12
King's Bench Walk

Birth Injuries – Clinical Negligence Mini Series

Causation: a neonatologist's perspective.

Dr Kevin Ives

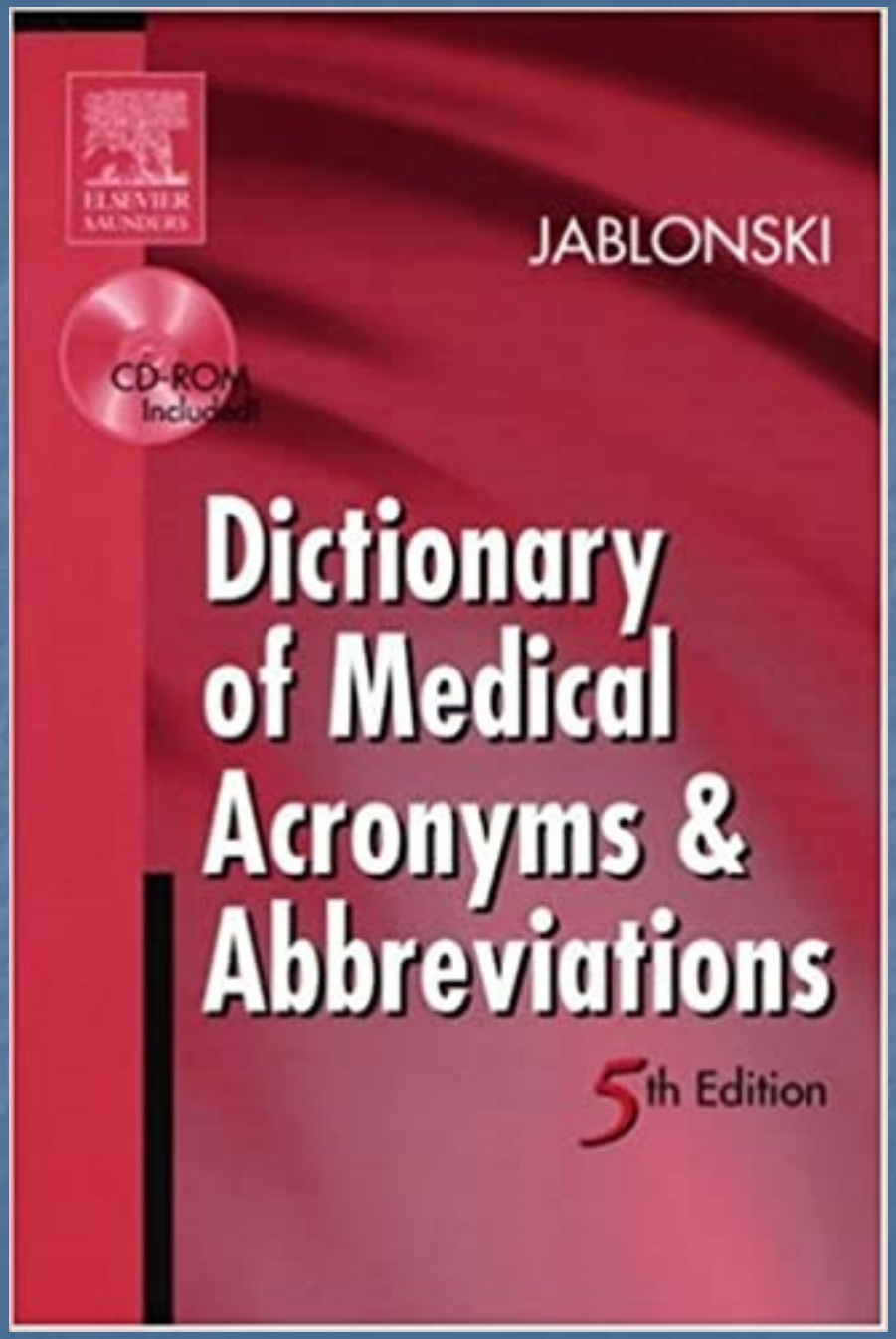
March 2023

Your world

Oxford University Hospitals NHS

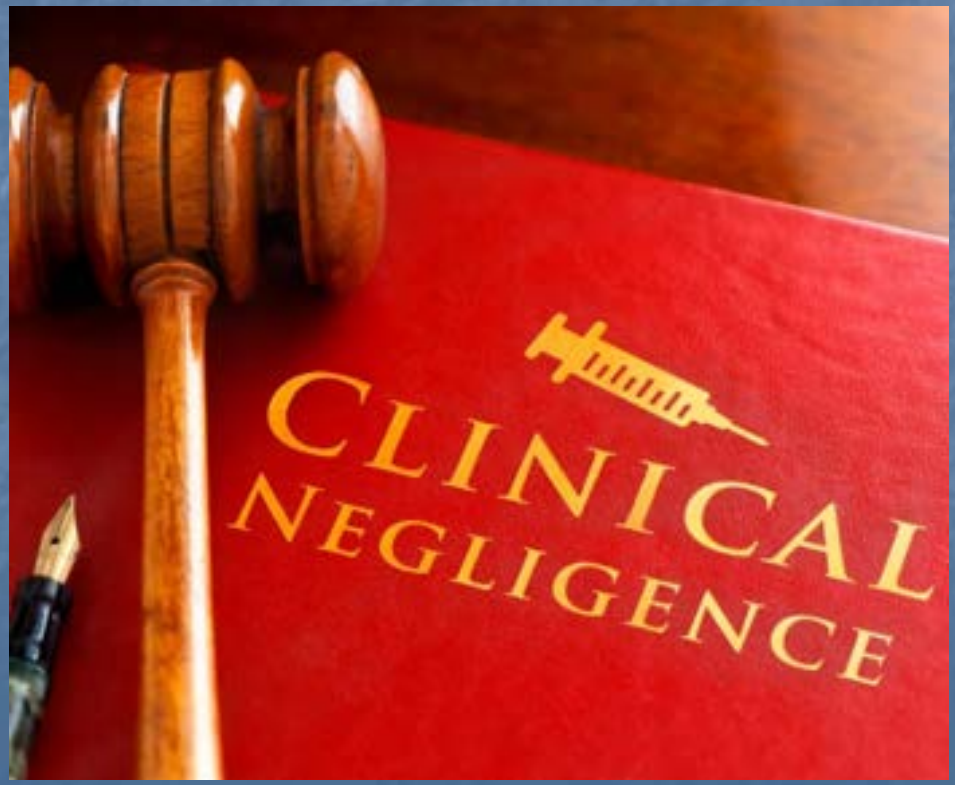
NHS Trust

My world



Both worlds converse in TLAs

Three Letter Abbreviations



Conditions associated with legal claims in neonatology

- HIE hypoxic ischaemic encephalopathy
- PVH periventricular haemorrhage
- PVL periventricular leukomalacia
- ROP retinopathy of prematurity
- NEC necrotising enterocolitis
- AIS arterial ischaemic stroke
- CLD chronic lung disease

Conditions associated with legal claims in neonatology

- HIE
- PVH
- PVL
- ROP
- NEC
- AIS
- CLD
- Hypoglycaemia
- Infection
- Drug error
- Extravasation
- Limb ischaemia
- Kernicterus
- Death

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Birth Injuries

- **Birth injury** refers to damage or injury to the fetus/infant before, during, or just after the birthing process.
- **Birth trauma** refers specifically to mechanical damage sustained during delivery
- The two may be combined.

Causation and omissions:

- Failure to detect and follow up a significant abnormality on newborn examination.

Examples

- *Congenital heart disease*
- *Developmental dysplasia of the hip*
- *Cataracts*
- *Hydrocephalus*

iatrogenic

/ʌɪˌatrə(ʊ)ˈdʒɛnɪk/

adjective

relating to illness caused by medical examination or treatment.

injuries

‘Brought forth by healer’

Anticipated complications of premature or term birth

Not necessarily any fault of the carer

Conditions that have potential iatrogenic causes

- Drug reaction
- Pneumothorax
- Retinopathy of prematurity (ROP)
- Intestinal perforation
- Extravasation injury
- Limb ischaemia

Clinical negligence and causation in iatrogenic injuries

A claim may be made when there has been an unacceptable delay in recognising or treating these disorders that results in harm.

