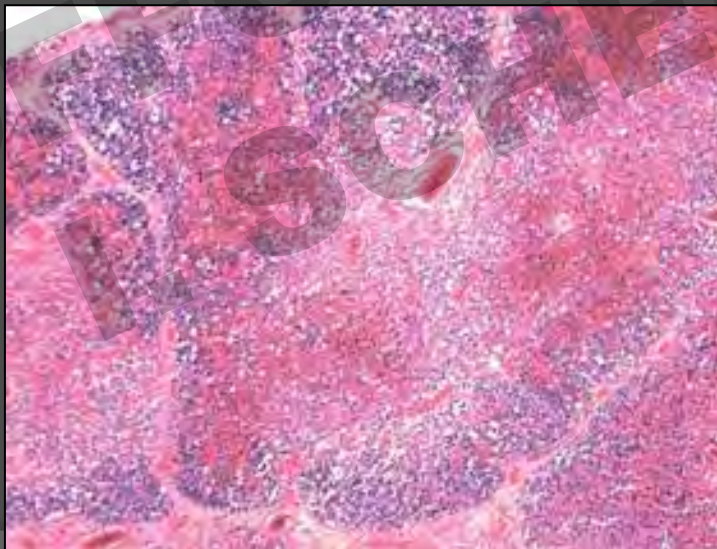
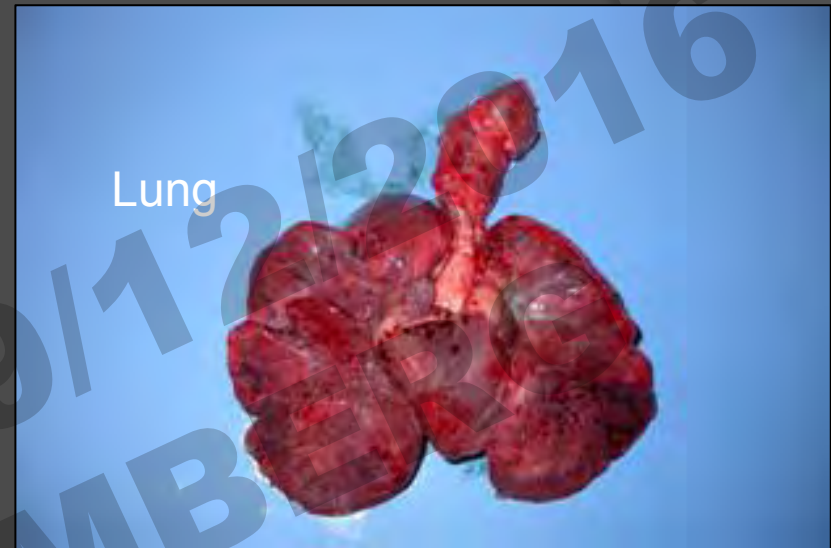
Early deaths

Internal examination

- ◎ Petechial haemorrhages



More in abruption



Lungs

- Non inflated
- Subpleural haemorrhages
- Patchy distension (CPR)
- Meconium stain
- Sometimes massive haemorrhage



Brain

Oedema

Haemorrhage in falx

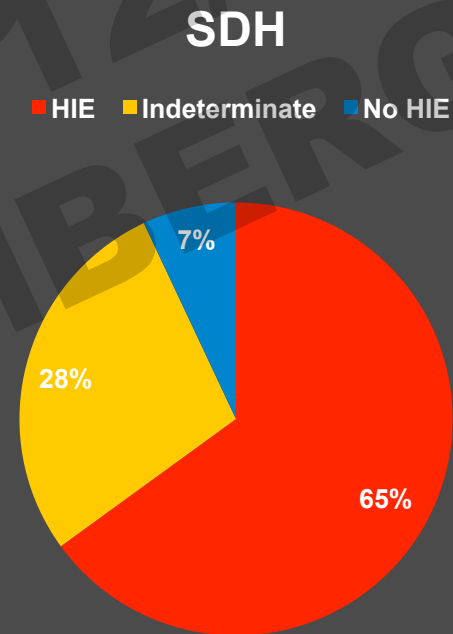
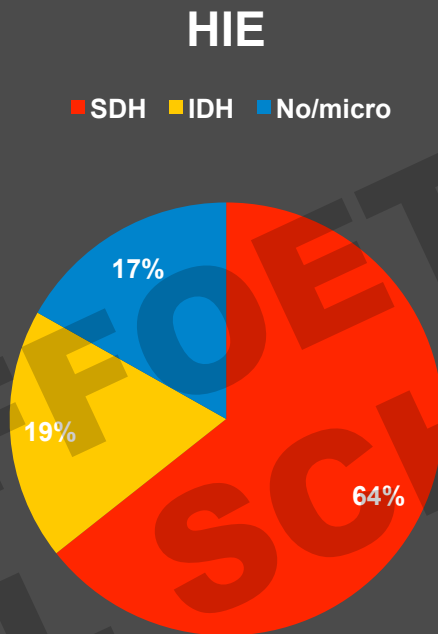
Subdural haemorrhage

IVH in prems



Subdural haemorrhage

- Strong association with birth asphyxia



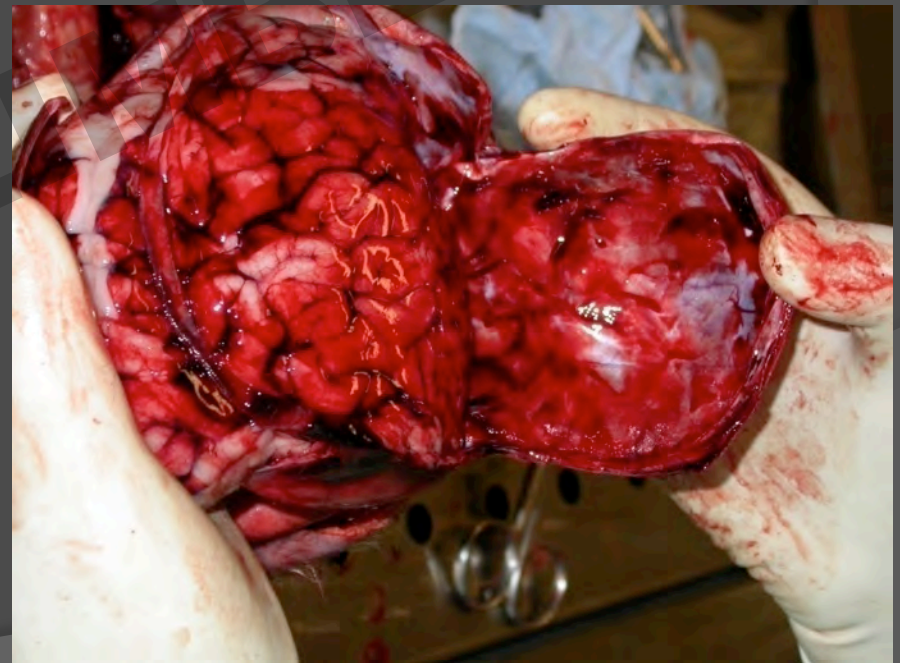
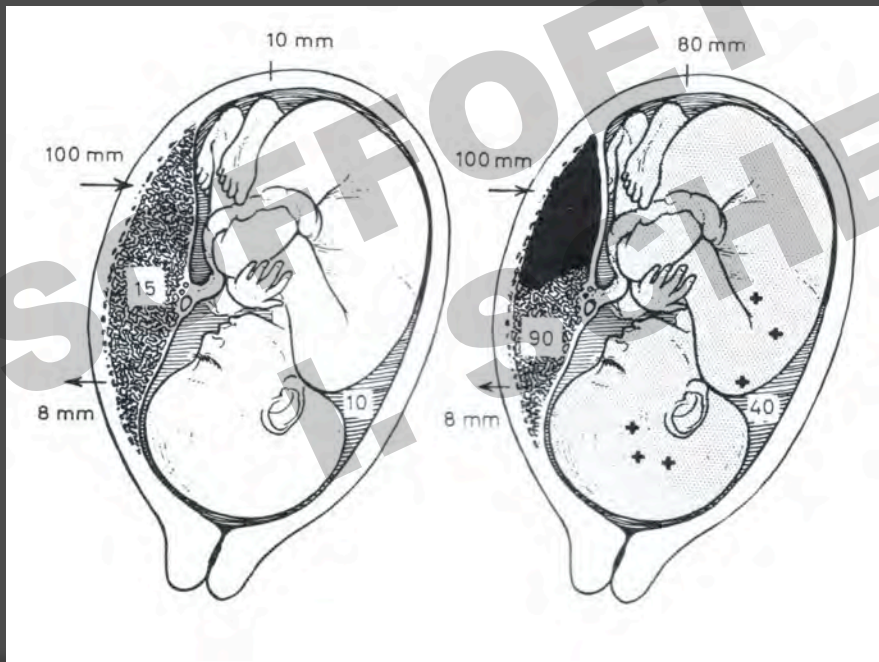
- May be asymptomatic

