Cerebral palsy: neurology and neuroradiology

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Facts

- Mum/dad/family statement
- Records (mum, child, red book) CTG, scalp pH, foetal movements, cord gases, Apgar scores, neonatal condition
- Cooled?
- Imaging
- SUI report
- See child

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- Normal pregnancy
- SOL
- 39 +6
- Intermittent monitoring
- Concerns about bradycardia
- Brady 80/min (1725)
- Category 1 CS (birth at 1745). No comment about placenta or cord
- Cord gases taken
- Birth: Apnoeic and bradycardic (Apgars 0, 3). Cardiac massage and intubation
- Heart rate greater than 100 at 5 mins of age
- Neonatal encephalopathy with seizures
- Cooled
- MRI scan showed thalamic high signal
- Dyskinetic CP

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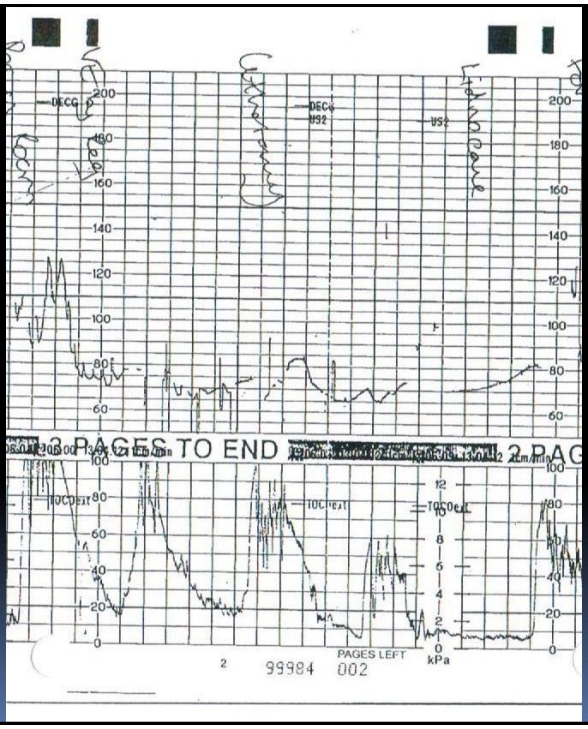

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Apgar score

	0	1	2
Heart rate (P)	Absent	<100	>100
Respiratory effort (R)	Absent	Weak	Good, crying
Muscle tone (A)	Flaccid	Some flexion	Well flexed
Reflex irritability (G)	No response	Grimace	Cough or sneeze
Colour (A)	Pale or blue	Body pink, hands/feet blue	Completely pink

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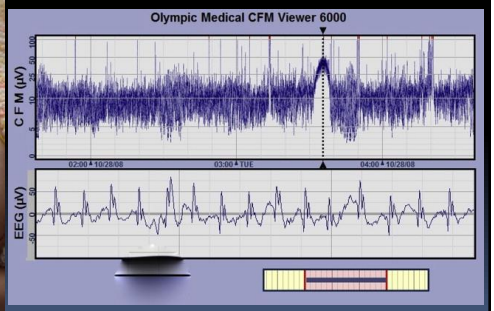
Neonatal encephalopathy

	MILD (1)	MODERATE (2)	SEVERE (3)
Conscious level	Hyperalert	Lethargic	Stuporose
Tone	Normal	Mild hypotonia	Flaccid
Suck	Weak	Weak or absent	Absent
Seizures	None	Common	Uncommon



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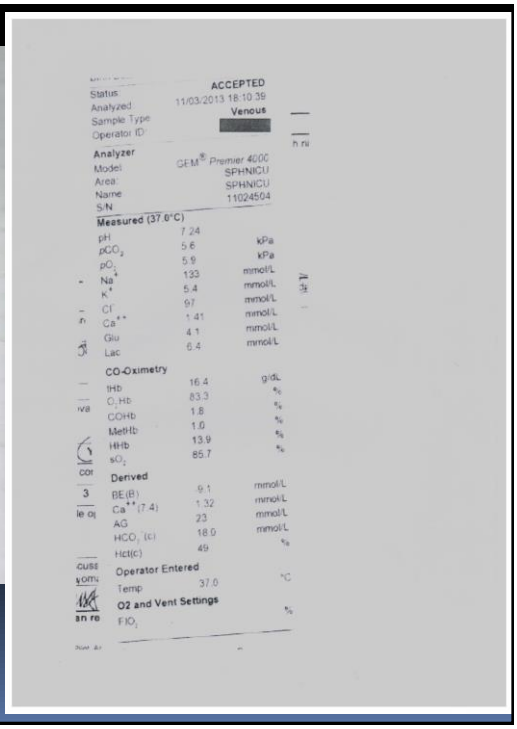
Normal Umbilical Cord Blood Gases

	Venous Blood Normal Range (Mean ± 2SD)	Arterial Blood Normal Range (Mean ± 2SD)
pH	7.25 – 7.45	7.18 – 7.38
PCO_2 (mmHg) *(kPa)***	26.8 – 49.2 *3.57 – 6.56*	32.2 – 65.8 *4.29 – 8.77*
PO_2 (mmHg) *(kPa)***	17.2 – 40.8 *2.29 – 5.44*	5.6 – 30.8 *0.75 – 4.11*
HCO_3^- (mmol/L)	15.8 – 24.2	17 – 27
BD* (mmol/L)	0 to 8	0 to 8

Table 1
Reprinted with permission from Elsevier, in part from Yeomans ER, Hauth JC, Gilstrap LC III, Strickland DM. Umbilical cord pH, PCO_2, and bicarbonate following uncomplicated term vaginal deliveries Am J Obstet Gynecol 1985;151:798-800.
Data are mean values ± 2 standard deviations (SD).
* Base deficit, estimated from data.
** 1 kPa = 7.50 mmHg; 1 mmHg = 0.133 kPa
Note: "Normal" is arbitrarily defined as the mean ± two times the standard deviation (approximately 95.4% of a normally distributed population).



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Normal Umbilical Cord Blood Gases		
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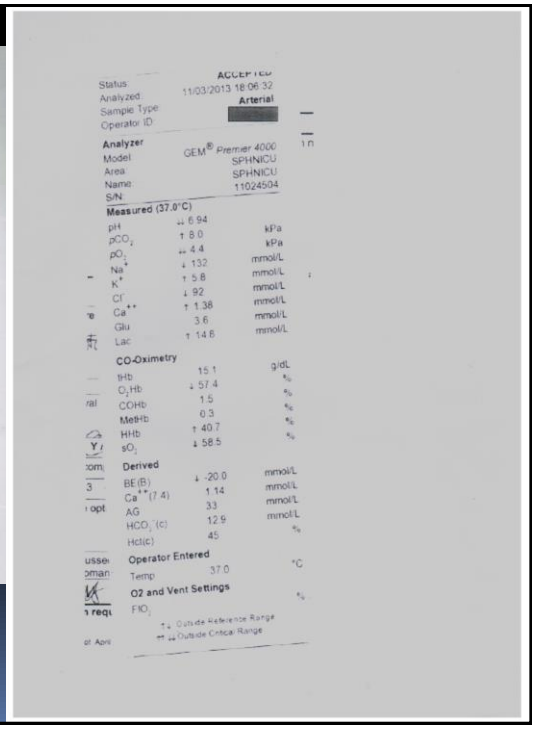

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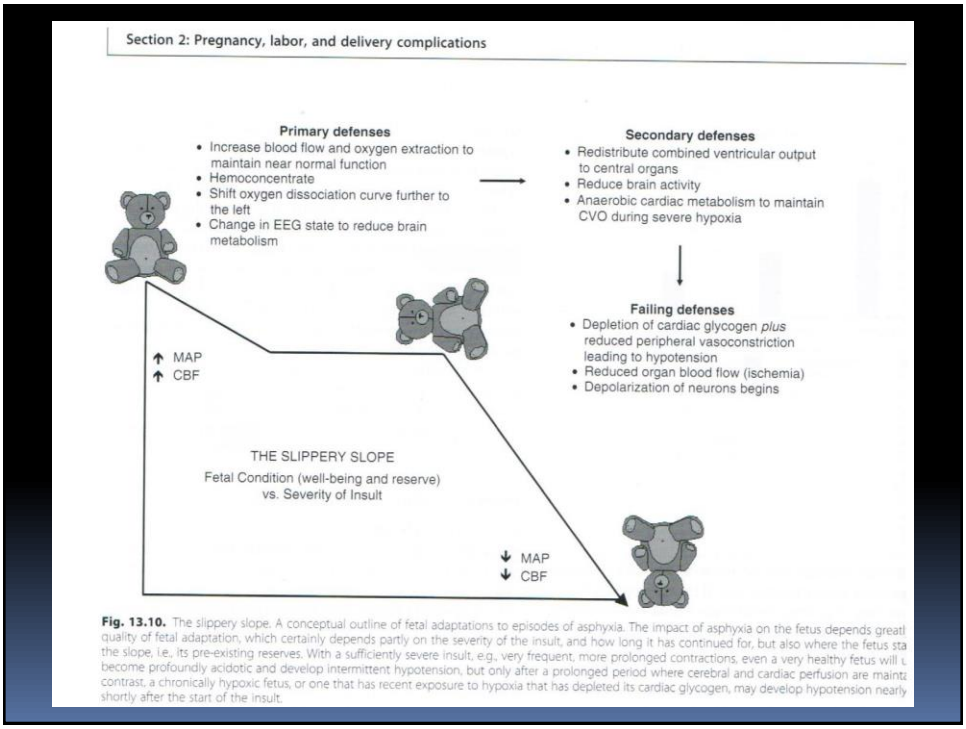

Fig. 13.10. The slippery slope. A conceptual outline of fetal adaptations to episodes of asphyxia. The impact of asphyxia on the fetus depends greatl quality of fetal adaptation, which certainly depends partly on the severity of the insult, and how long it has continued for, but also where the fetus sta the slope, i.e., its pre-existing reserves. With a sufficiently severe insult, e.g., very frequent, more prolonged contractions, even a very healthy fetus will u become profoundly acidotic and develop intermittent hypotension, but only after a prolonged period where cerebral and cardiac perfusion are maintz contrast, a chronically hypoxic fetus, or one that has recent exposure to hypoxia that has depleted its cardiac glycogen, may develop hypotension nearly shortly after the start of the insult.

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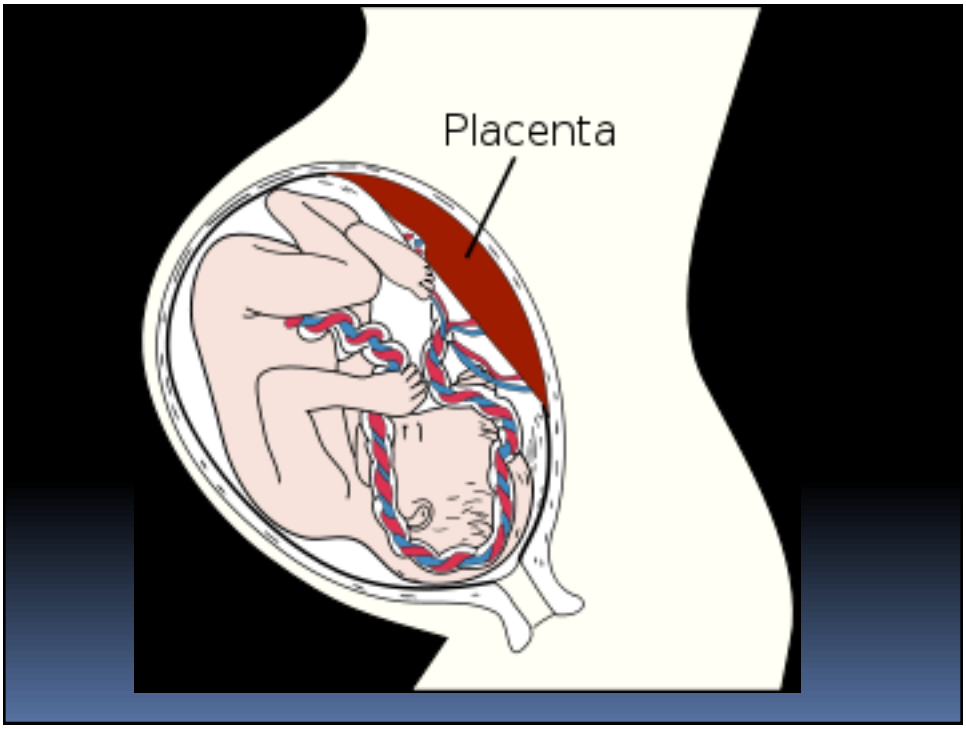
Placenta

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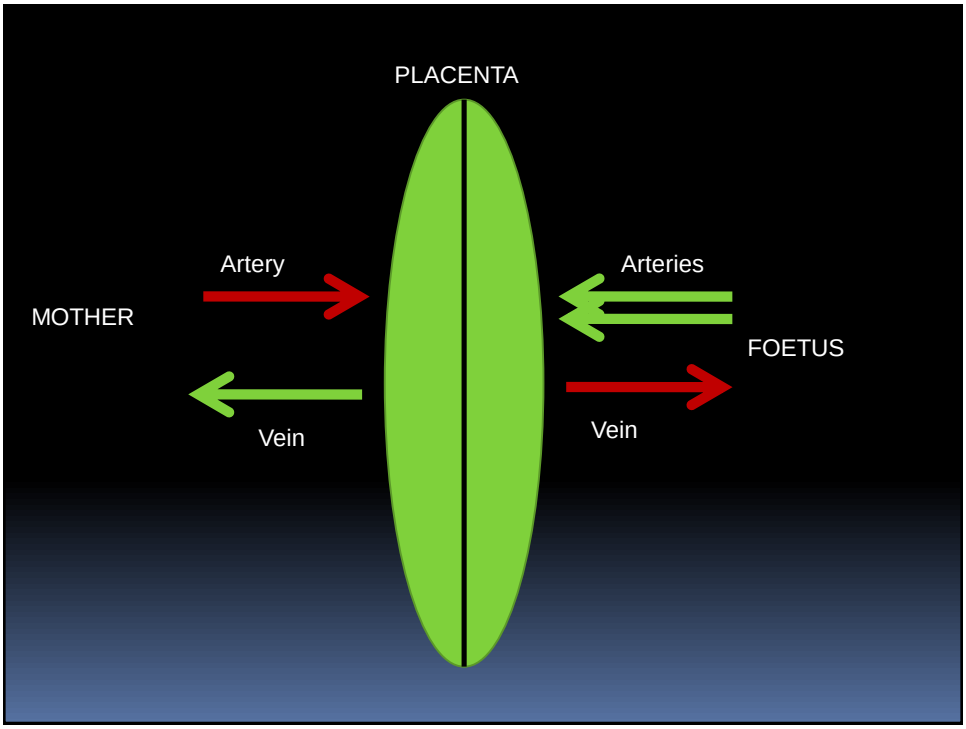
PLACENTA
Artery
MOTHER
Vein
Arteries
FOETUS
Vein

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- Is there neurological impairment?
- Is there injury?
- What type of injury is it?
- How long was the insult?
- When did it start?
- When did it finish?
- When did it become damaging?
- When would delivery have to have been to avoid all injury?
- What was the mechanism?