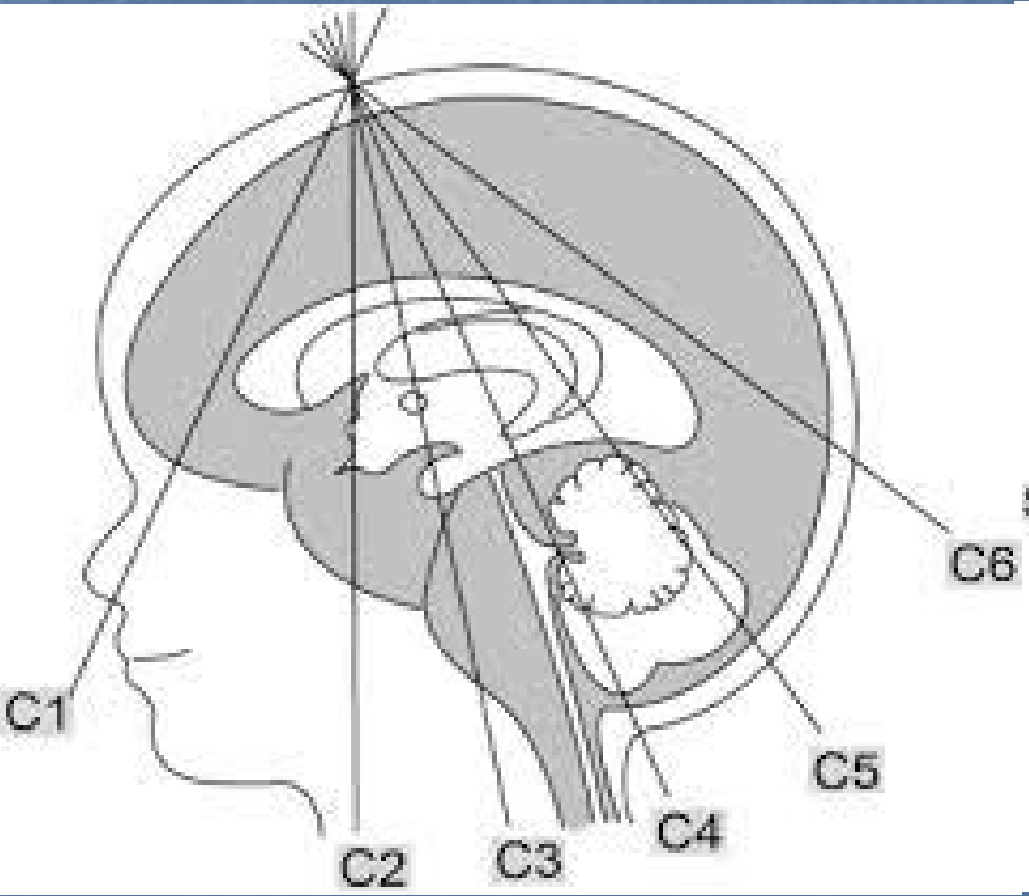
Causation moves with the times

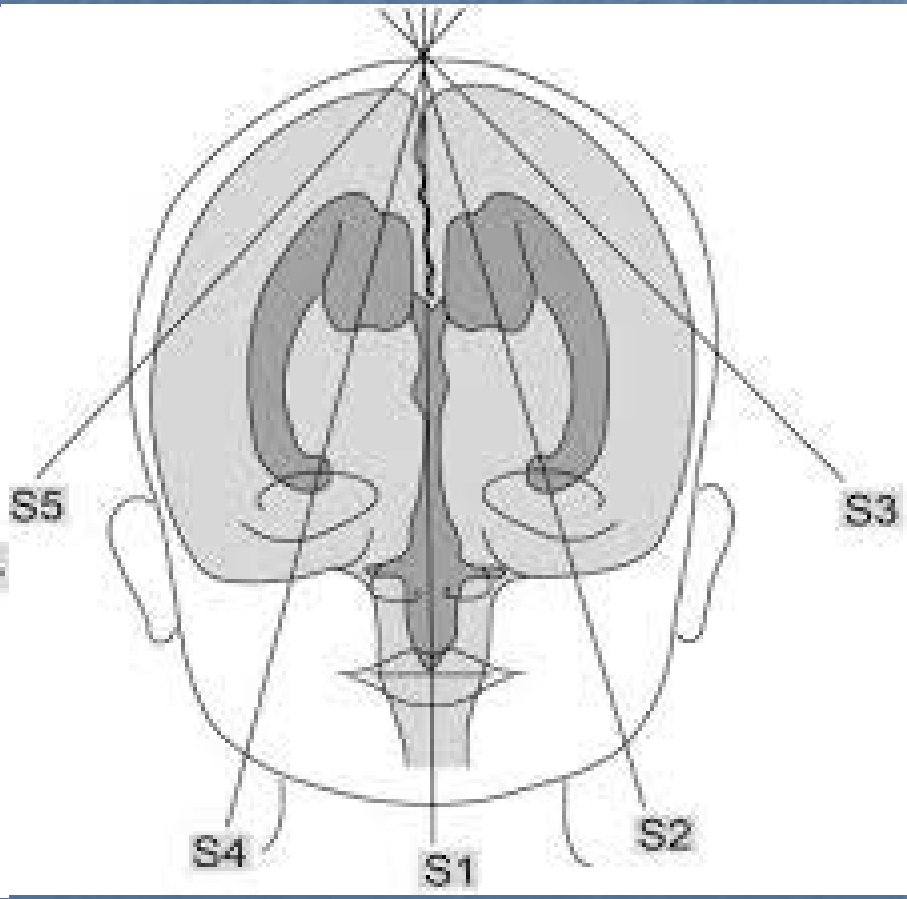
Opinion on causation reflects latest science.