SDH: large versus small bleeding

- ⊙ Large bleeding: associated with trauma
 - Rupture of bridging veins due to asymmetrical compression
 - Tearing of tentorium or falx
 - Obstruction (& rupture) of vein of Galen
- ⊙ Thin film bleed: associated with hypoxia or asymptomatic
 - capillaries in falx and tentorium

Placental abruption

Interruption of venous return with retrograde increase in venous pressure and intracranial and intrathoracic hemorrhages



Asymptomatic bleeding

SDH is common along the interhemispheric fissure and over the convexities on MRI

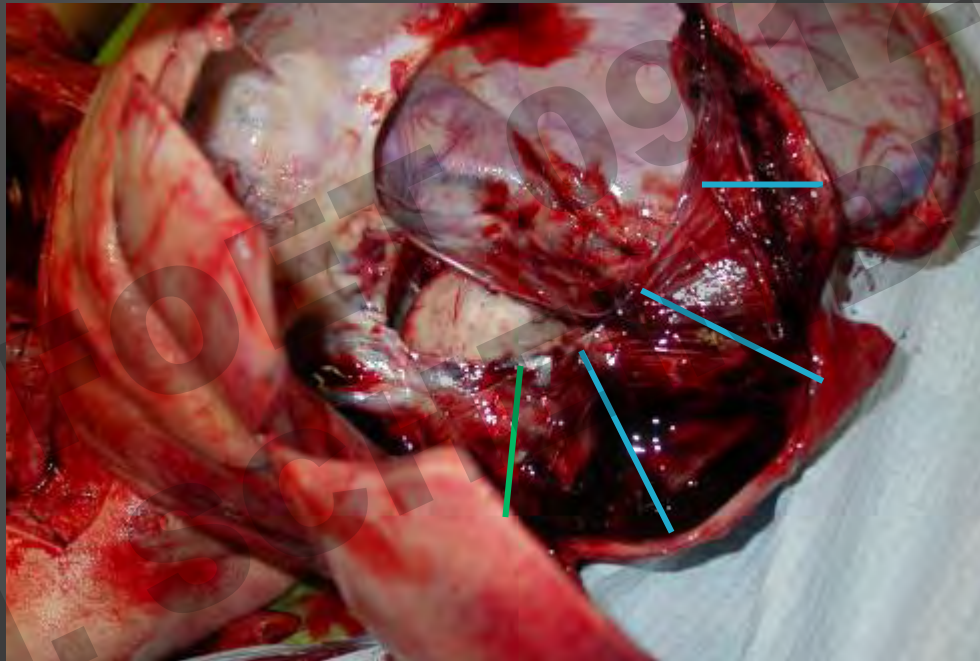
Diffuse falcine IDH explains MRI images



More about SDH

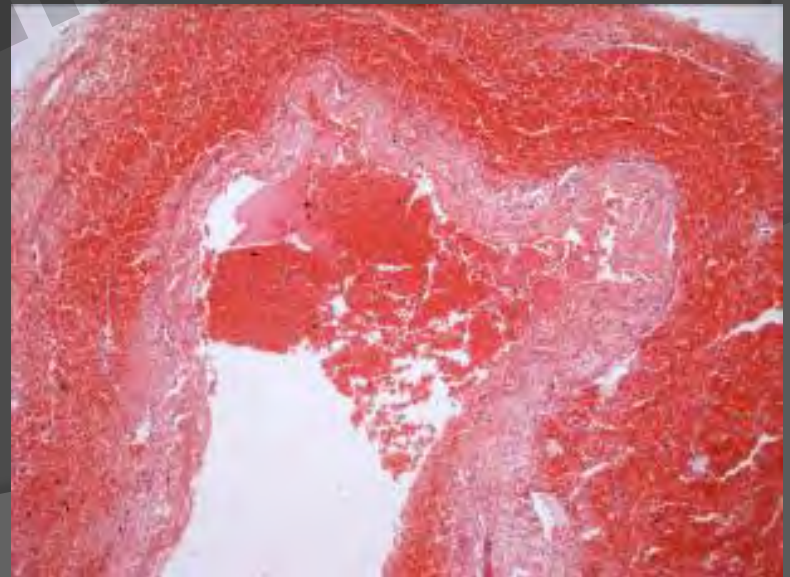
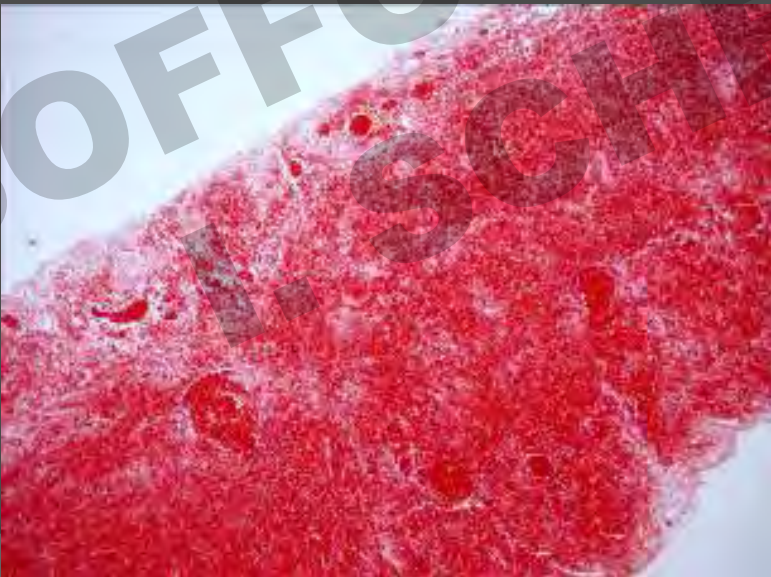
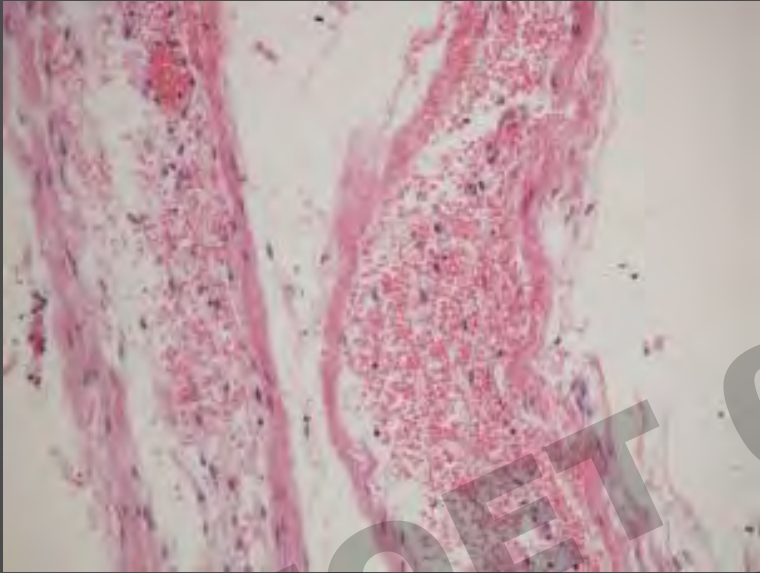
- ⦿ Small bleed in posterior fossa can cause brain stem compression & respiratory arrest
- ⦿ Presence of tear indicates severe trauma
- ⦿ Remember: intracranial bleeding is not always due to trauma
 - Medical disorders (thrombocytopenia, etc)
 - Hypoxia (especially in abruption).
 - Normal vaginal delivery