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- Working back and working forward approach

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TOTAL PERIOD OF HYPOXIC ISCHAEMIA	BRAIN DAMAGING PERIOD OF HYPOXIC ISCHAEMIA	CLINICAL FINDINGS	
0-10 mins	Zero	Normal	
10-15 mins	0-5 mins	Bilateral dystonic (extrapyramidal) cerebral palsy ambulant (likely to be) self feeding and able to use speech (but dysarthric). Any learning difficulties are usually mild. Speech often dysarthric.	GMFCS Level II
15-20 mins	5-10 mins	Bilateral dystonic (extrapyramidal) cerebral palsy usually non-ambulant) and hand function impaired. bulbar palsy leading to speech and feeding difficulties and a requirement for communication aids. Intellect usually impaired to some degree	GMFCS Level III
20-25 mins	10-15 mins	Bilateral spastic/dystonic CP, non ambulant and often quite a paucity of movement with very poor motor function. Intellectual impairment significant. Very dependent children often requiring gastrostomy feeds and usually wholly dependent for hygiene etc. Usually no speech.	GMFCS Levels IV or V
>25 mins	>15 mins	Death or unable to move or swallow (usually gastrostomy fed). Severe or profound cognitive impairment. Microcephaly	GMFCS Level V

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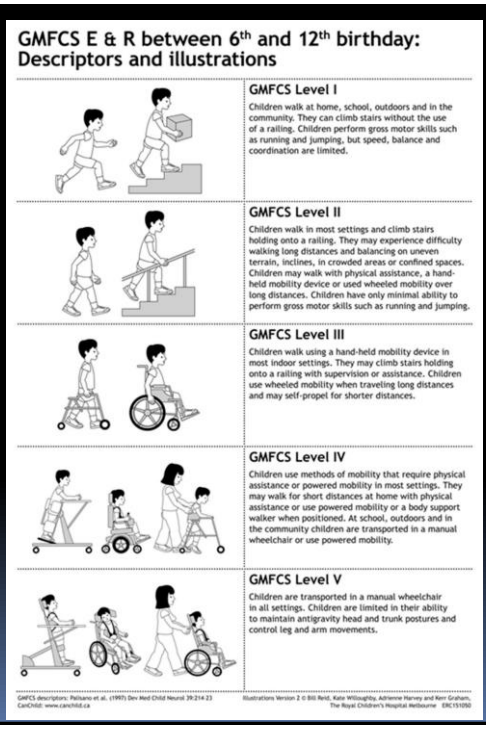

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	Acute profound	Chronic partial
Insult duration	0-25 mins	At least an hour-days
Reserve	10 mins	Very variable
Parts of brain damaged	Basal ganglia first	"Watershed"
Type of CP	Dyskinetic	Spastic/Learning difficulties
Mechanism	Cord compression	Placental failure (and others)

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Independent risk factors for cerebral palsy (adapted from 2017 NICE CP guideline)

Antenatal Factors

- Preterm birth
- Chorioamnionitis
- Maternal respiratory tract infection or genito-urinary infection treated in hospital

Perinatal Factors

- Low birth weight (at increased risk if birth weight <1.5kg)
- Chorioamnionitis
- Neonatal encephalopathy (as a result of, eg, sepsis, hypoxic-ischaemic injury)
- Neonatal sepsis
- Maternal respiratory tract infection or genito-urinary infection treated in hospital

Postnatal Factors

- Meningitis or other infections
- Head injury

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Cerebral palsy in under 25s: assessment and management (NICE Guideline NG62) Jan 2017

Diagnosing cerebral palsy:

- Identify independent risk factors for cerebral palsy. Refer any child with one or more risk factors for clinical and developmental follow-up, by a multidisciplinary team, until 2 years corrected gestational age.
- Recognise abnormal signs suggestive of cerebral palsy and make an urgent referral to a child development service for a multidisciplinary assessment
- Arrange an MRI scan if the aetiology of cerebral palsy is unclear from the history, developmental assessment, clinical examination or cranial ultrasound results.

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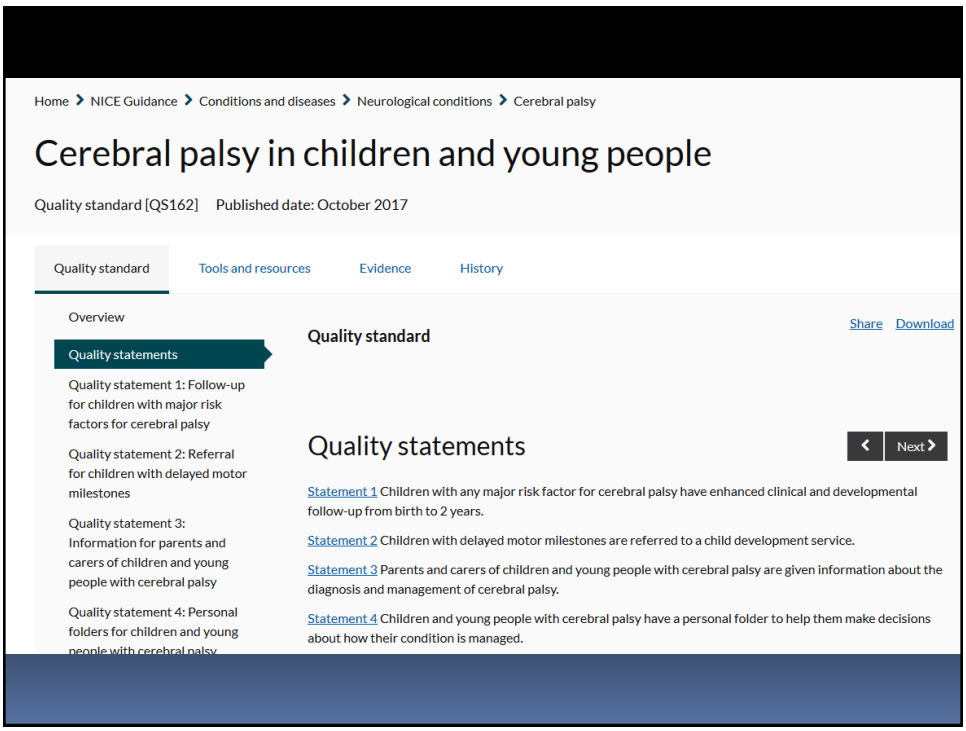

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Types of disabilities

- Motor (Cerebral palsy)
- Visual impairment/deafness
- Learning difficulties (mental retardation, developmental delay)
- Autism and other behavioural disorders
- Epilepsy
- Specific cognitive problems (especially memory)

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What is cerebral palsy?

Cerebral palsy describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of CP are often accompanied by disturbances of sensation, perception, cognition, communication, behaviour, by epilepsy and by secondary musculoskeletal problems. (Modified after Bax et al. 2005)

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Other features associated with cerebral palsy

- Mental retardation
- Epilepsy
- Visual/hearing impairment
- Feeding and swallowing difficulties
- Behavioural difficulties
- Contractures/hip dislocation/scoliosis

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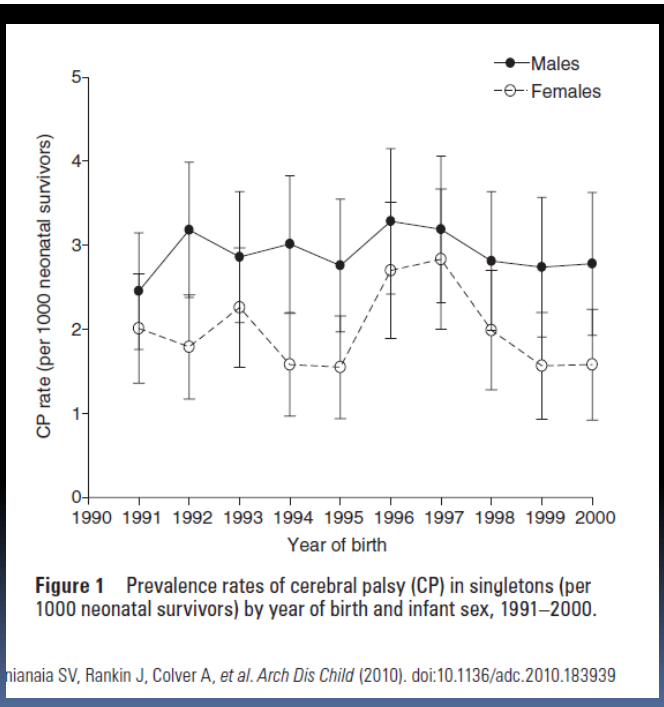

Figure 1 Prevalence rates of cerebral palsy (CP) in singletons (per 1000 neonatal survivors) by year of birth and infant sex, 1991–2000.

nianaia SV, Rankin J, Colver A, *et al. Arch Dis Child* (2010). doi:10.1136/adc.2010.183939

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Estimates of proportion of cerebral palsy in term and near-term infants attributed to major causes in population based studies

Neuroimaging based	
Stroke	22%
Congenital malformation	15%
White matter disorder	12%
Hypoxia/ischaemia	5%
Clinical studies	
Intrauterine exposure to inflammation	11-12%
Intrapartum hypoxia/ischaemia	6%
Complications of multiple birth	5%

(from Nelson et al 2008)

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Types of cerebral palsy

- Spastic (diplegia, hemiplegia, quadriplegia)
- Dyskinetic
- Others (ataxic, hypotonic)

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- Different types of CP are commoner at different gestations (preterm-diplegia, term dyskinetic, hemiparesis, quadriplegia)
- Different types of CP have different causes:

  Hemiplegia: strokes and malformations

  Diplegia: intrauterine infection, preterm membrane rupture, multiple gestation

  Dyskinetic CP: acute profound hypoxia/ischaemia, kernicterus, genetic

  Spastic quadriparesis: chronic partial hypoxia/ischaemia, genetic, infections

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Causation

- Is the diagnosis of cerebral palsy correct and what is the type (physical examination)?
- If so what has caused it (neuroradiology report on MRI scan)
- **Was there evidence of intrapartum hypoxia/ischaemia of potentially damaging severity (CTG, history, scalp pH)?**
- What was the condition at birth? (Apgar scores, cord gases)
- Was there neonatal encephalopathy? (neonatal notes)
- Is the outcome one that intrapartum hypoxia/ischaemia could explain?

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Potential causes of brain injury at or around the time of birth (acute or chronic)

Mother	Placenta/Uterus	Cord	Foetus/baby
Shock	Excessive uterine action	Prolapse	Infection
Trauma	Abruption	Compression	Bleeding
Maternal hypotension	Poor function	Entanglement	Twin complications
	Chorioamnionitis		
	Emboli		
	Uterine rupture		

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Electronic monitoring (CTG)

- Assume prevalence of CP is 3/1000 live births
- Assume 10% of CP is caused by intrapartum hypoxia/ischaemia
- 0.3/1000 is prevalence of CP due to intrapartum hypoxia/ischaemia (an uncommon cause of a rare outcome)
- Would need study of very large numbers of labours to show reduction in CP
- "Birth can be a hazardous journey: electronic fetal monitoring does not help"

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Neonatal encephalopathy ("HIE")

- Thought to be an inevitable intermediary between asphyxial birth injury and CP.
- Causes of neonatal encephalopathy (intrapartum hypoxia/ischaemia, metabolic, infections)

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Table 1. Hypoxic ischaemic encephalopathy score.

Sign	Score 0	1	2	3
Tone	Normal	Hyper	Hypo	Flaccid
LOC	Normal	Hyper alert, stare	Lethargic	Comatose
Fits	None	Infreq $< 3\ d^{-1}$	Frequent>2/day	
Posture	Normal	Fisting, cycling	Strong, distal flexion	Decerebrate
Moro	Normal	Partial	Absent	
Grasp	Normal	Poor	Absent	
Suck	Normal	Poor	Absent ± bites	
Resp.	Normal	Hypervent	Brief apnoea	IPPV (apnoea)
Font	Normal	Full, not tense	Tense	
				Total score per day–

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Cooling for newborns with hypoxic ischaemic encephalopathy (Review)

Jacobs SE, Berg M, Hunt R, Tarnow-Mordi WO, Inder TE, Davis PG

THE COCHRANE COLLABORATION®

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PLAIN LANGUAGE SUMMARY

Cooling for newborns with hypoxic ischaemic encephalopathy

There is evidence that induced hypothermia (cooling) of newborn babies who may have suffered from a lack of oxygen at birth reduces death or disability, without increasing disability in survivors. This means that parents should expect that cooling will decrease their baby's chance of dying, and that if their baby survives, cooling will decrease his/her chance of major disability. A lack of oxygen before and during birth can destroy cells in a newborn baby's brain. The damage caused by the lack of oxygen continues for some time afterwards. One way to try to stop this damage is to induce hypothermia - cooling the baby or just the baby's head for hours to days. This treatment may reduce the amount of damage to brain cells. This review found that there is evidence from trials to show that induced hypothermia helps to improve survival and development at 18 to 24 months for term and late preterm newborn babies at risk of brain damage. More research is needed to understand which infants need cooling and the best way of cooling, including duration of treatment and method of cooling.