Sampling



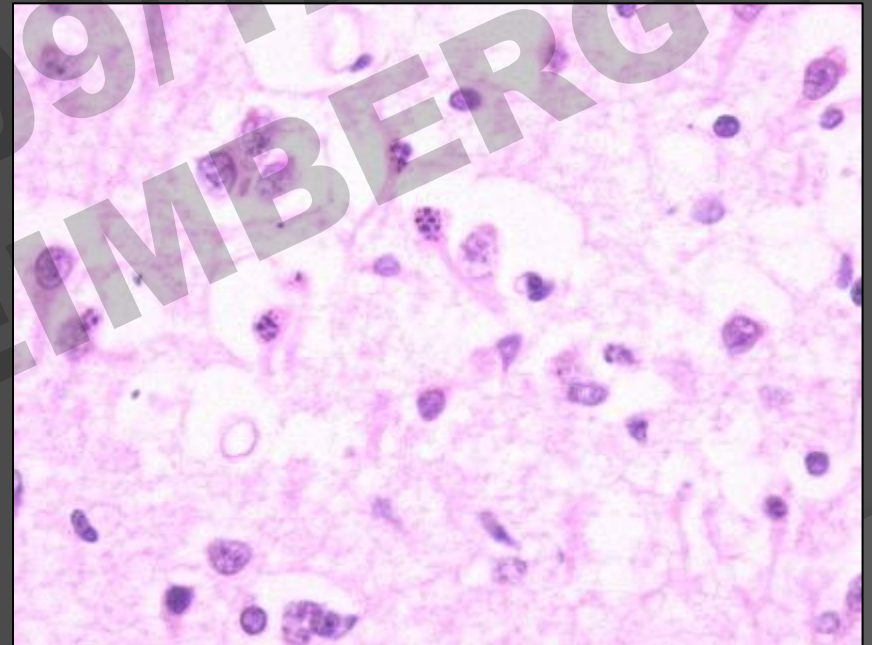
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Grading



Histology of the brain

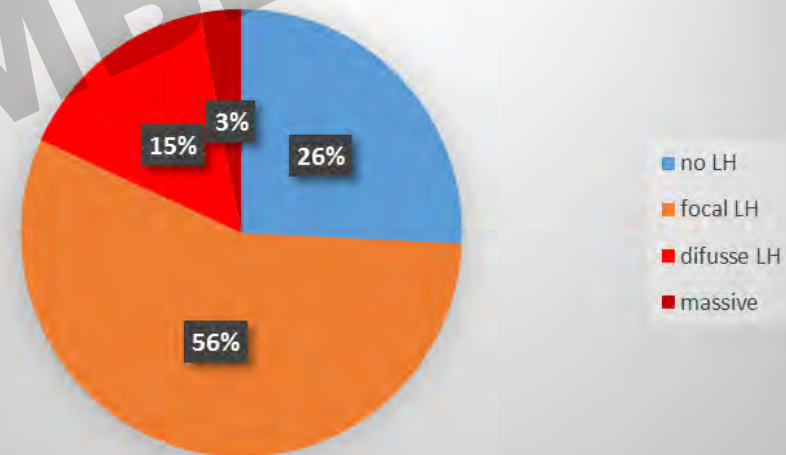
- Cytoplasmic vacuolation in white matter
- Neuronal pyknosis and apoptosis
- Other evidence of hypoxia



Lung haemorrhage



Peripartum asphyxia



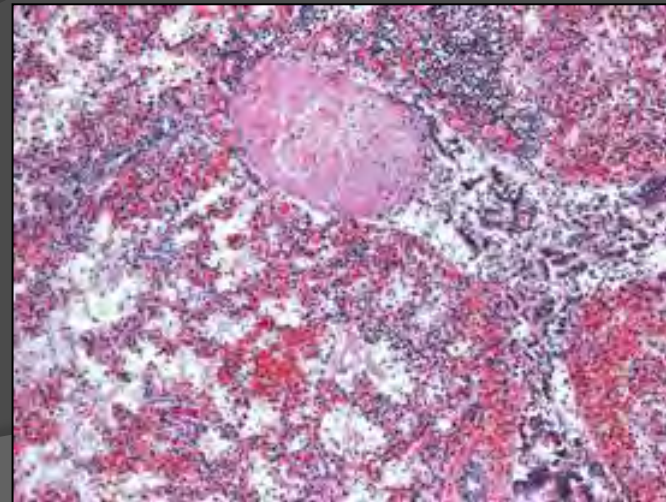
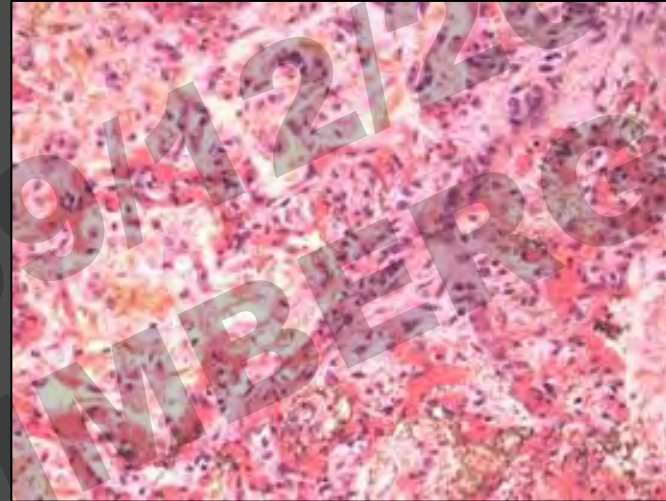
Hemorrhages in other organs

- Adrenal haemorrhage
- Renal and paratesticular hemorrhage
- Sub-capsular hepatic haemorrhage may induce severe shock



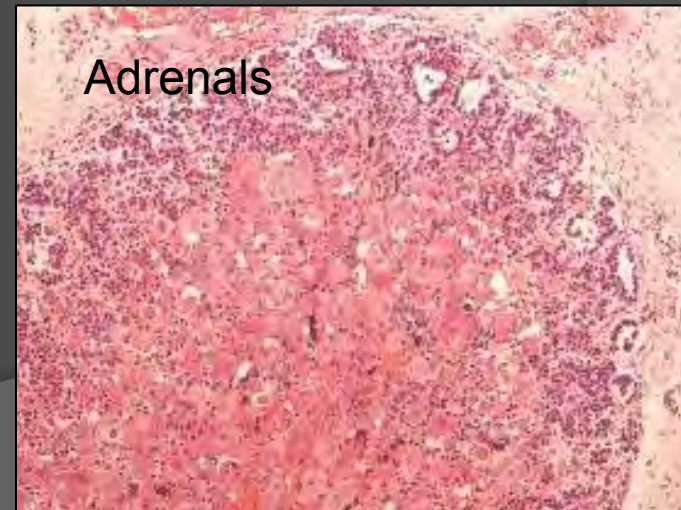
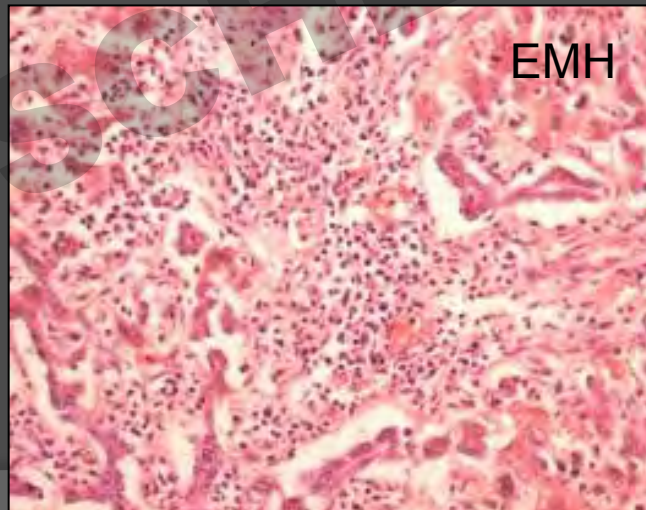
Histology of the lungs

- Squames and squamous plugs
- Meconium
- Haemorrhages
- Check capillary and vein distribution
- Exclude infection



Histology of other organs

- Thymus stress related changes
- Adrenal stress related changes
- Circulating NRBC and EMH



Late deaths

- ⦿ Infants that survive for some days after intrapartum asphyxia
- ⦿ Intense congestion generally resolved
- ⦿ Meconium staining in skin and respiratory tract not prominent
- ⦿ No meconium in stomach

Brain

- Swollen with flattened convolutions
- Marked cortical pallor
- Congested sub-cortical white matter.
- Pattern of damage depending on infant's age and duration of asphyxia



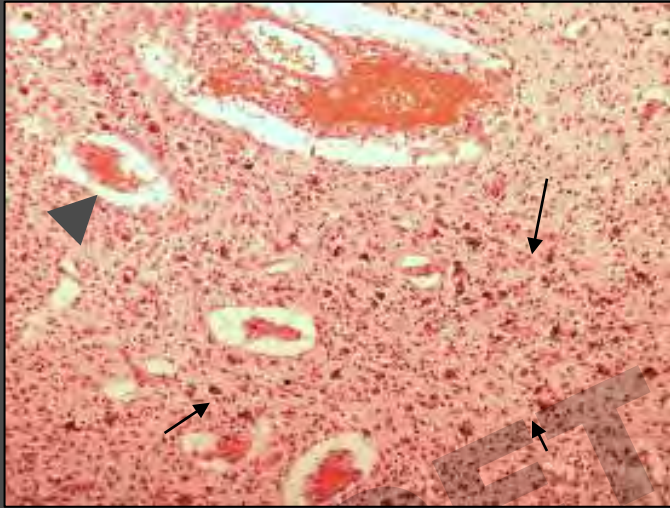
Hypoxic-ischaemic encephalopathy in full term infants

- Bilateral damage to cortical grey matter in watershed areas in depth of sulci
- Shrinkage and gliosis after several weeks
- Cellular necrosis and apoptosis
- Capillary proliferation and mineralization of necrotic neurons
- Multifocal ischaemic necrosis

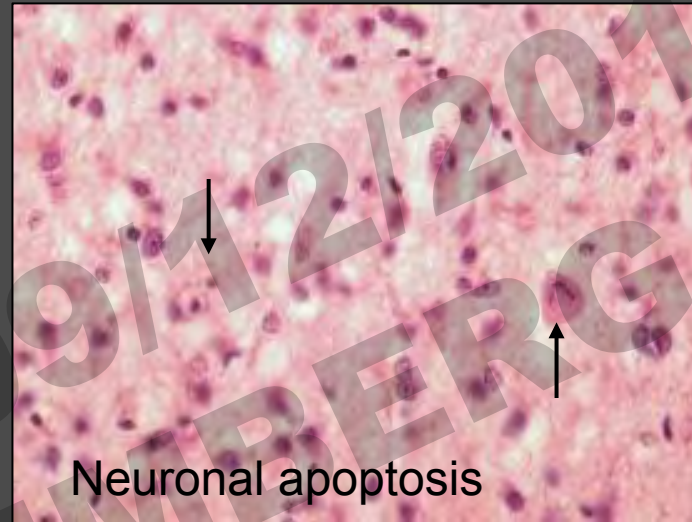
Approximate timing of hypoxic lesions

- Microglial proliferation 3h-3d
- Macrophages 4-5d
- Astrocyte proliferation 12h-4d
- Astrocyte fibrillary gliosis 6d
- Endothelial swelling 1-3d
- Endothelial reduplication 5d
- Neuronal apoptosis 12-48h
- Mineralization 8-14d

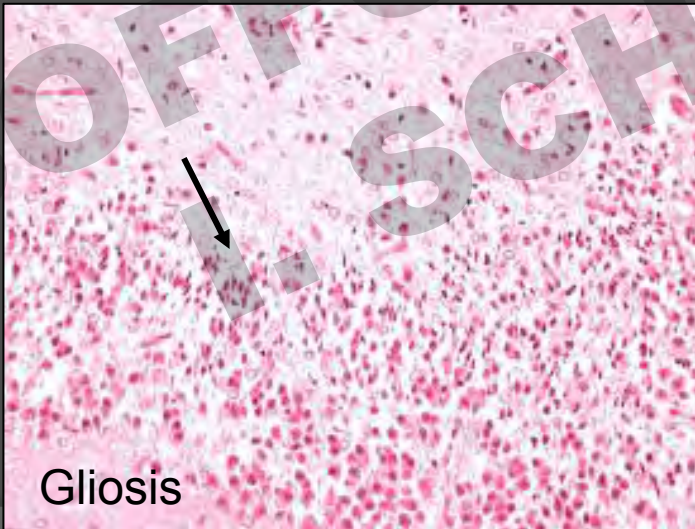
Brain hypoxia



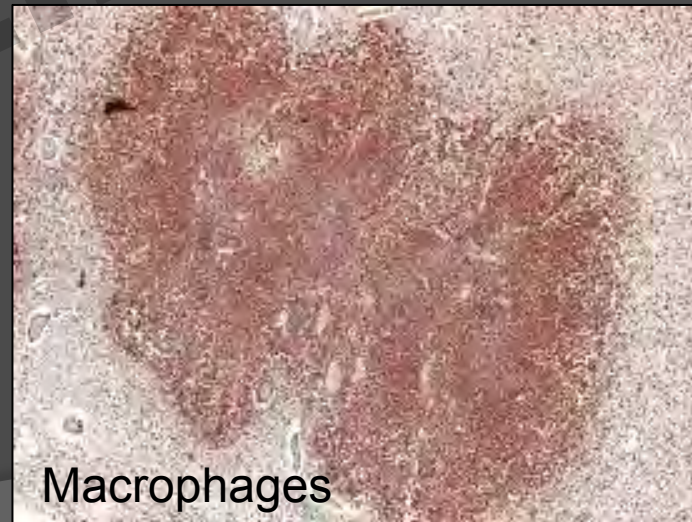
Capillary reduplication + calcification



Neuronal apoptosis



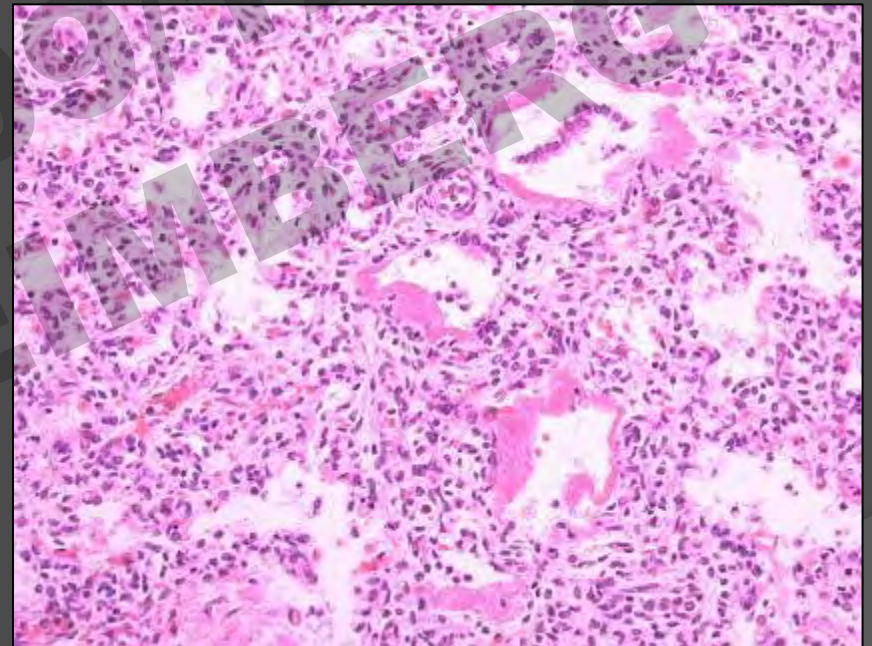
Gliosis



Macrophages

Lungs

- Aspirated squames last for many weeks
- HMD
- Small thrombi in pulmonary arterioles
- Minor inflammation
- Arteriole muscularization



Heart & kidneys

⊙ Heart

- focal papillary muscle necrosis

⊙ Kidneys

- ATN
- massive parenchymal haemorrhage (rare)
- medullary necrosis (rare)