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Causes of brain injury around the time of birth other than intra-partum hypoxia/ischaemia

- Kernicterus
- Hypoglycaemia
- Hypernatraemia
- Birth trauma
- Failure of adequate resuscitation (Antoniades and East Sussex NHS Trust)
- Hydrocephalus
- Over ventilation

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Neuroimaging in perinatal injury

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Normal imaging

- Seen in about 15% of CP
- ? Repeat scan
- ? Neuroradiology report
- Is it damage that cant be seen with present technology
- Metabolic/genetic

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Modalities

- What can we use?
  - Ultrasound
    - antenatal
    - postnatal
  - Computed tomography (CT)
  - Magnetic resonance imaging (MRI)
    - advanced imaging
    - fetal MRI

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Ultrasound

- Easily available at cotside
- Safe
- Transducer on fontanelle
- Good for intraventricular haemorrhage; ventricular size, established focal parenchymal lesions
- Poor for more subtle parenchymal pathology
- Operator dependent

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Ultrasound

- Relatively insensitive
- Parenchymal abnormalities take variable time to develop
  - Acute
  - Longer term
- Depends on severity of insult
- Changes usually not specific in terms of cause

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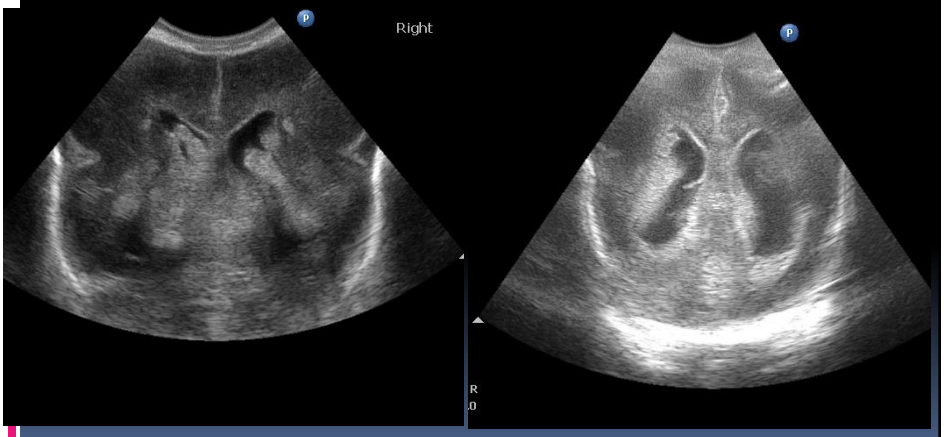
Intraventricular haemorrhage



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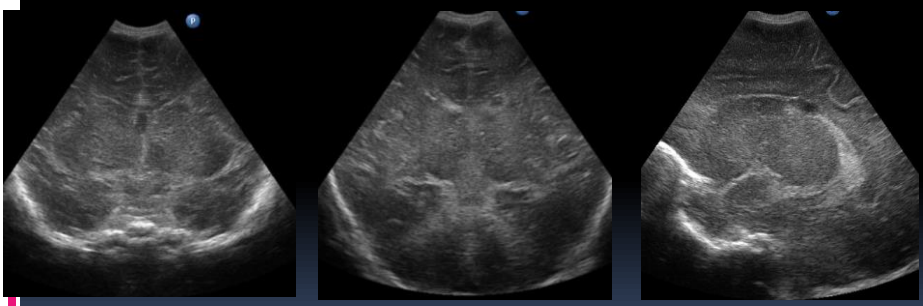
More subtle abnormality



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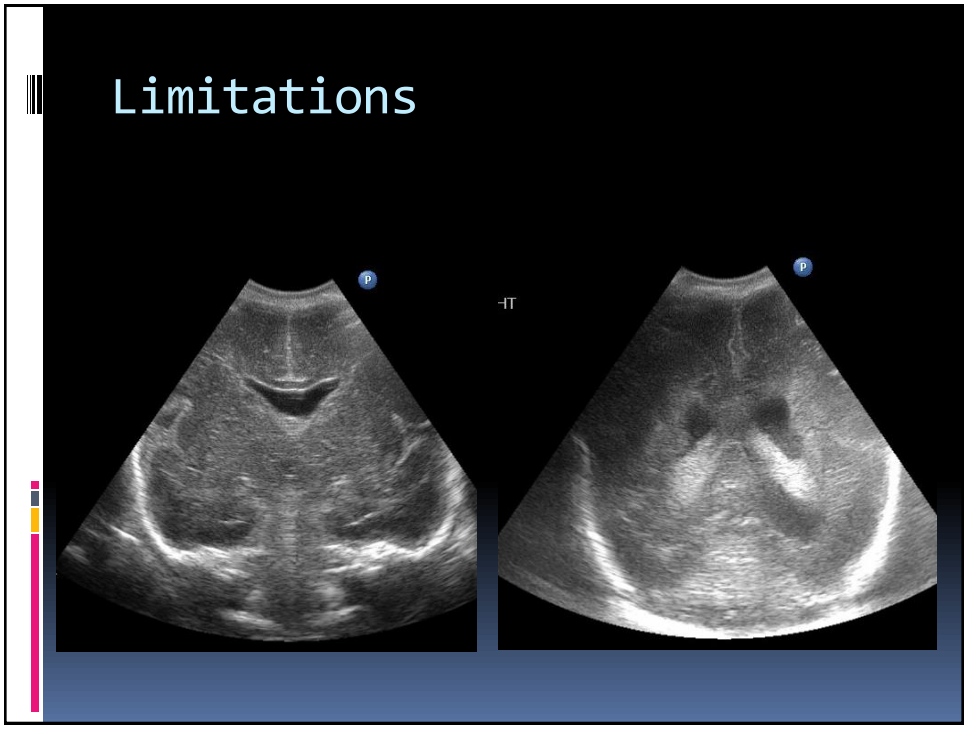

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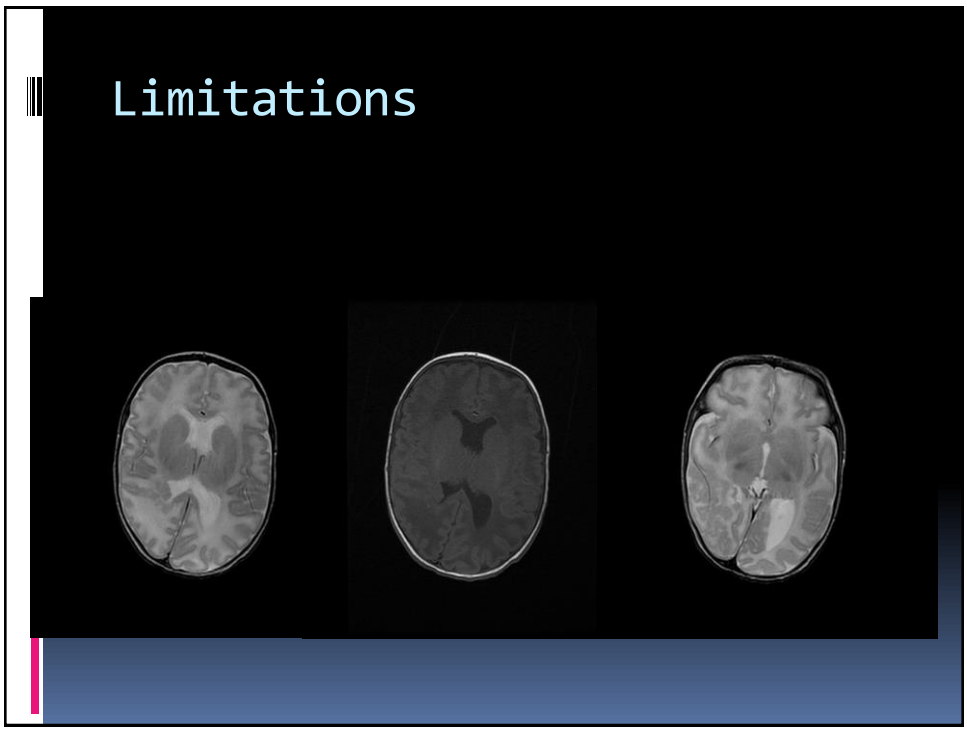

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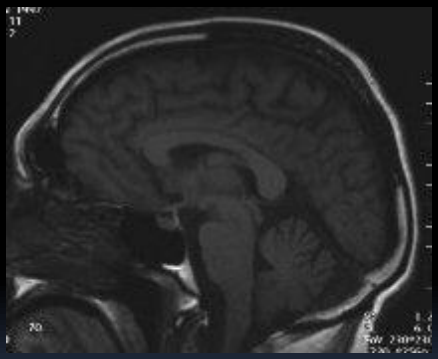
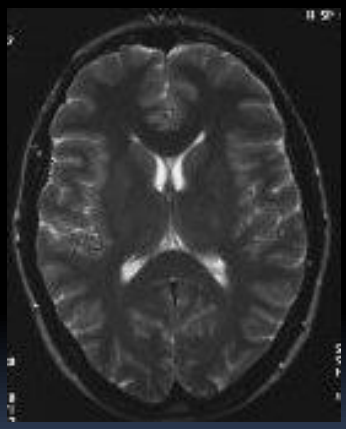
Normal brain





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Brain development

- Prenatal
- Postnatal

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Brain development: prenatal

- Grey matter (nerve cells)
  - cell migration
- White matter (nerve fibres)
  - myelination
- Blood supply
- Synaptic maturation

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Fetal MRI

- Brain development
  - lots going on…
  - which means that…..
  - a barn door abnormality on a postnatal MRI
  - may not be evident on fetal MR

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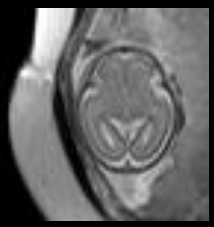
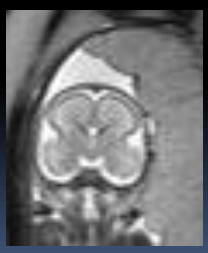
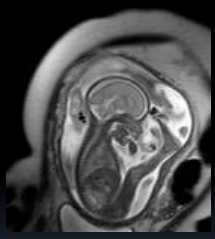
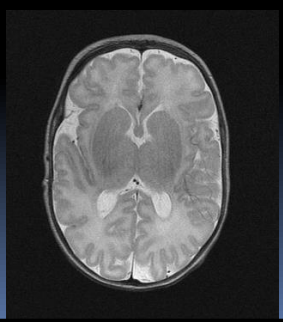
Fetal MRI 24 weeks

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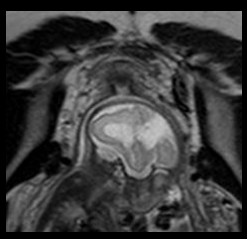
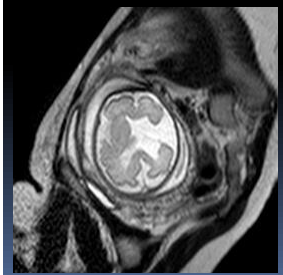
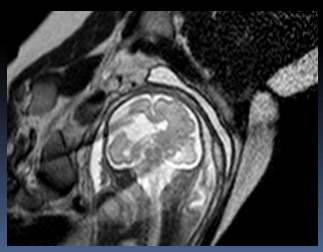
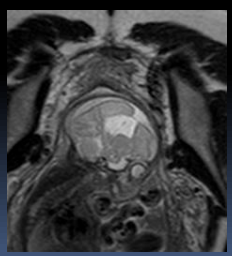
Fetal MRI: schizencephaly at 28 weeks

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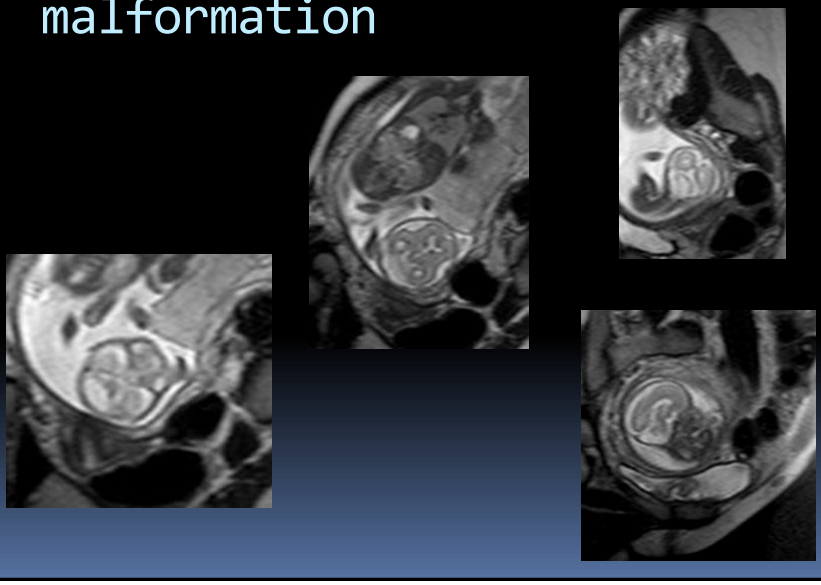
Fetal MRI: Dandy Walker malformation



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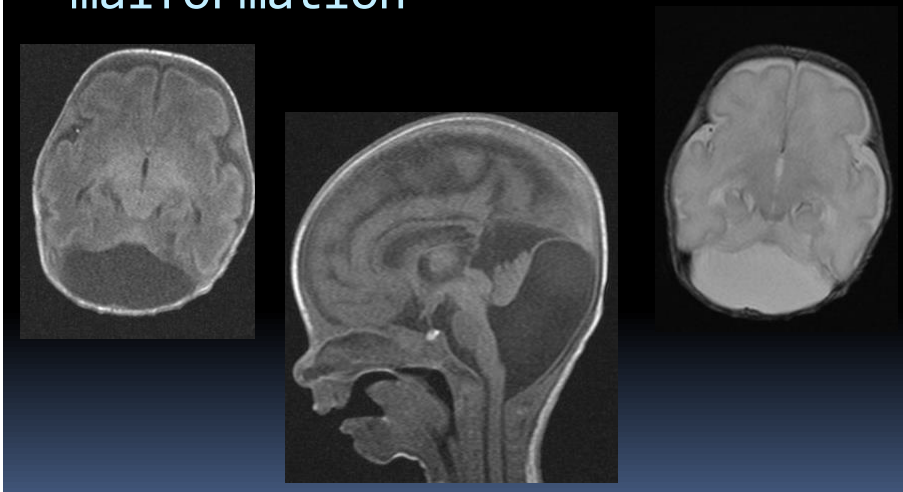
Fetal MRI: Dandy Walker malformation



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Brain development

- Good knowledge of normal appearances
- Know how appearances change with normal maturation
- Only then can abnormal be appreciated and interpreted

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Brain development:postnatal

- Myelination
  - Term
  - Immature (unmyelinated) white matter
    - low signal T1
    - high signal T2

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Myelination

- White matter maturation (myelination)
  - increasing T1 signal (becomes brighter)
  - decreasing T2 signal (becomes darker)

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Myelination

- Assessment
  - T1 weighted images
    - better up to ~ 9 months
  - T2 weighted images
    - better after ~ 9 months
  - radiologically complete by 2 years post term

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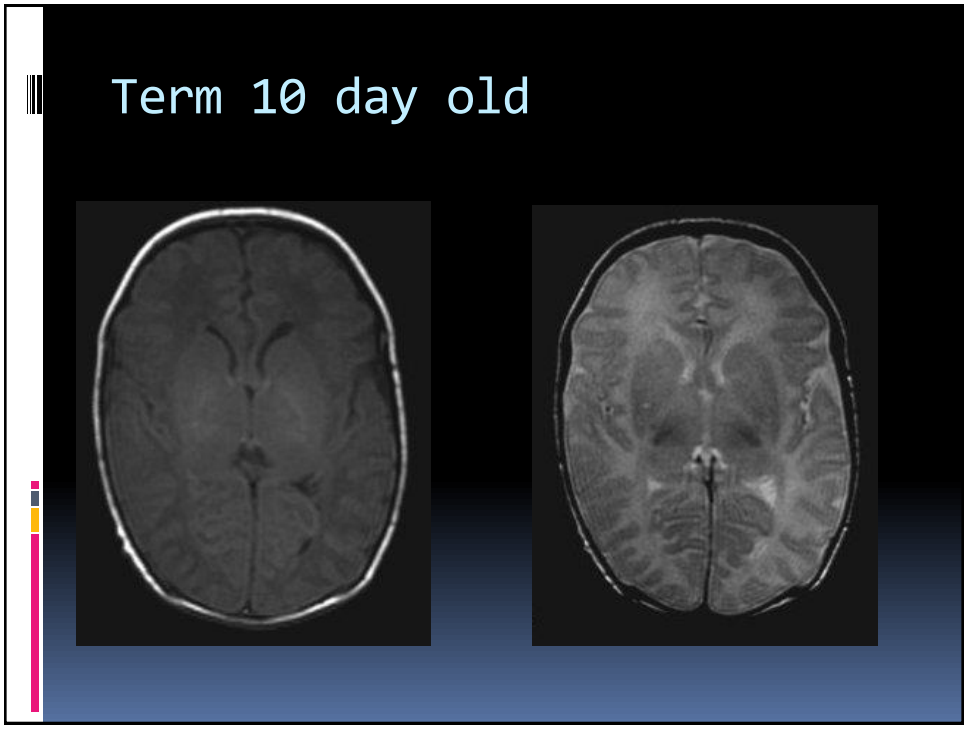

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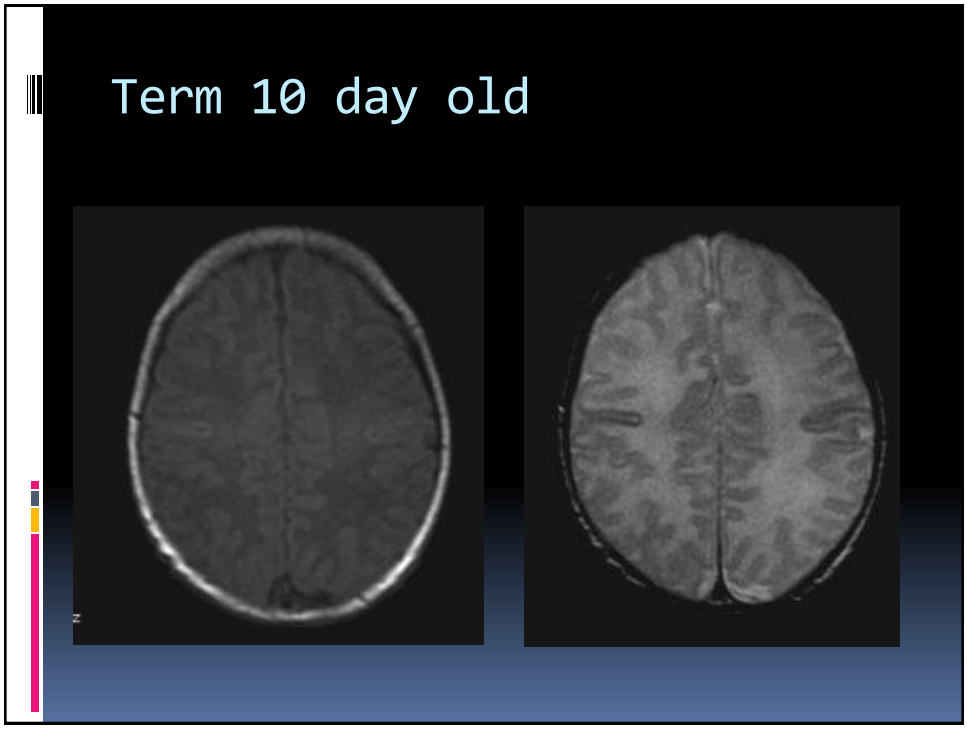
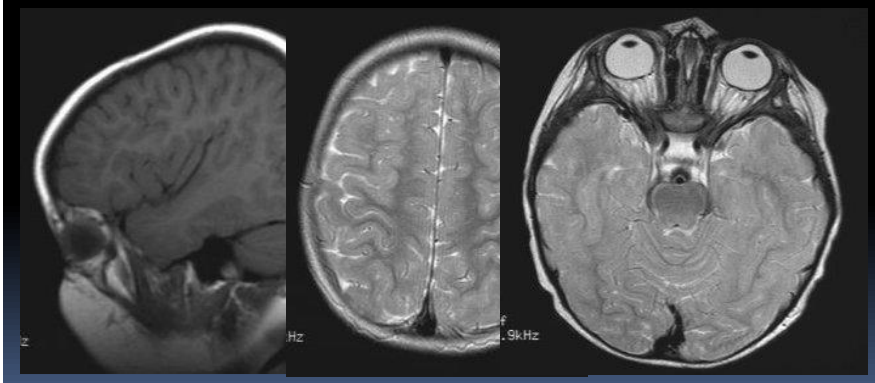


58

18 months



59

Brain injury

- Scans can show evidence of structural brain damage
  - Acute: swelling / oedema
  - Long term: scarring (gliosis); tissue loss / atrophy
- Functional change can occur in absence of structural change

60

Brain injury and myelination

- Unmyelinated white matter = higher signal on T2 scans
- Most acute injury associated with increased tissue water = increased signal on T2 scans
- Gliosis (scarring) = increased signal on T2 scans

61

Assessment of structural brain damage

- Myelination complete (scan appearances) at 2 years of age
- Best time to assess extent of changes is therefore after the age of two years
- May not be able to adequately assess presence and / or extent of damage until then

62

Issues

- What is the likely nature of the causative event?
  - MRI pretty good at this
- When did it occur?
  - MRI (or any other imaging modality) not good at this
  - Requires correlation with obstetric and paediatric evidence

63

Patterns of pre / perinatal injury

- Asphyxia
  - Acute near total
  - Chronic partial
  - Mixed
- Periventricular leukomalacia
- Perinatal infarcts
- Hypoglycaemia
- Trauma

64

Cerebral palsy

- Imaging pattern predicts clinical findings
  - parasagittal (watershed) brain injury
    - spastic quadriplegia
  - isolated basal ganglia damage
    - choreo-athetosis
  - periventricular leukomalacia
    - spastic diplegia
  - focal brain infarcts
    - hemiplegia

65

Asphyxiated term infants

- Pattern of abnormality relates to
  - severity of hypoxia / hypoperfusion
    - mild
    - moderate
    - severe
  - duration of insult
    - short
    - long or intermittent
  - Susceptibility
    - gestational age
    - superimposed insult

66

Asphyxiated term infants

- Acute near total asphyxia
  - Placental abruption / prolapsed cord / shoulder dystocia
  - lesions occur in most metabolically active areas
    - posterior putamina (basal ganglia)
    - ventrolateral nuclei of thalami
    - perirolandic white matter and cortex
    - hippocampus

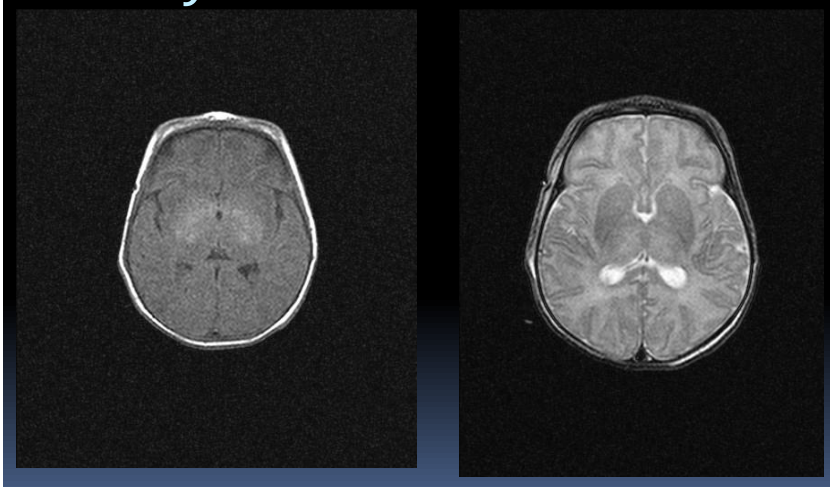
67

Acute near total asphyxia

- Gospel:
- Normal infant can withstand 10 minutes of such an insult
- Unlikely to survive insults 25 min +
- Assessment of duration
  - Putamina only: closer to lower end of time window
  - Full house: closer to longer end of time window

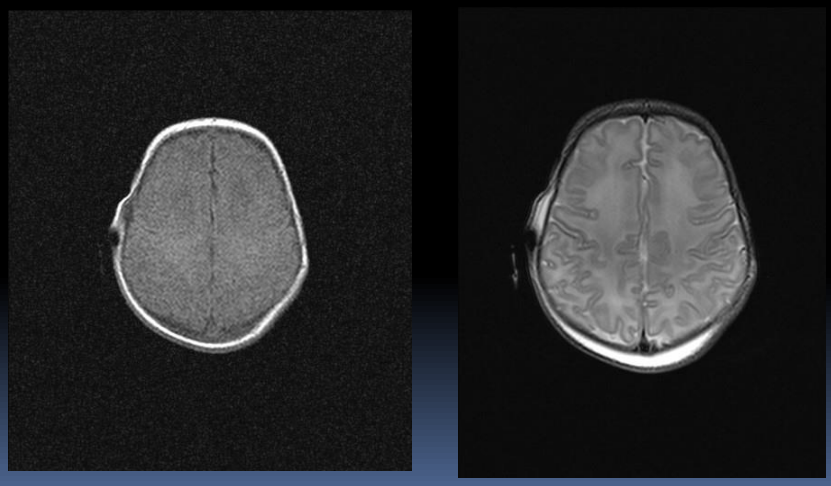
68

Acute near total asphyxia: early scan



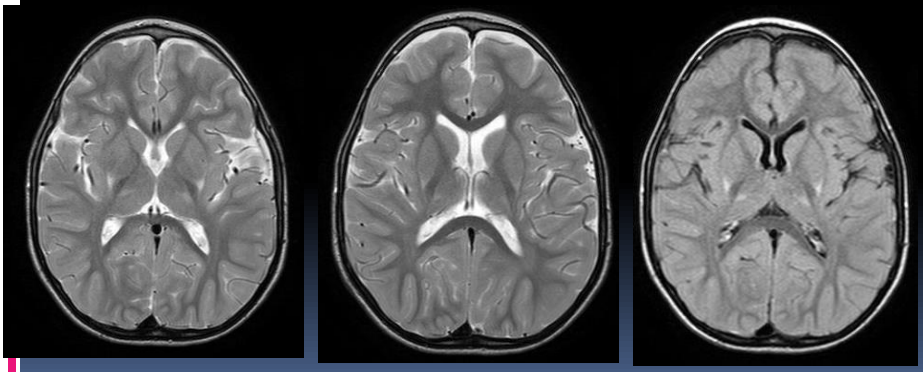
69

Acute near total asphyxia



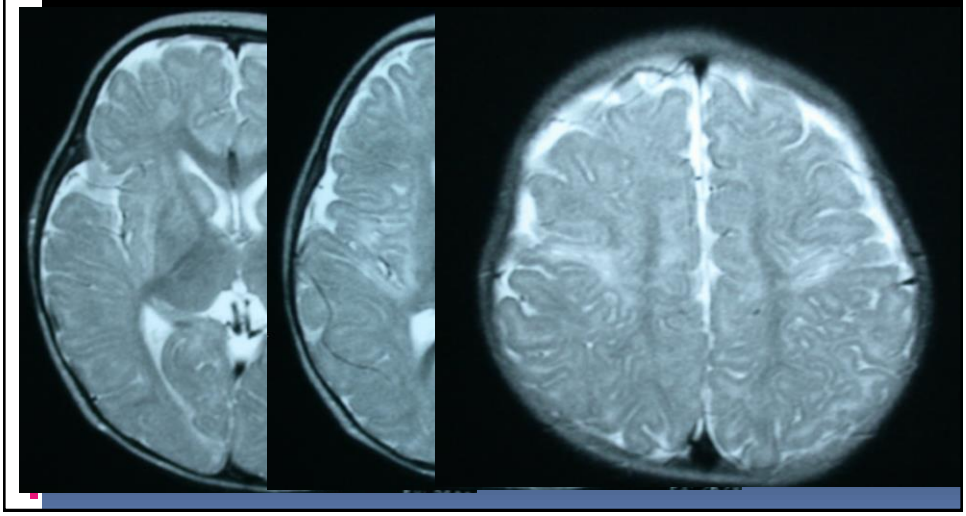
70

Acute near total asphyxia: later imaging



71

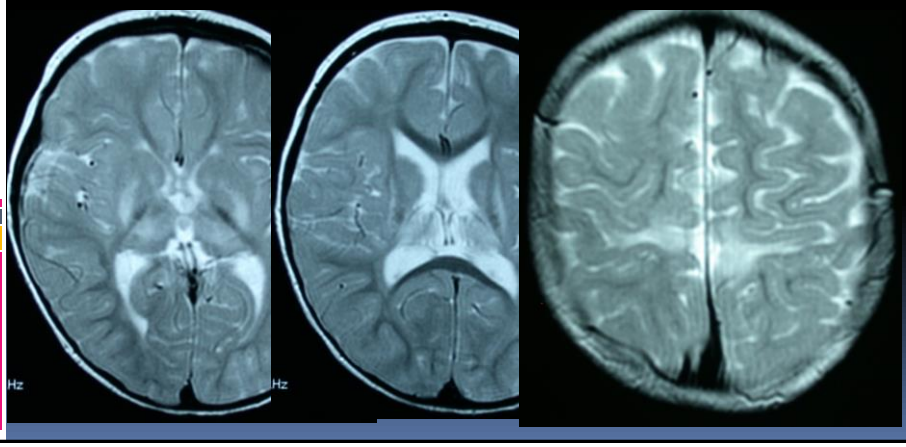
Acute near total asphyxia



72

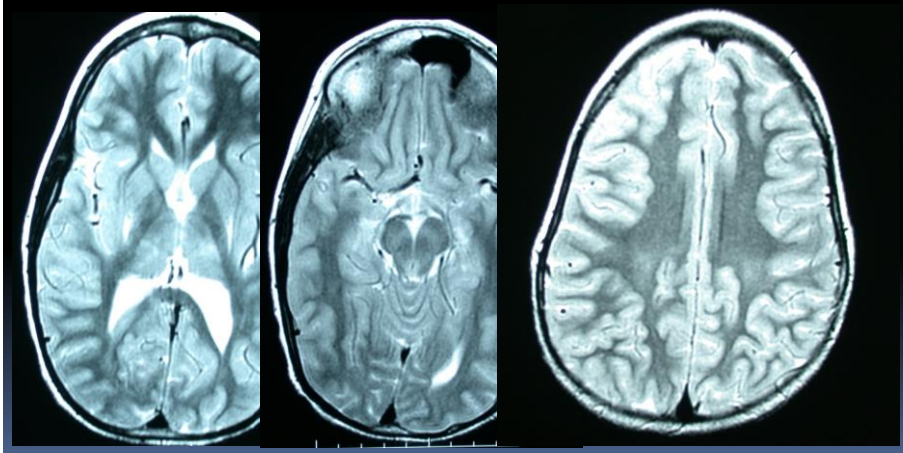
Acute near total asphyxia

- Imaged at 15 months



73

Acute near total asphyxia: imaged at 18 years



74

Chronic partial insults

- Watershed areas
- Cerebral perfusion from two sources
  - Vessels from Circle of Willis (majority)
  - Vessels from pial covering of brain (minority)
- From ~ 36 weeks gestation these meet at around the level of the depths of the sulci