Liver and gut

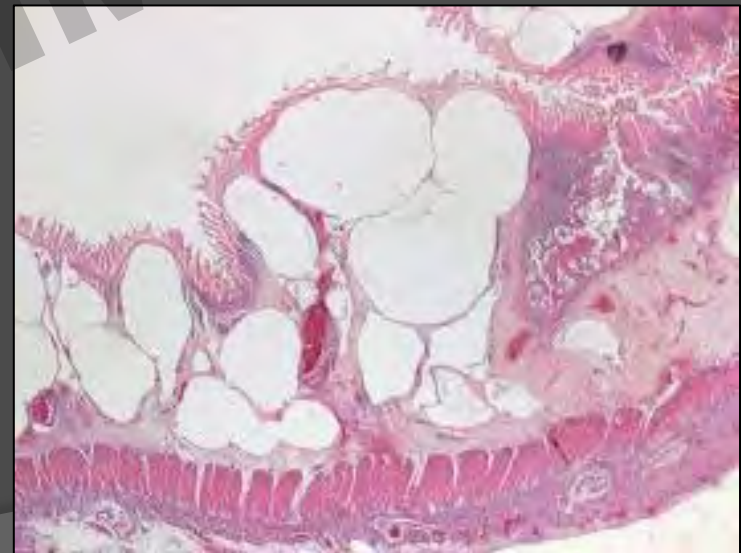
⊙ Liver

- fatty change (R>L = ante-partum anoxia)
- perivenular necrosis



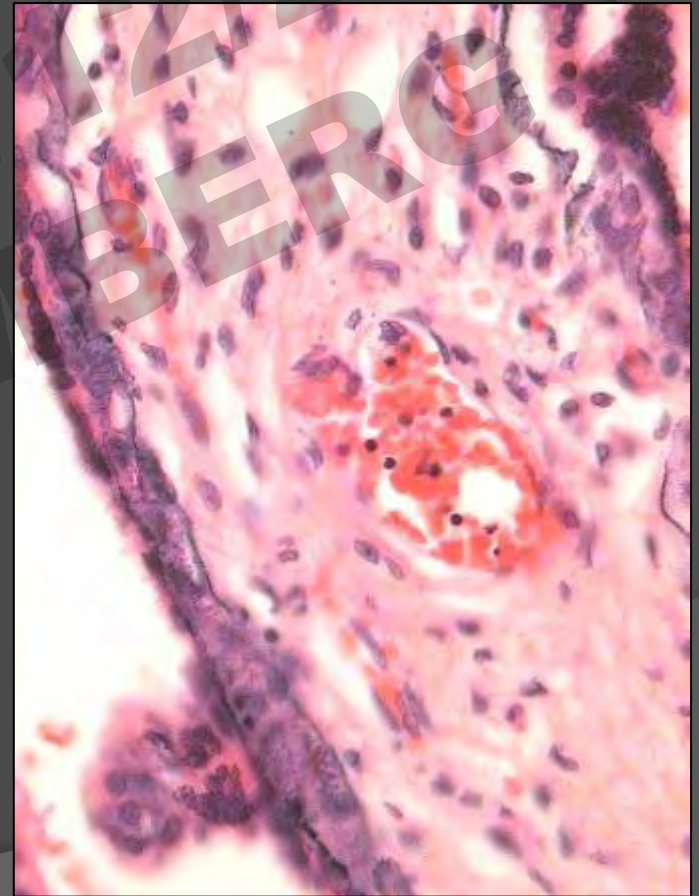
⊙ Intestines

- necrotizing enterocolitis (especially prems)



Placental examination

- May show prenatal onset of hypoxia
- Cause of death found by PM alone in only 47%
- Placental study needed in another 34%



IUGR and perinatal asphyxia

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Genetic factors



Baby's factors



Placental factors



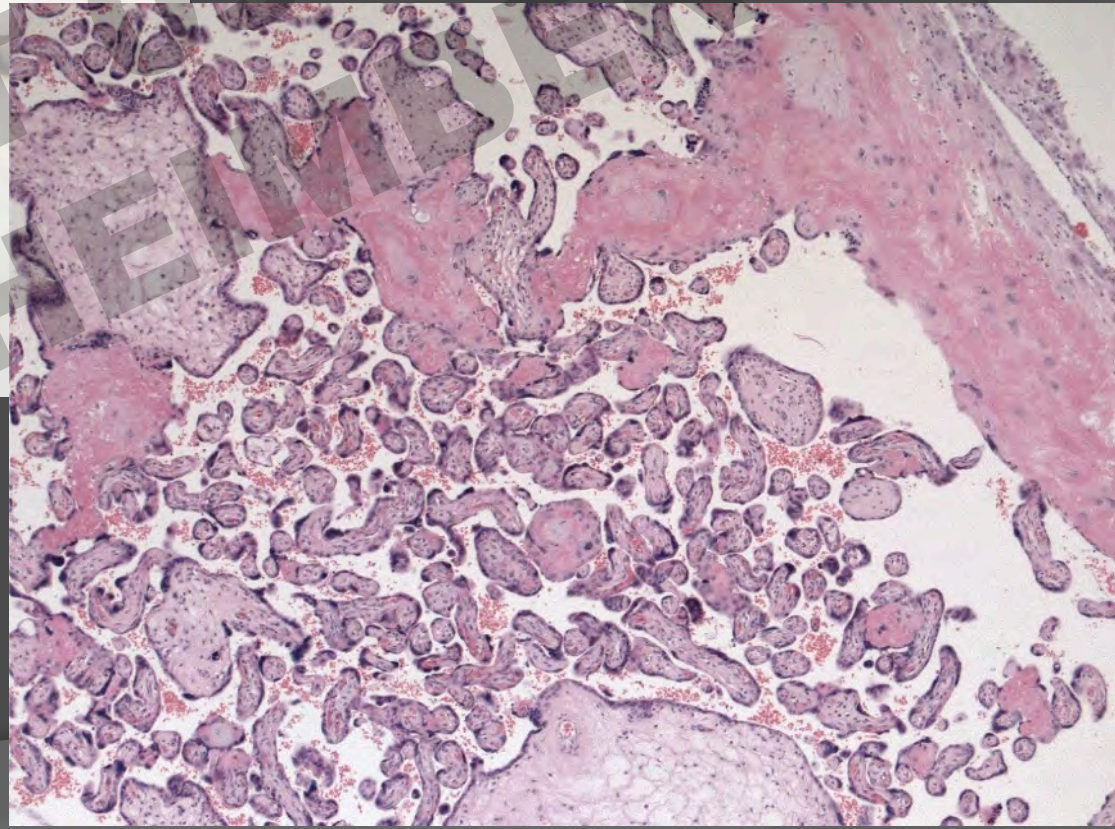
Maternal factors



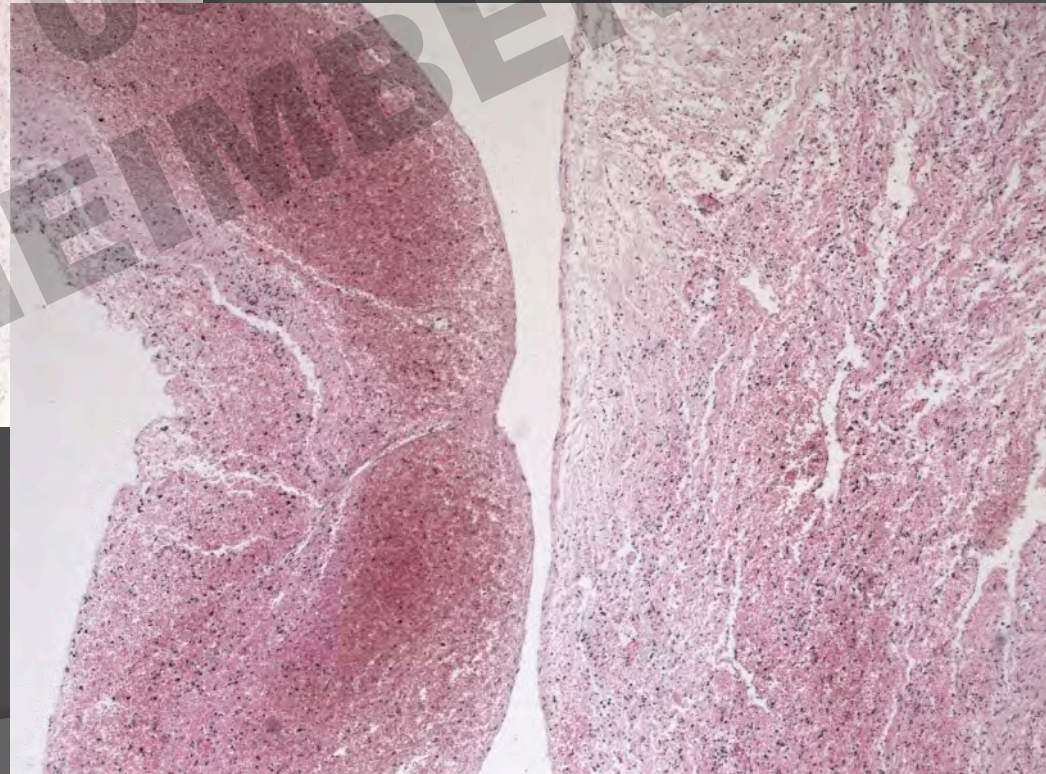
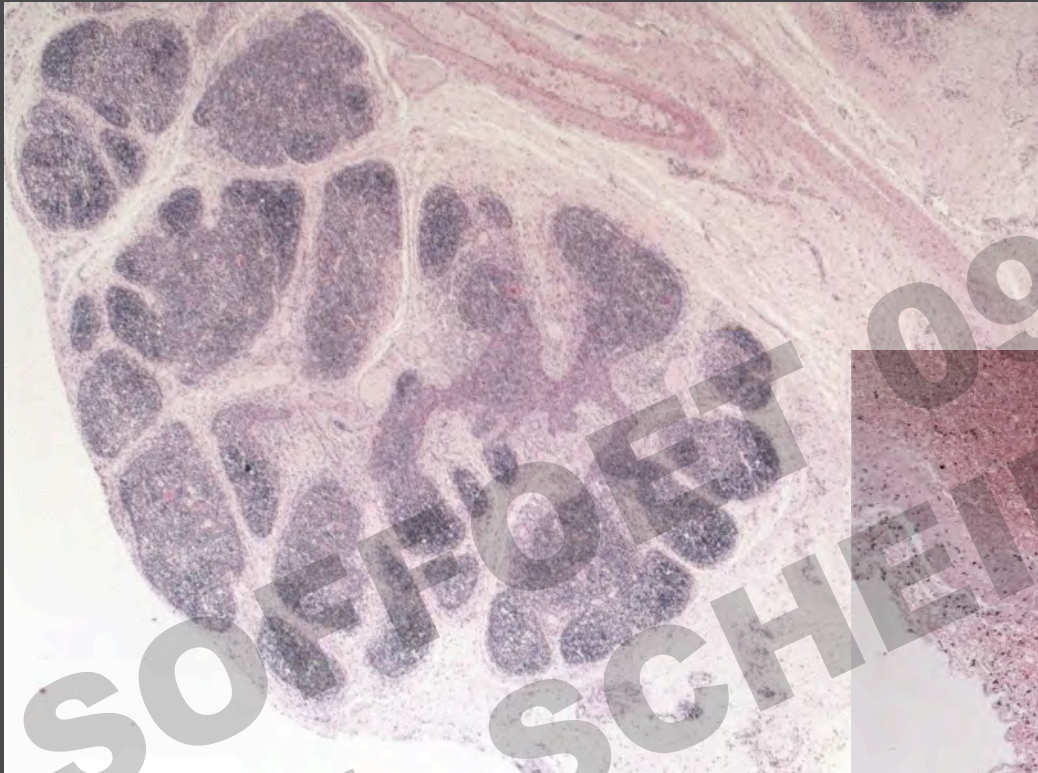
IUGR babies