75

Chronic partial hypoxia / hypoperfusion

- Moderate hypoxia over longer period or intermittent hypoxia
  - lesions in
    - cortex: ulegyria = atrophy at base of gyri
    - subcortical white matter: parasagittal, anterior and posterior watershed areas
    - more susceptible to perinatal events if previous intermittent hypoxia / poor perfusion / placental insufficiency

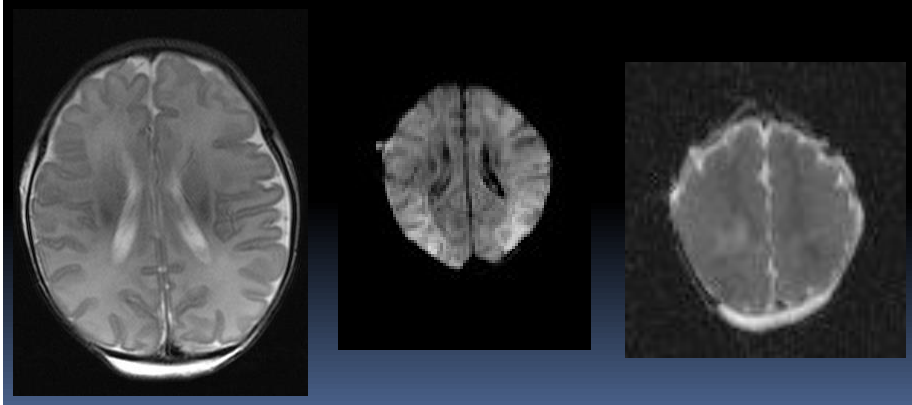
76

Chronic partial hypoxia

- Gospel: Normal infant can withstand 1 hour of such an insult before brain damage begins

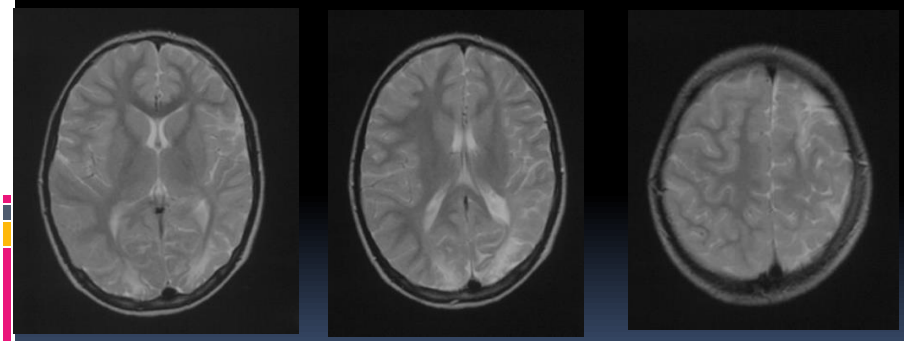
77

Chronic partial insult:neonatal imaging



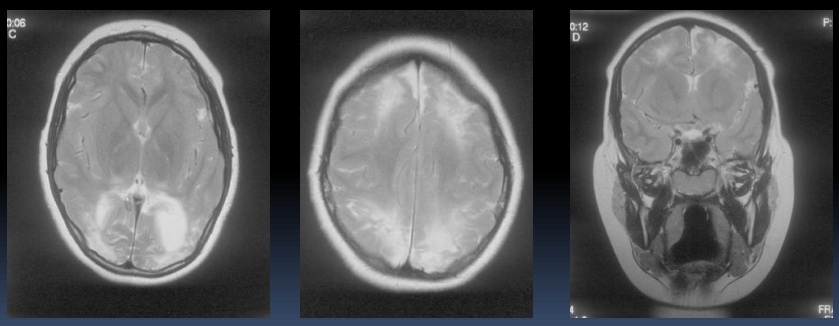
78

Chronic partial insult: later imaging



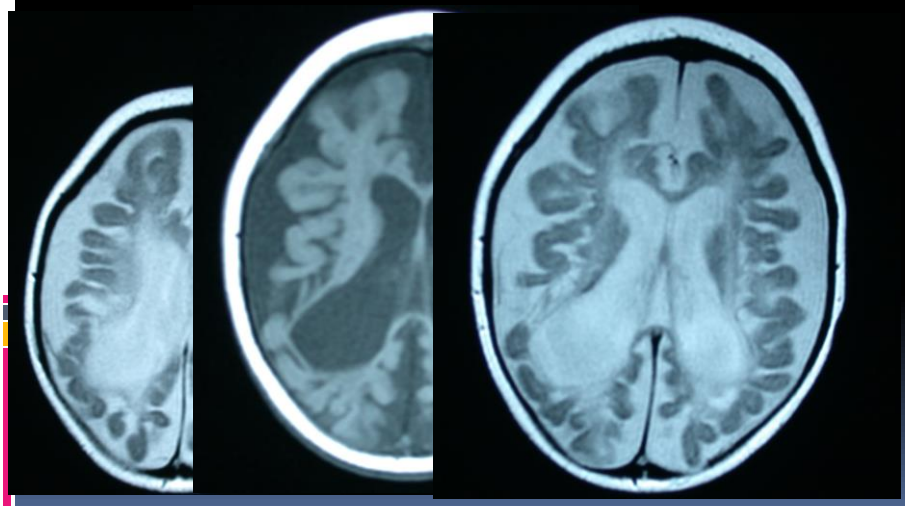
79

Chronic partial insult: later imaging gliosis,atrophy and ulegyria



80

Severe chronic partial insult



81

Progression of changes

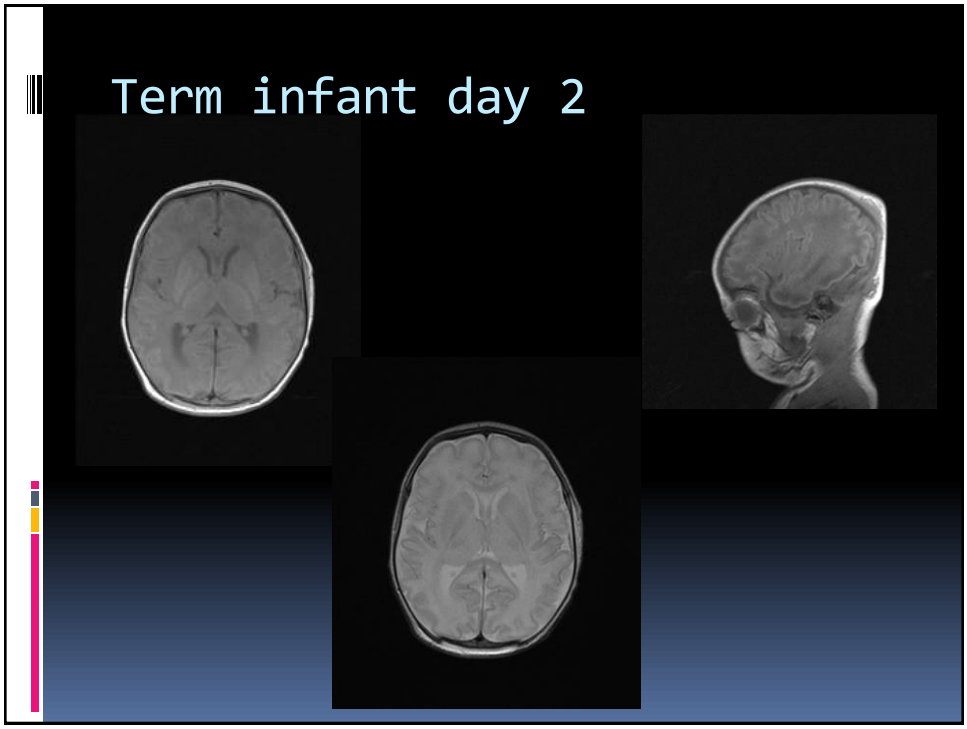
- Acute event

  Effects not static

  Scan appearances = snapshot

  Prognosis may not be clear from early scans alone

82



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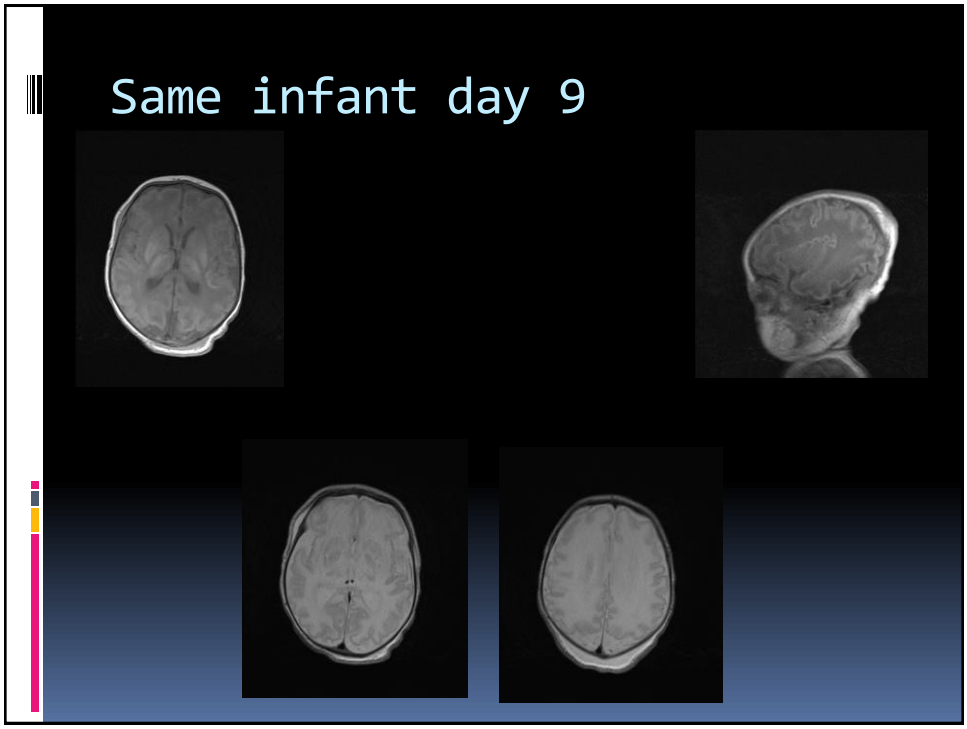


84

Longer term effects

- Severe injuries:
  - white matter volume loss
  - microcephaly
  - Chronic partial
    - white matter gliosis (more than just subcortical WM affected)
  - Acute near total
    - white matter normal signal (secondary degeneration)

85

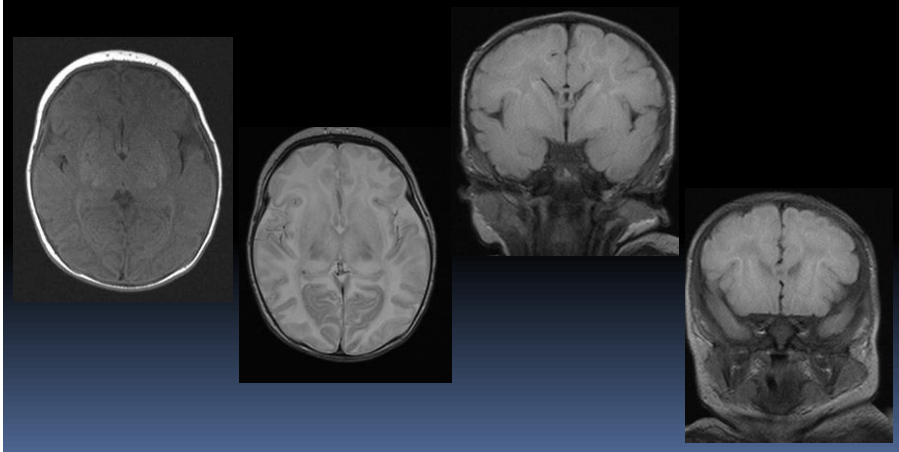
Mixed picture

- Severe chronic partial
  - can affect deep grey matter
  - more uniform basal ganglia and thalamic abnormality
  - white matter loss with gliosis
- Severe acute near total
  - more extensive deep grey matter involvement but
  - may be more widespread white matter involvement than just perirolandic
  - white matter loss without gliosis

86

Mixed insults: neonatal imaging

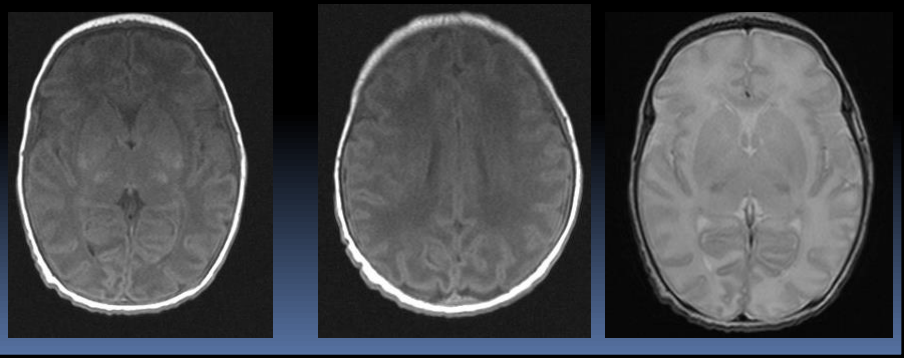
- Acute near total with chronic partial



87

Mixed insults: neonatal imaging

Acute near total insult with diffuse hemispheric signal change: commonly seen and resolves with no permanent change