IUGR at 26 weeks gestation



Atrophic thymus and IDH



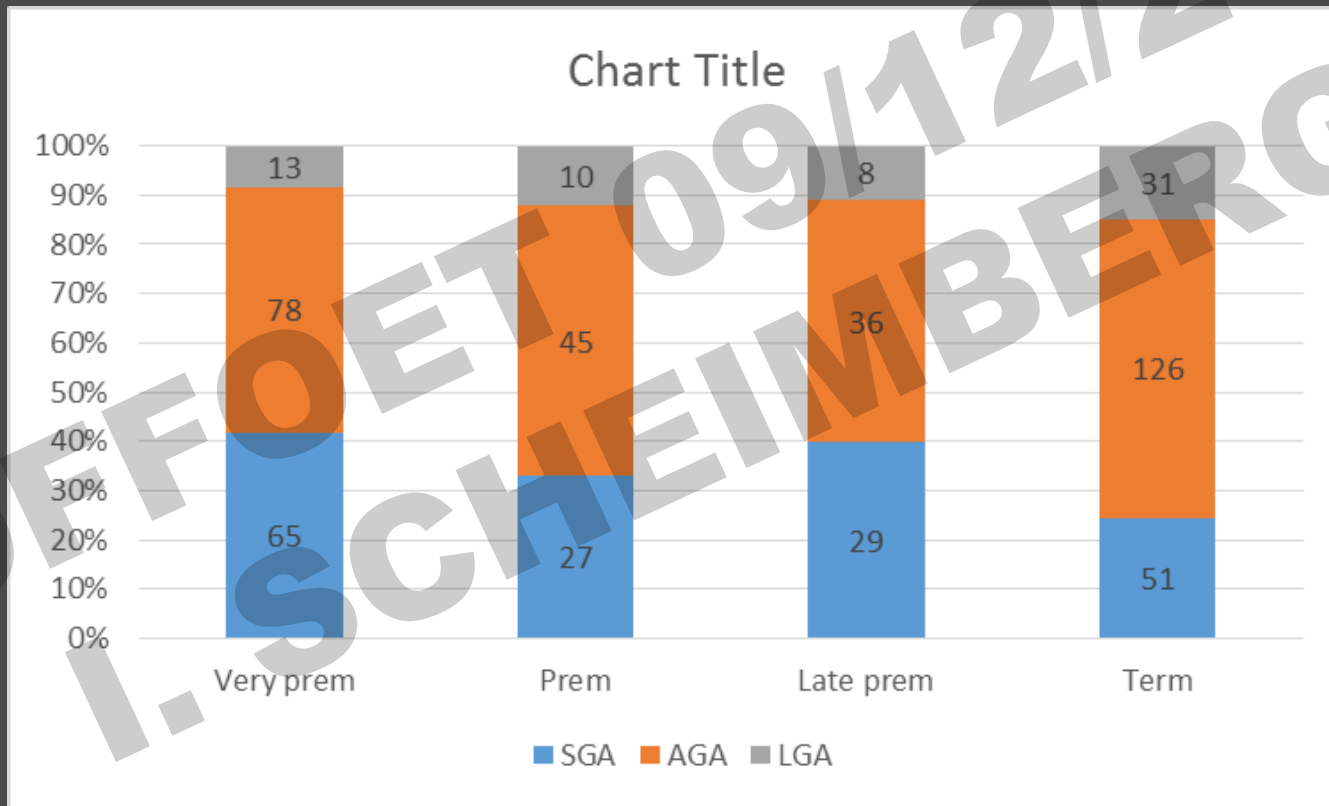
IUGR and IUD

- Fetuses with IUGR are at increased risk of IUD (preterm and term)
- The severity of the growth restriction is directly related to the risk of IUD
- There are higher mortality rates in cases of absent or reversed end-diastolic flow

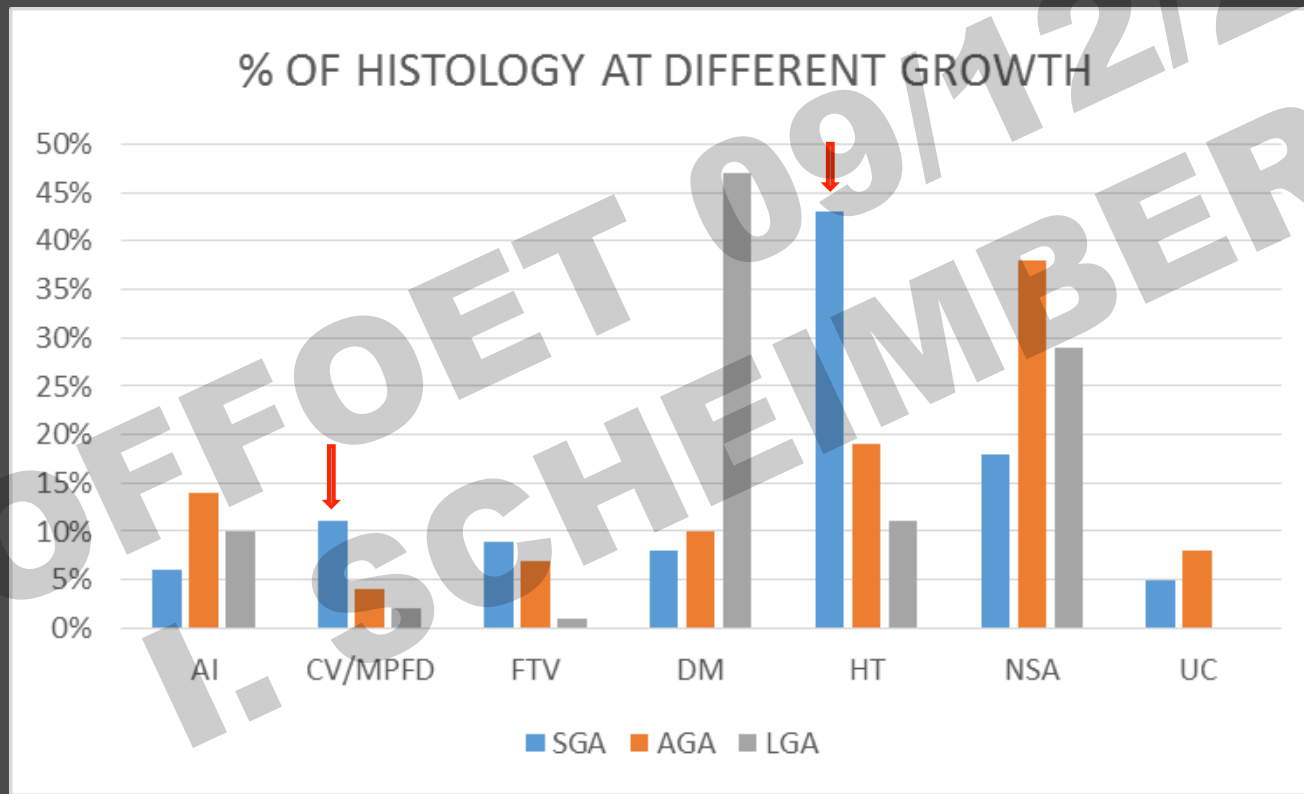
IUGR & perinatal asphyxia

- IUGR babies are already compromised due to placental insufficiency and chronic intrauterine hypoxia
- Transient diminished placental blood flow during labour is more likely to be poorly tolerated in IUGR
- Term SGA infants are more likely to need LW resuscitation & have a higher rate of HIE