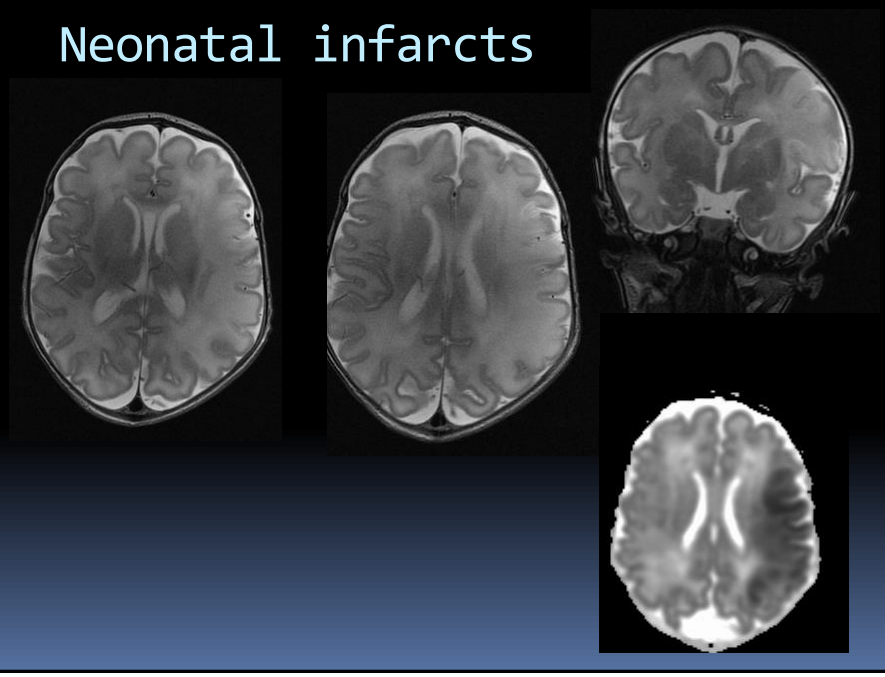
88

Perinatal infarcts

- No cause found in 25 – 47%
- Many conditions implicated as associated
- Asphyxia not really one of those conditions

89

Neonatal infarcts



90

Periventricular leukomalacia

- Commoner in pre-term infants
- Not causally specific
  - Hypoxia / hypoperfusion
  - Cytokine release due to ascending infection (chorio-amnionitis)
  - Hypocarbia
  - IVH

91

Periventricular leukomalacia

- Commonly occurs following insult ~ 26 – 34 weeks gestation
  - Damages pre-oligodendrocytes
  - Reduced white matter volume
- Causative insult at 28 weeks +
  - Gliosis and irregular ventricles
- Causative insult before 26 - 28 weeks (ish)
  - No gliosis and smoother ventricular margins

92

PVL

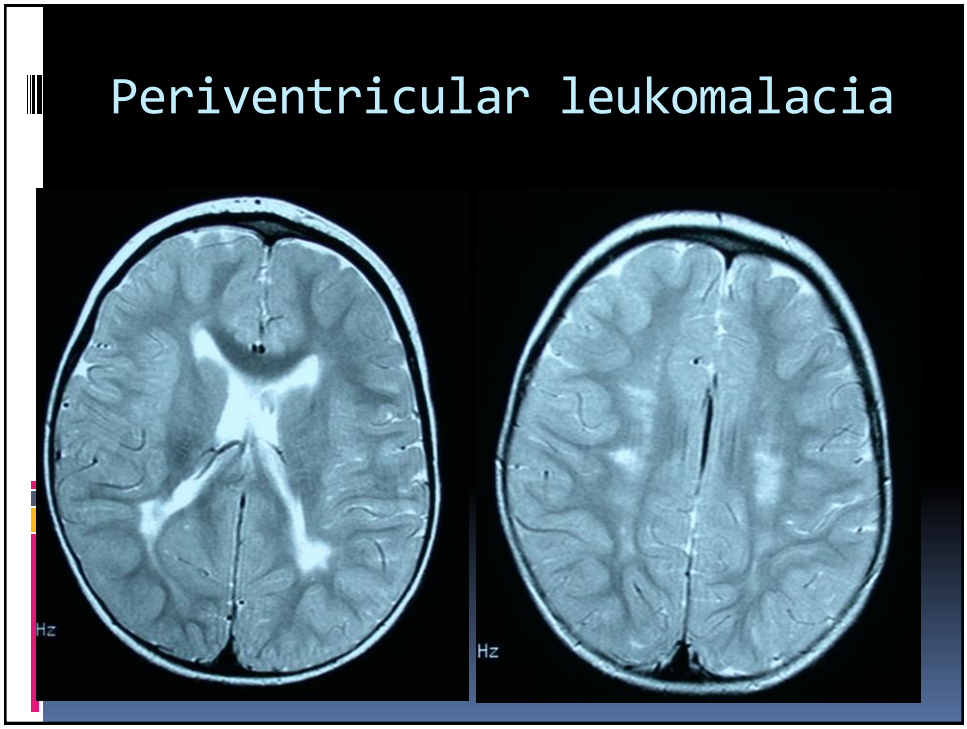
- Ultrasound
  - increased reflectivity min 24 - 48 hours post insult
  - cysts may evolve over 2 - 4 weeks
    - normal scan does not necessarily mean that insult has not already occurred

93

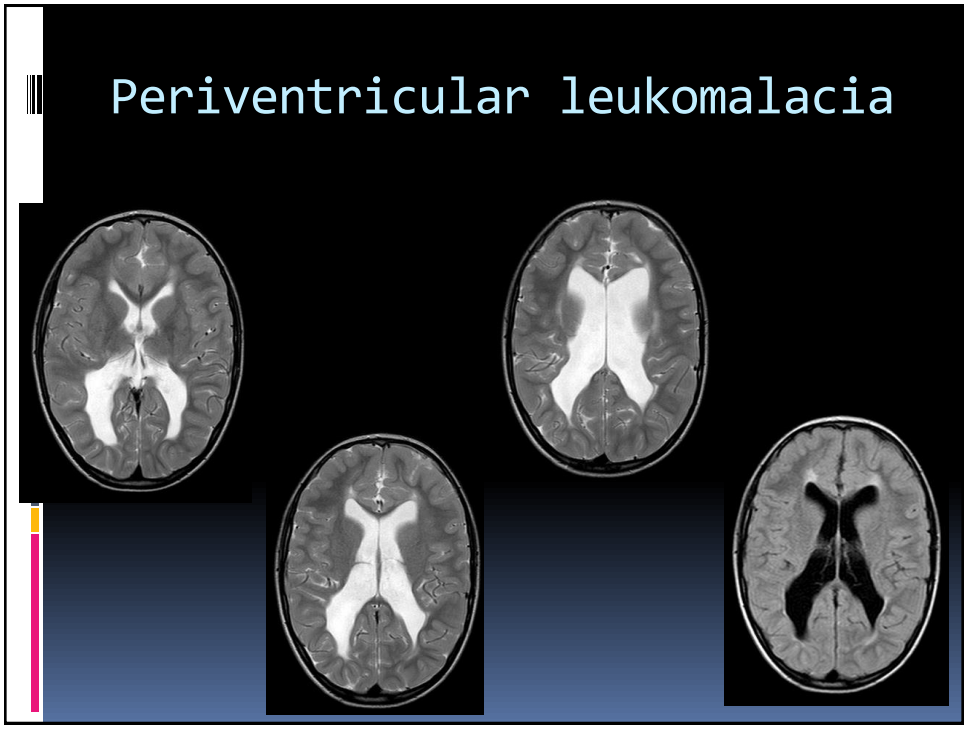
PVL

- MRI
  - classical appearances
    - large ventricles due to reduced volume of white matter
    - reduced white matter especially around trigones of posterior horns
    - irregular ventricular margins
    - gliosis (scarring) of residual white matter

94


Periventricular leukomalacia
Hz
Hz

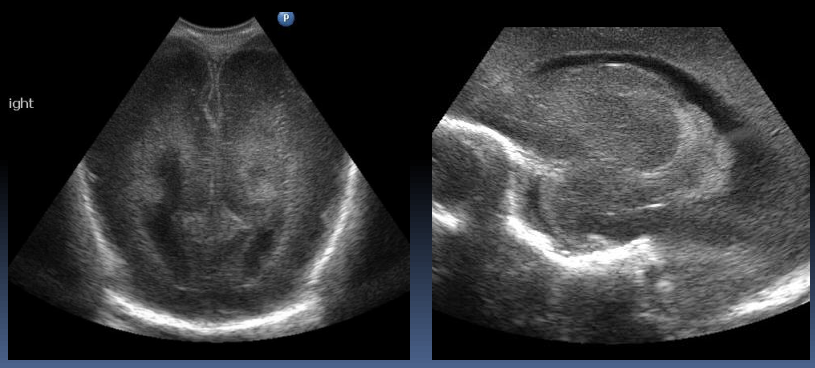
95


Periventricular leukomalacia

96

PVL

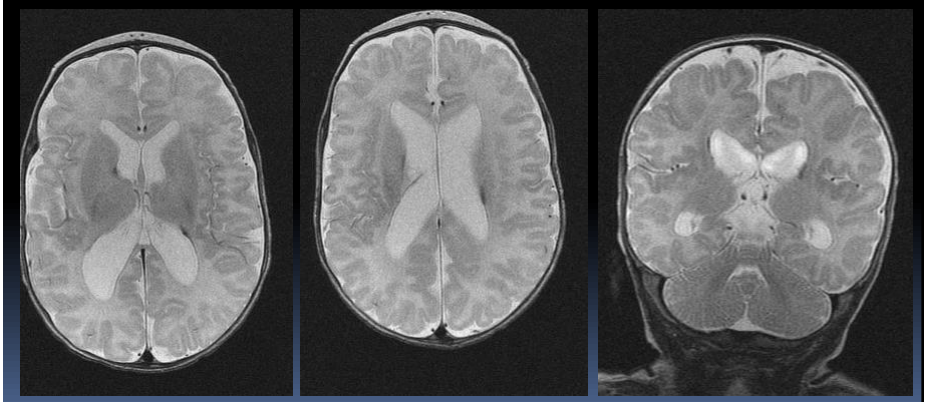
26 week twin: cerebral ultrasound day 1



97

PVL

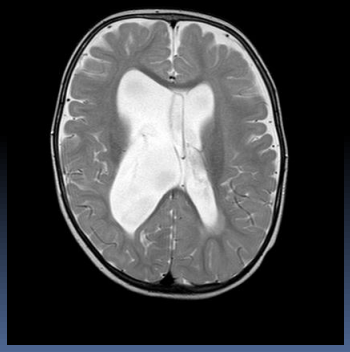
- MRI at 6 weeks post term

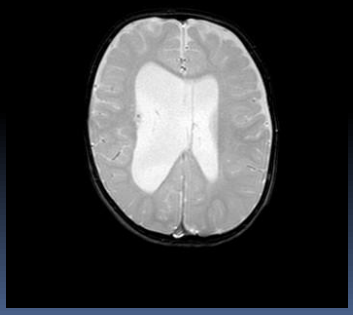


98

PVL

- 1 year later

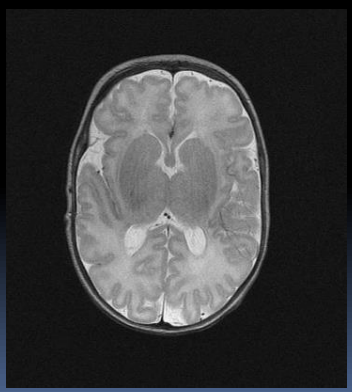




99

Prematurity not enough

Ex - prem 24 week scanned at term equivalent age



100

Hypoglycaemia

- Generalised insult
- Energy failure (cf hypoxia / hypoperfusion)
- Typically localised rather than generalised distribution
- Inferior parietal and occipital regions

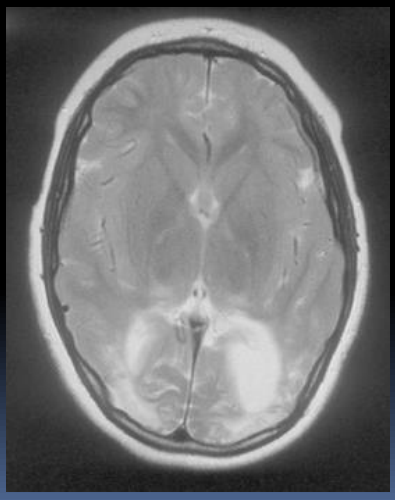
101

Hypoglycaemia

- Usually following profound / prolonged hypoglycaemia
- Unrecordable blood sugar
- In symptomatic infants: seizures

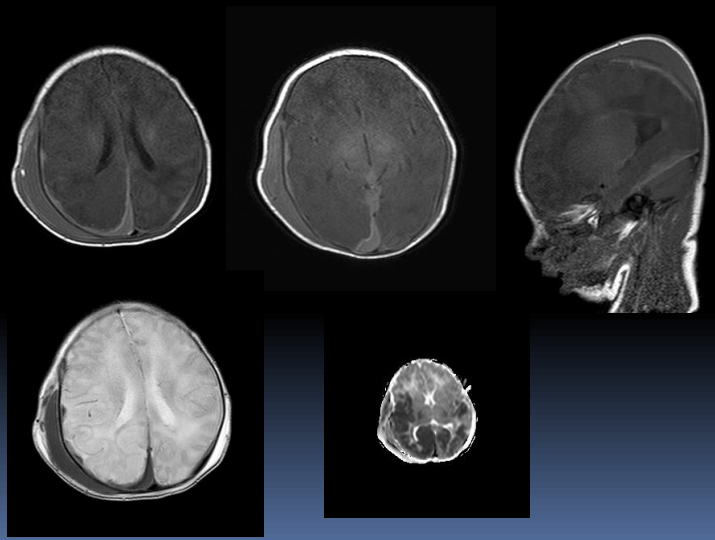
102

Hypoglycaemia



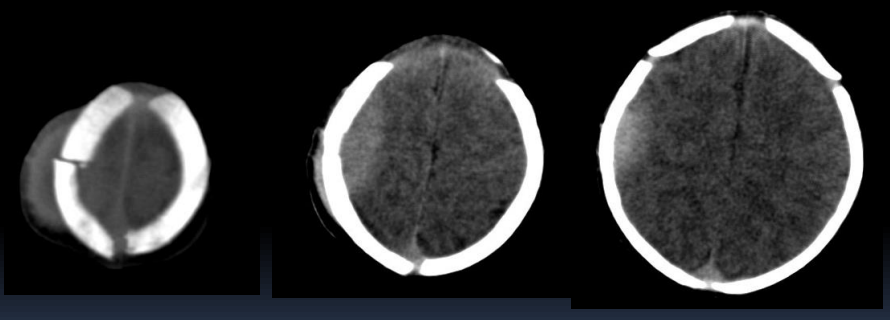
103

Birth trauma: Failed forceps



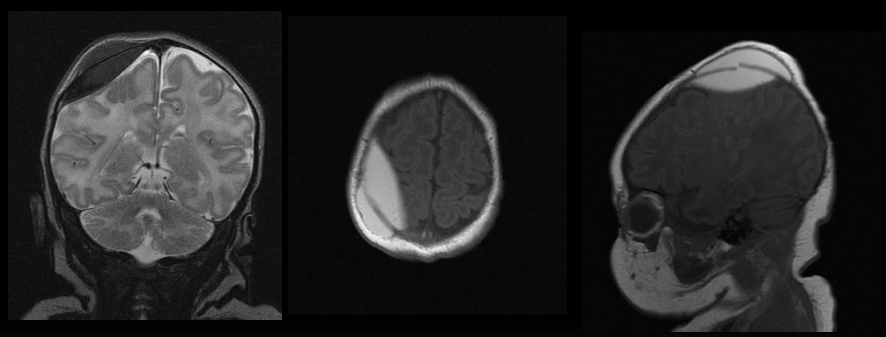
104

Birth trauma: Fracture and EDH



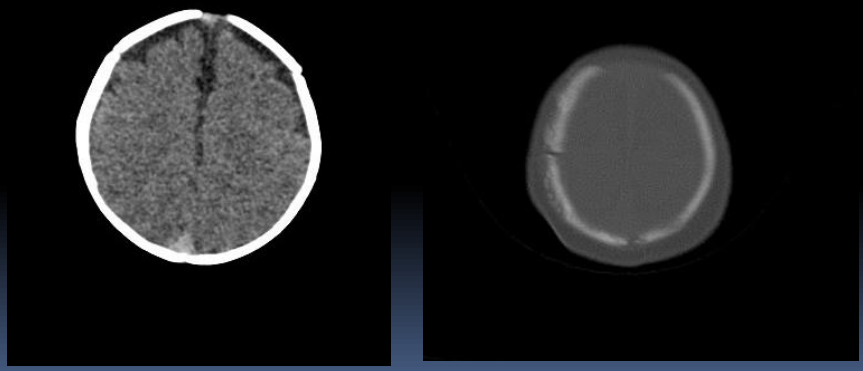
105

Fracture and EDH



106

6 Weeks later



107

Hypoxia and hippocampus

- Hippocampus very sensitive to hypoxia
- Hippocampal damage seen in cases of birth asphyxia
- Complex functions including memory circuits
- ?? Relation of birth asphyxia to later memory problems

108

Hypothesis

- Birth asphyxia causes hippocampal damage in absence of typical structural changes on scans
- Hippocampal damage leads to identifiable problems with certain types of memory
- Implies direct causal effect

109

Memory problems

- Impairments of episodic memory
  - Memory for events
- Relative preservation of semantic memory
  - Memory for facts

110

Structure v function

- Evidence base: main papers:
  - Gadian et al Brain (2000) 12: 499-507
  - Cooper et al Cerebral Cortex (2015) 25: 1469 - 1476

111

Results

- Both show reduced hippocampal volume in index cases
- Reduced regional deep grey matter volume
- Possible structural correlates

112

Evidence base

- Possible problems
  - Case selection
  - Study groups
  - Confounding variables
  - ? Study with infants with known structural damage secondary to HIE?

113

Conclusions

- MR best modality for demonstrating damage
- Assessment of long term damage best made at 2 years +
- Good at assessing type of insult(s)
- Cannot assess timing of insult from scan appearances alone

114

Every picture tells a story

- It just might not be the one you want to hear!

115

116

Neil Stoodley

BIRTH INJURY: SOMETHING NEW?

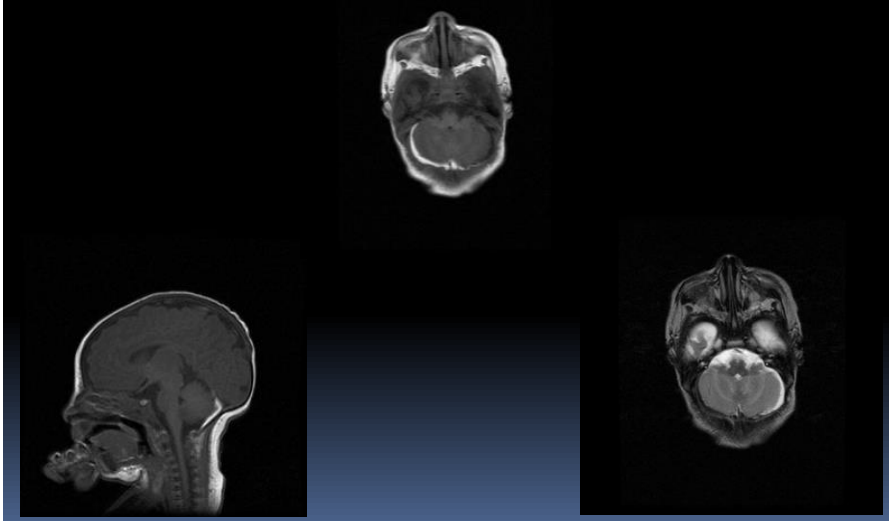
117

Birth-related subdural bleeding

- Normal term infants
- Incidence varies with mode of delivery
  - Elective Caesarean section <1%
  - Normal vaginal delivery 9%
  - Failed forceps and / or Ventouse 46%
  - Pathological studies 66%

118

Birth-related



119

Birth injury: Common mechanisms

- Asphyxia
  - Chronic partial
  - Acute near total
- Hypoglycaemia
- Trauma
  - Fractures
  - Extra-axial bleeds
  - Contusions

120

A different picture

- No definite typical ischaemic change
- Often no evidence fracture / direct trauma
- Early (days) subcortical cystic change
- Seen much earlier than in chronic partial encephalomalacia
- "Watershed" distribution
- Cysts usually haemorrhagic

121

Clinical

- Delivered with poor respiratory effort and bradycardia
- Variable Apgar scores
- Usually good cord gases
- Surprisingly pink

- Longer term: cognitive deficits commoner; not typical asphyxial outcomes

122

Unifying history

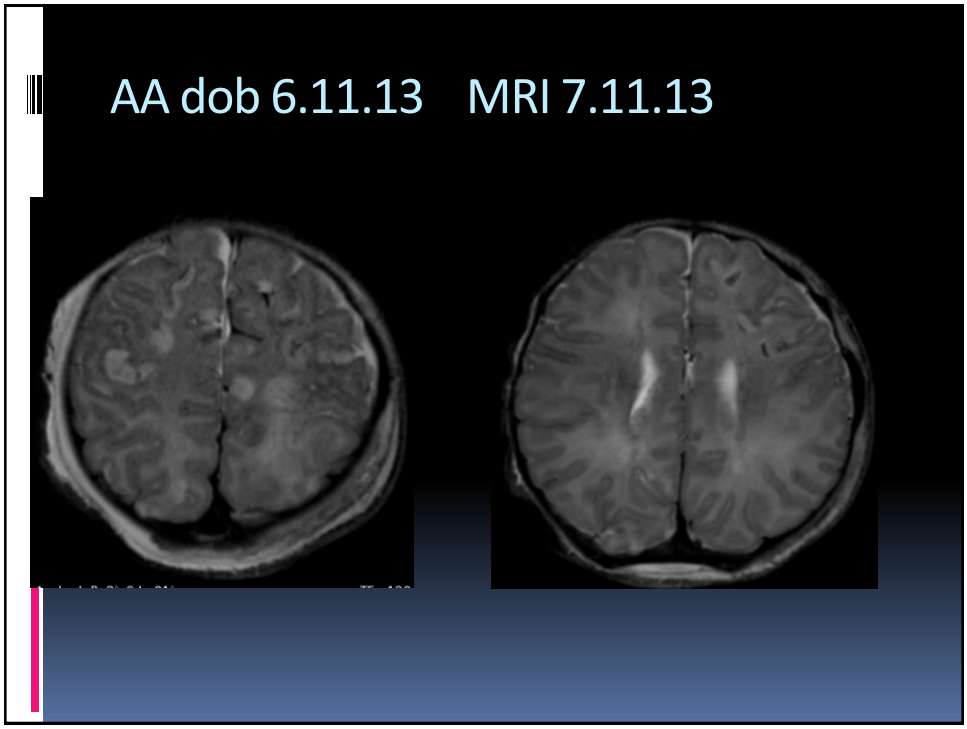
- Prolonged second stage
- Impaction of fetal head
- Difficult disimpaction
  - Instrumental delivery
  - More often emergency Caesarean with disimpaction from below

123

AA

- 41 weeks
- Full dilatation 1445; no progress at 1635
- Decision for trial of instrumental delivery
- Review: decision EmLSCS starts 1805
- ST2 can't deliver head; neither can ST4
- Consultant disimpacts head from below
- Delivery 1811
- Apgars 0 at 1,5,10
- Cord gases pH 7.24 base excess -7.4mmol/l

124



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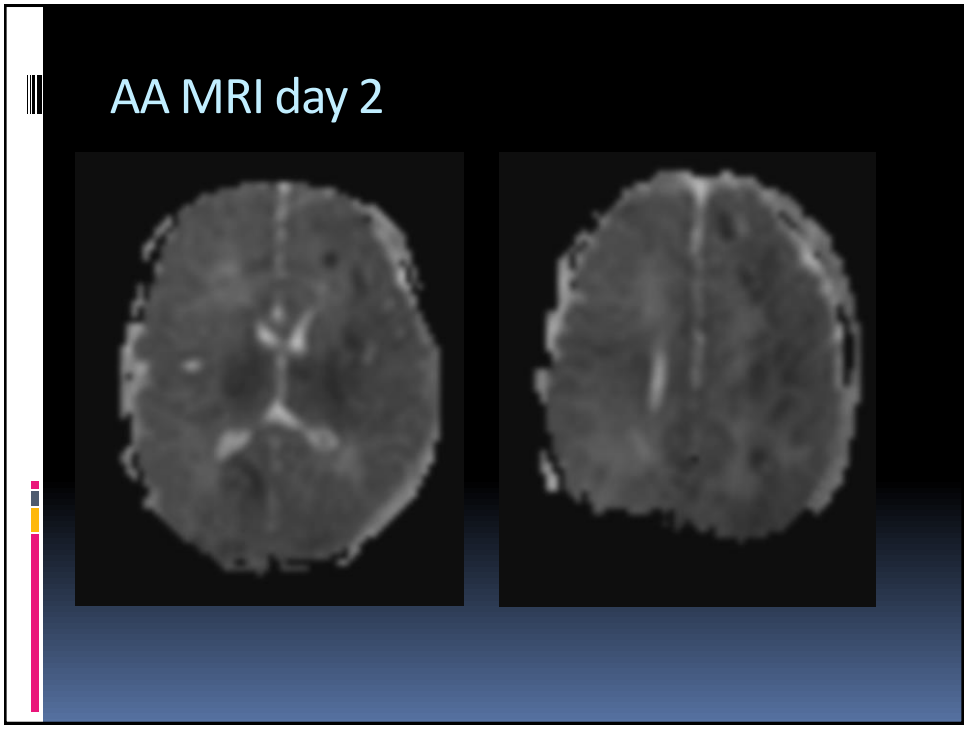


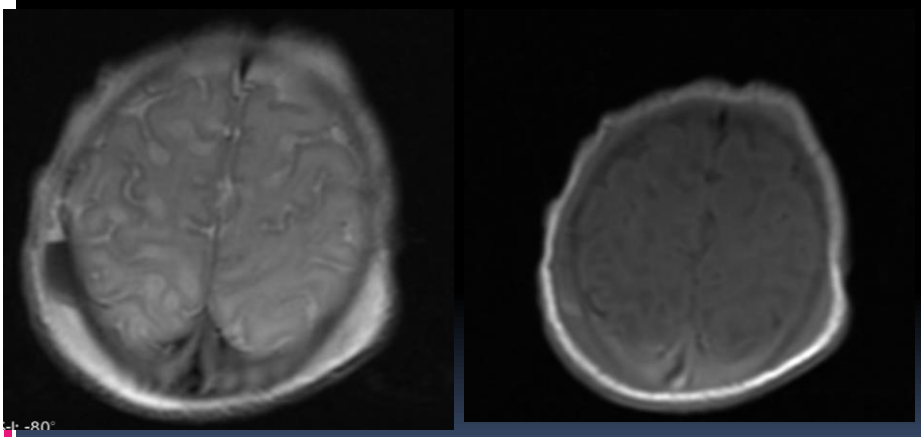
126

BB

- 36 weeks
- FD 0733; variable decels 0820
- Decision for trial instrumental 0900
- Forceps on 0935; traction 0937 and 0940
- Forceps off : Em LSCS
- Delivered 0945
- Apgars 9 at 1, 10 at 5 and 10
- Arterial pH 7.22 BE -7.7mmol/l