SGA babies may be preterm or term

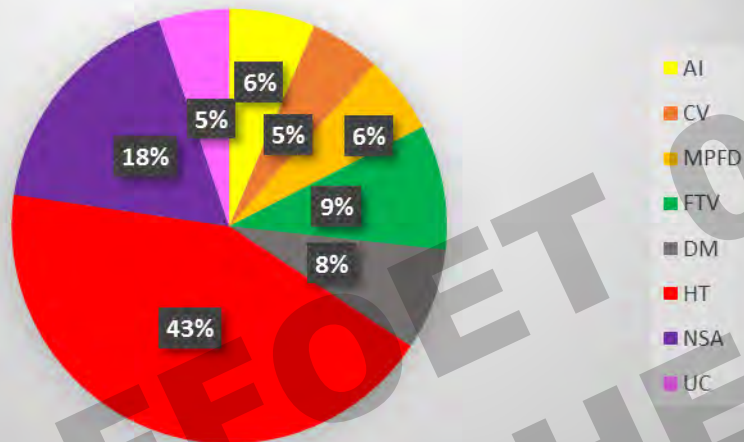


Small babies show specific placental histology

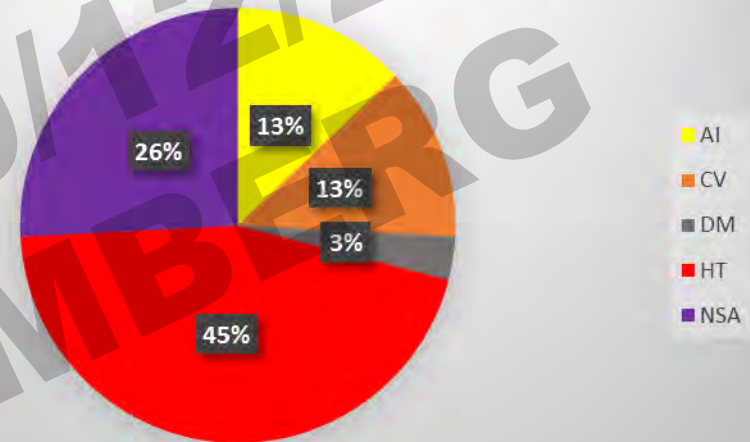


Placental histology in SGA babies

Dead babies SGA Placental histology



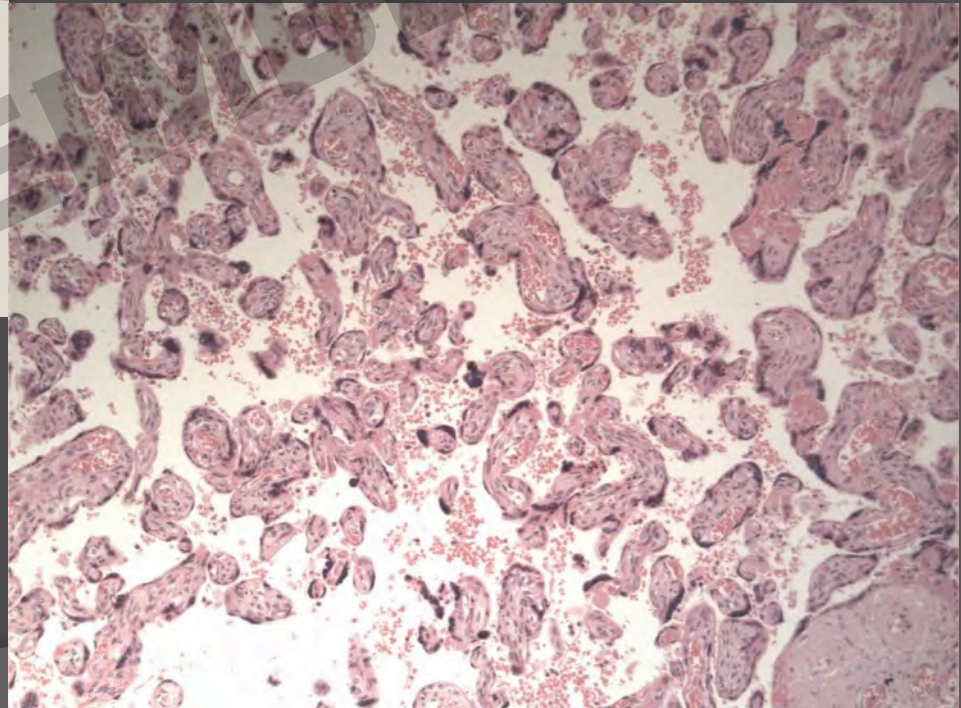
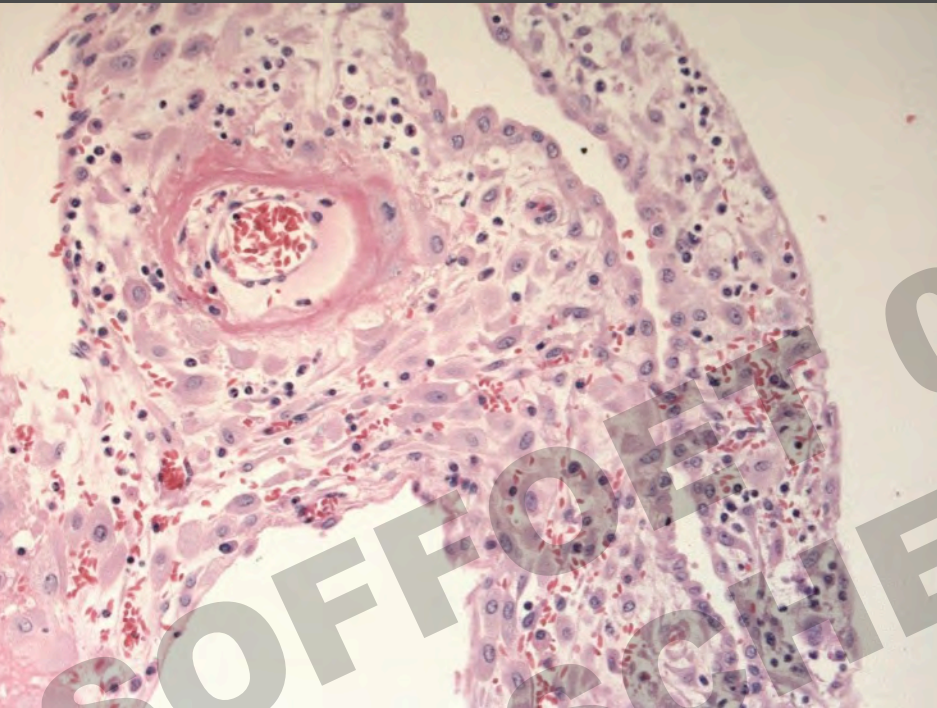
SGA live placental histology



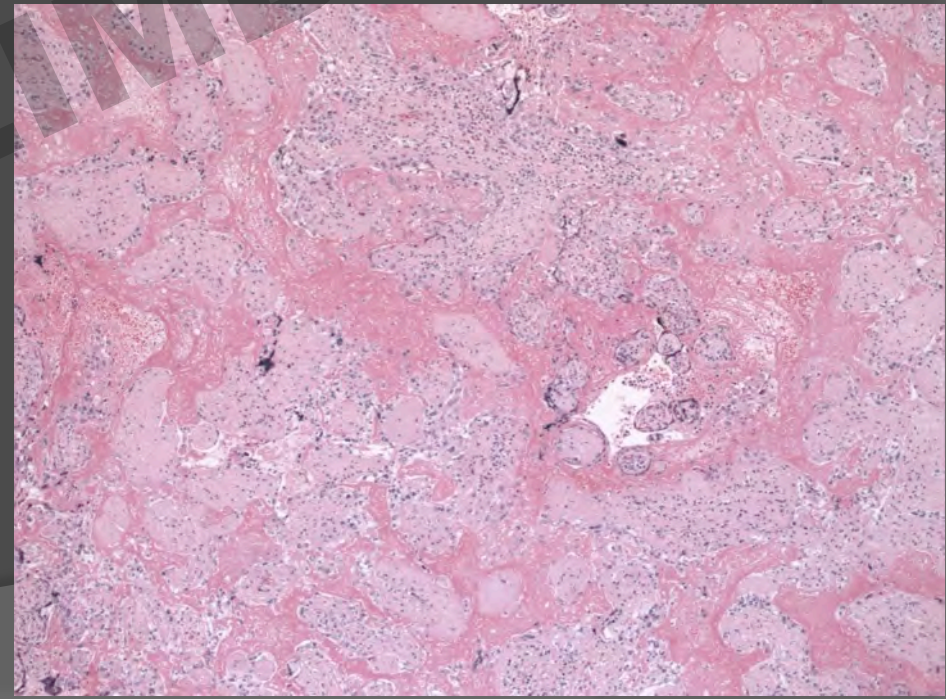
Live babies

- No MPFD or FTV in live babies & no cord accidents
- Same % of maternal vascular malperfusion
- More acute inflammation, chronic villitis and NSA

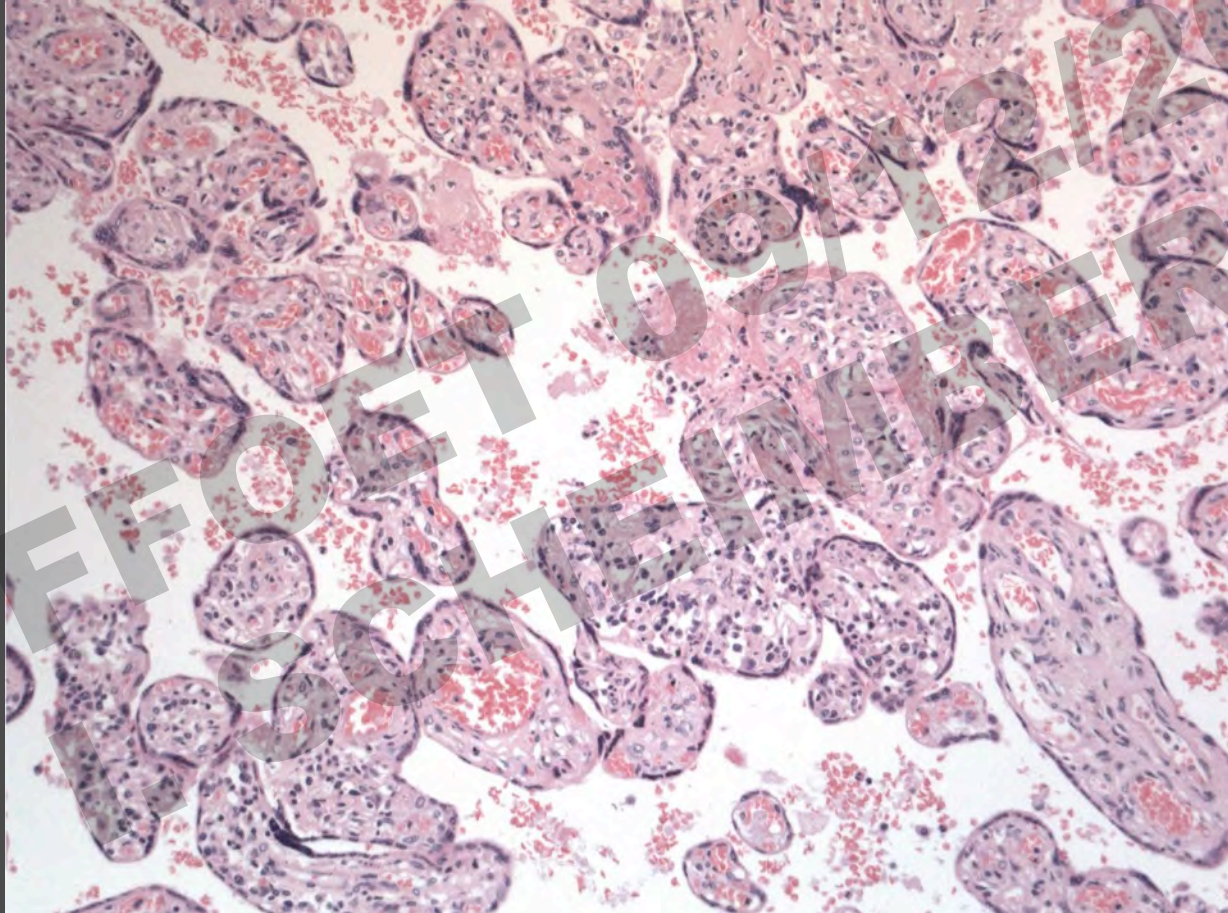
Maternal vascular malperfusion



MPFD



VUE



Birth trauma

- Death due to mechanical birth injury:
0.2-0.7/1000 births
- Term and preterm (Larroche: 11% of preterm babies who died).
- Related to asphyxia:
 - 1) asphyxiated fetus → ↓ muscle tone & engorgement cerebral vessels
 - 2) common causation factors
 - 3) trauma may cause brain swelling and bleeding → asphyxia.

Risk factors for fetal injury

- Instrumental delivery
- Macrosomia
- Prematurity
- Abnormal fetal presentation
- Prolonged labour
- Precipitous delivery

MANY CONDITIONS THAT
PREDISPOSE TO
INTRAPARTUM ASPHYXIA
ALSO INCREASE THE RISK
OF BIRTH TRAUMA

Instrumental delivery

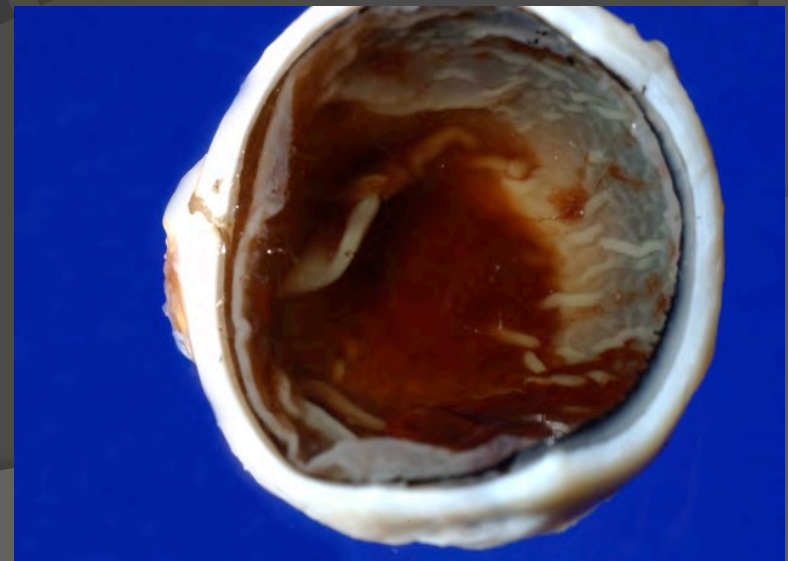
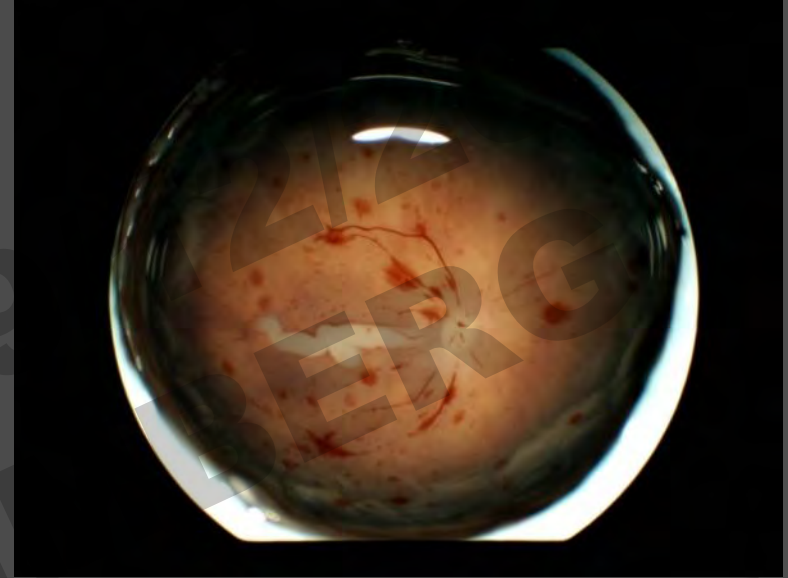
- ⦿ Usually done when fetal hypoxia
- ⦿ Greatest risk of birth trauma
- ⦿ Death more likely if brain/spinal cord injury.
- ⦿ Relative contribution of trauma and asphyxia may be difficult to assess
 - low cord O₂ & pH
 - major bleeding

Predisposing factors

- Asphyxia
- Instrumental delivery
- Malposition
- Maternal diabetes and obesity
- Obstructed or prolonged labour
- Feto-pelvic disproportion
- Macrosomia or fetal abnormality.
- Epidural anaesthesia

Retinal haemorrhages

- Common finding in neonates
- 78% of babies born by ventouse had RH
- The majority resolved in 16 days but in 2 cases there were still present at 31 and 58 days of age.
- Mechanism
 - prolonged build up and sudden decrease in intra cranial pressure & rapid compression decompression of the fetal skull



Thank you