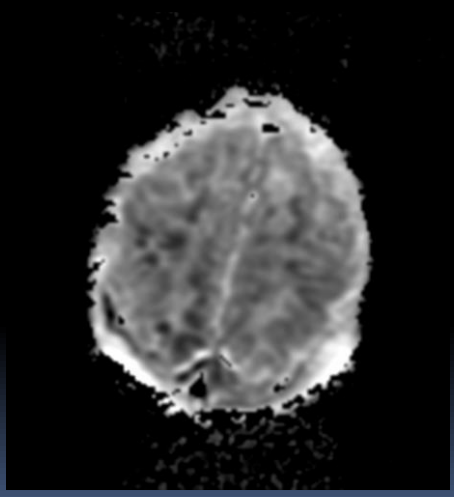
127

BB MRI day 2



128

BB MRI day 2

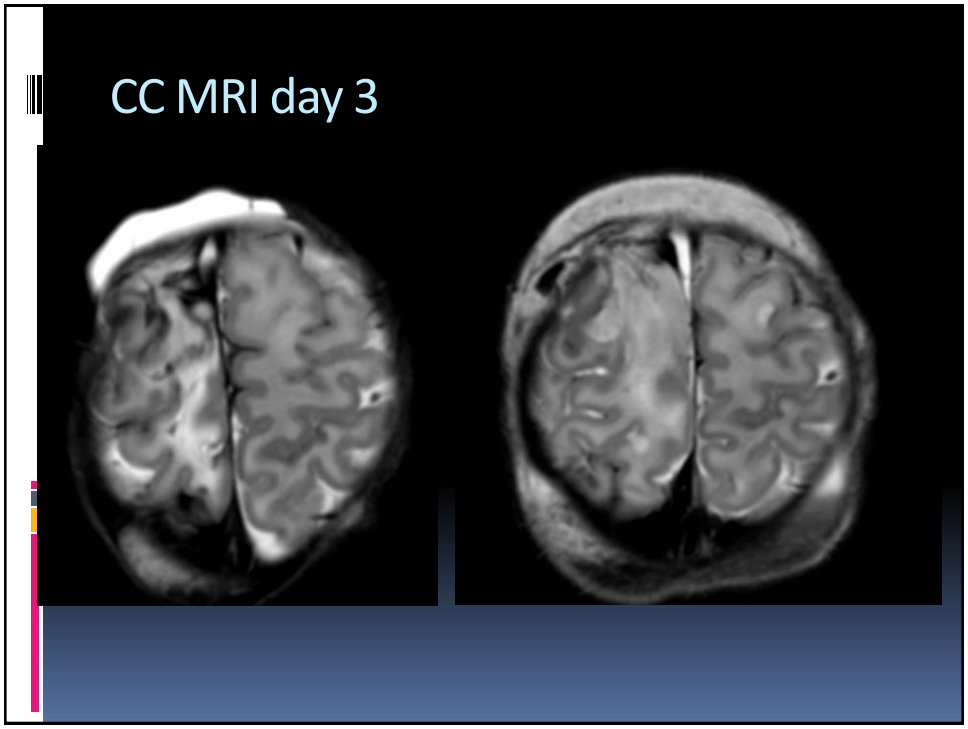


129

CC

- Twin IOL 37 weeks
- Trial of instrumental 2155: Ventouse x 4
- Delivery 2250
- Apgars 7 at 1, 9 at 5
- pH 7.14 BE -9.6
- Large scalp haematoma and anaemic

130



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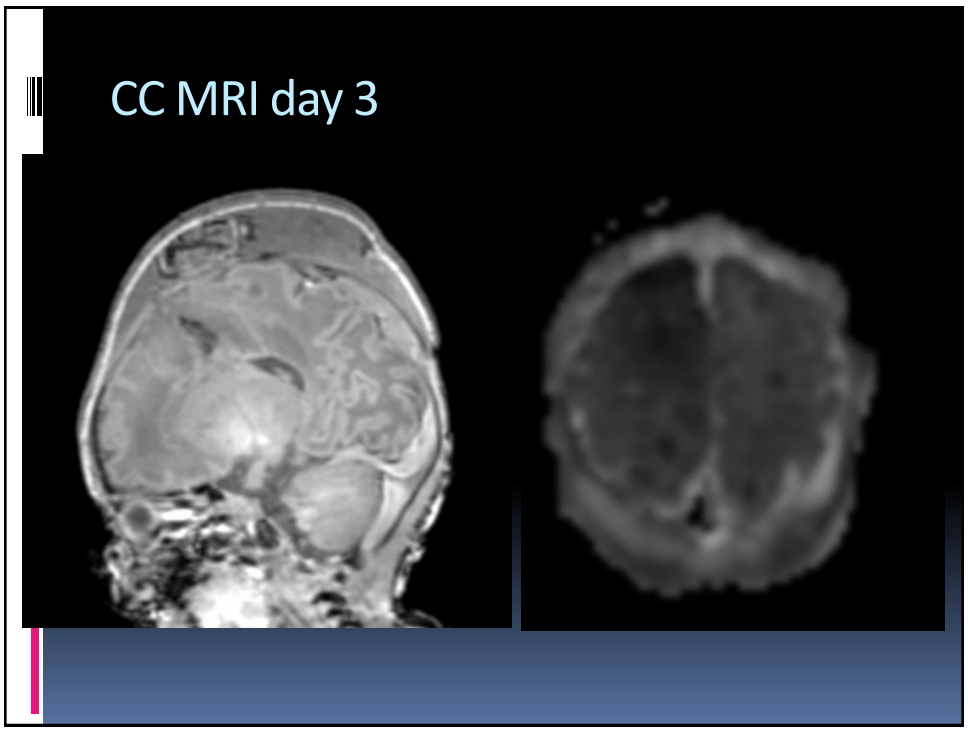


132

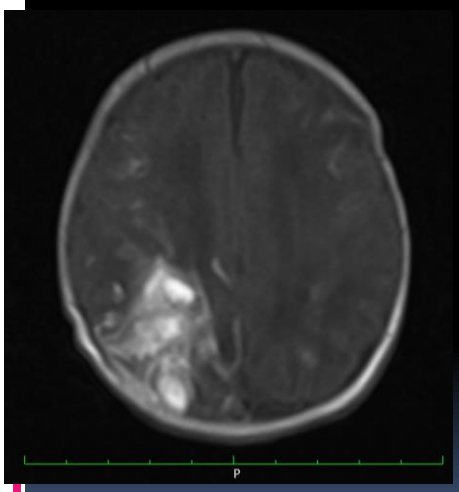
DD

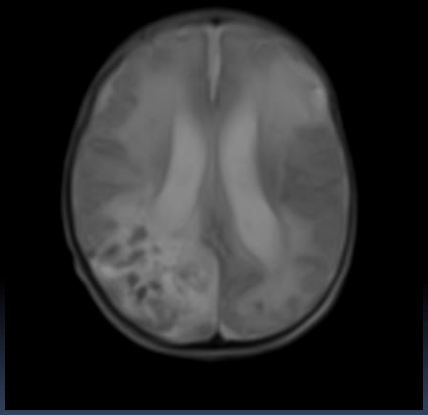
- SROM at 35 weeks: IOL and Synto 1245
- CTG pathological 1720: EmLSCS
- Delivery 1805
- Apgars 3 at 1, 9 at 5 and 10 at 10
- pH 7.15, BE -5.3mmol/l

- CT day 2: right parietal fracture

133

DD MRI day 8





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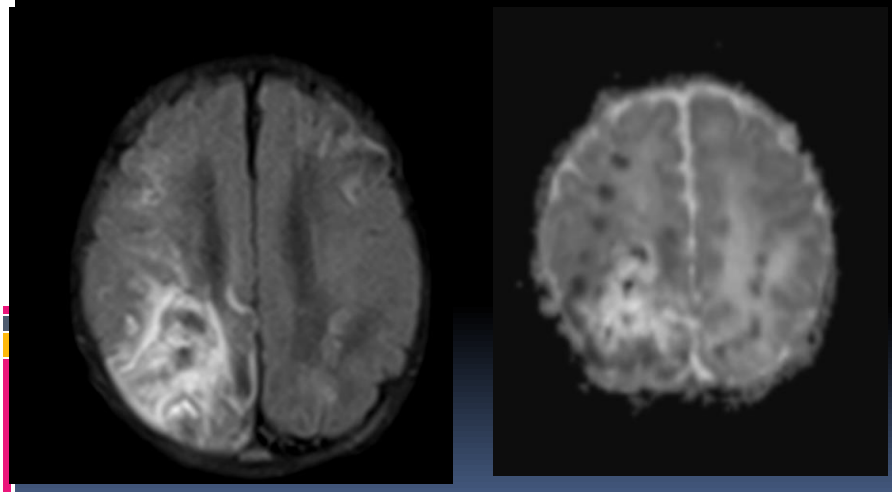
DD MRI day 8



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EE

- IOL at term
- Full dilatation 1819; no progress by 1928
- Trial of instrumental 1940: EmLSCS 2026
- Delivery 2034
- pH arterial 7.17 BE -6.3mmol/l
- pH venous 7.2 BE -6.6mmol/l
- Grunting therefore NNU
- CT: right-sided SDH: decompressive craniectomy

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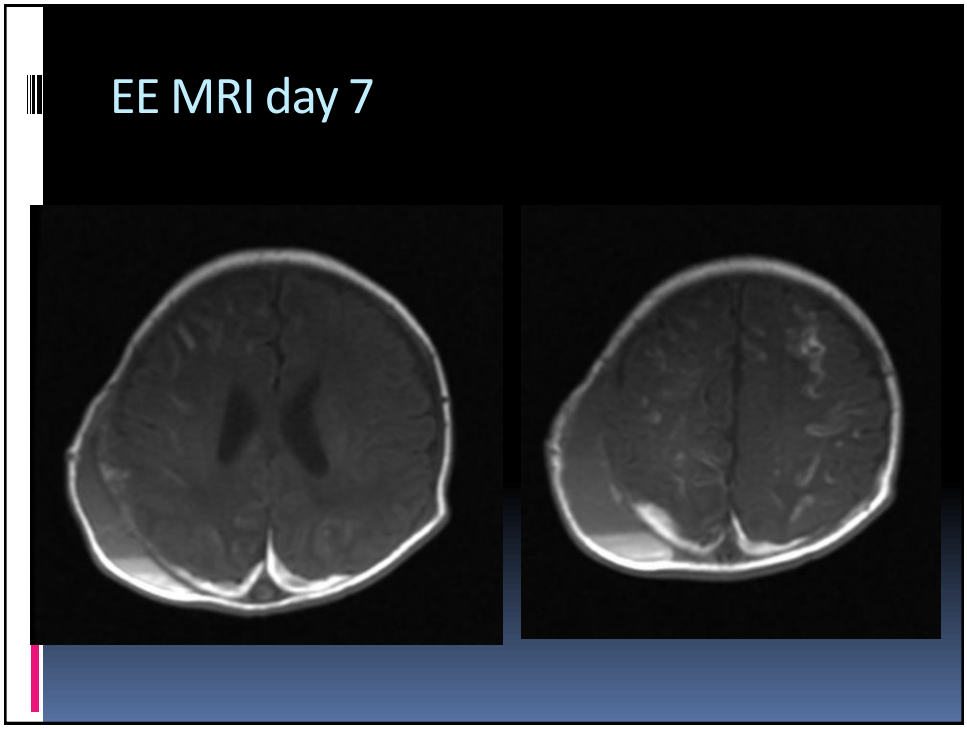

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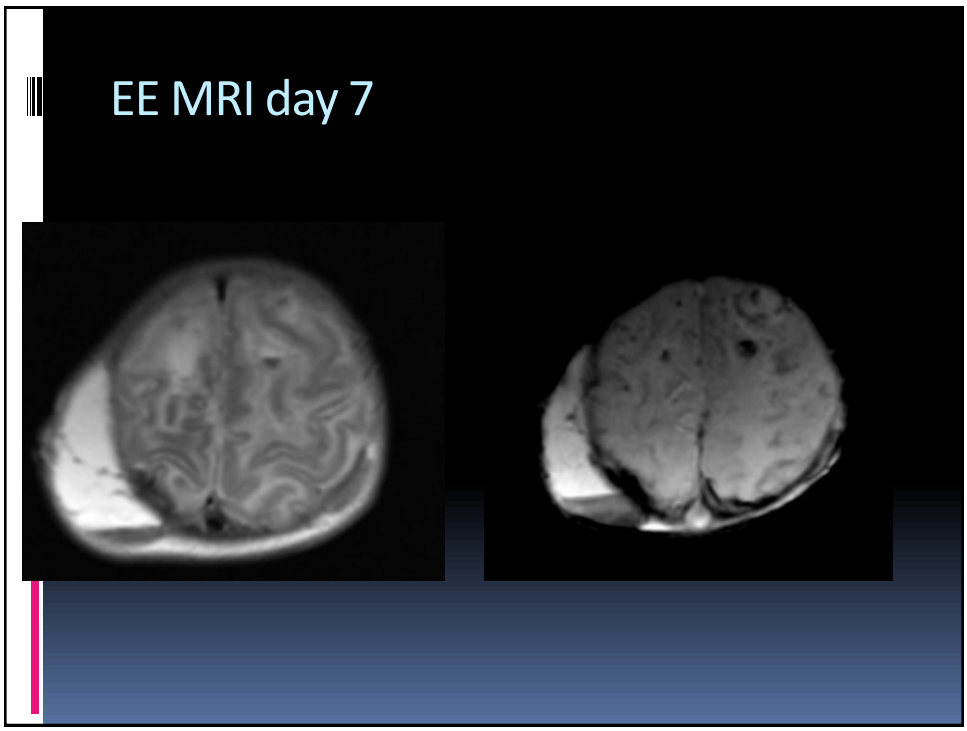

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FF

- Spontaneous onset labour at term
- FD 0047; pushing 0200; decelerations
- Trial of instrumental at 0259; Ventouse x 3
- Delivery 0315
- Apgars 0 at 1 and 5, 3 at 10 and 15, 6 at 20

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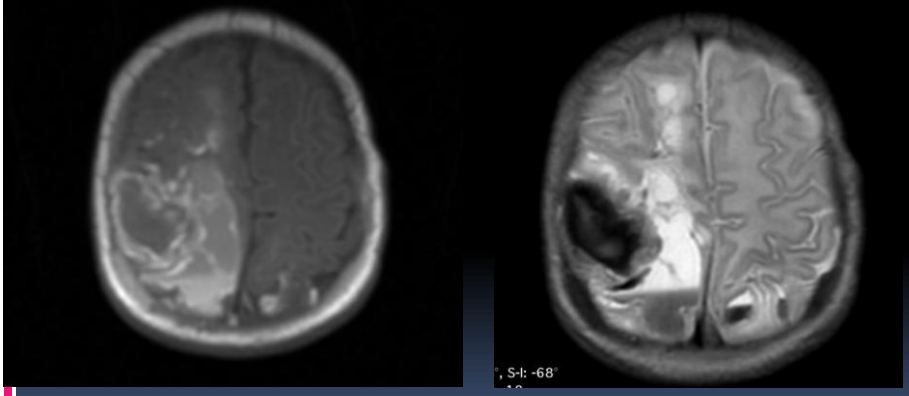
FF MRI day 6



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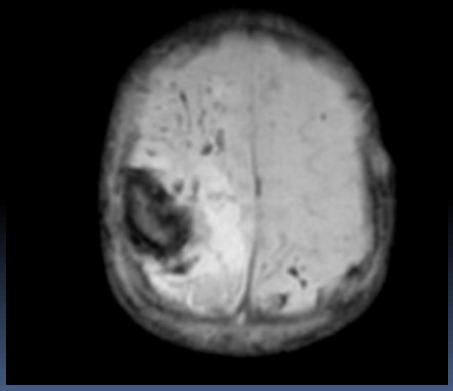
FF MRI day 6



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Hypothesis

- Different mechanism of injury
- Venous hypertension secondary to raised intrathoracic pressure
- Exacerbated by direct head pressure associated with impaction
- Venous watershed ischaemia
- More often associated with haemorrhage

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Hypothesis

- Multifocal subcortical haemorrhagic lesions
- Similar appearance to traumatic diffuse axonal injury
- Obviously different mechanism but…
- Similar outcome??
- Possible cause of cognitive problems

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