Examples in neonatology:

- *Recognition of link between maternal chorioamnionitis and cerebral white matter injury → cerebral palsy*
- *Brain damaging consequences of hypocarbia (low carbon dioxide levels from over-ventilation) in preterm infants → PVL*

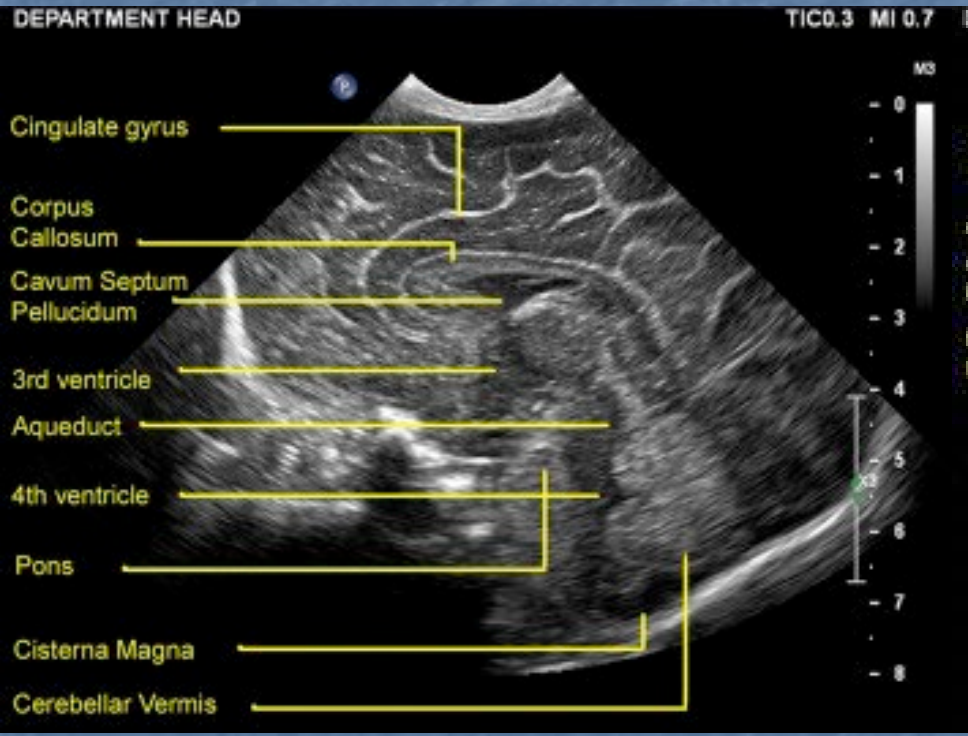
Cranial ultrasonography

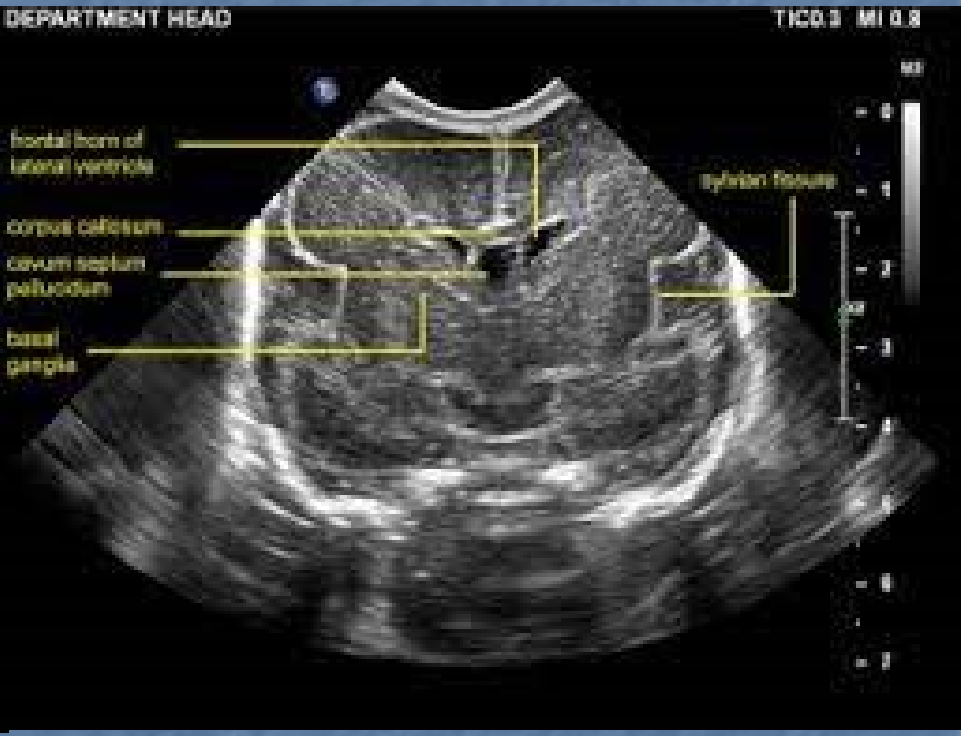




Dr Janet Rennie

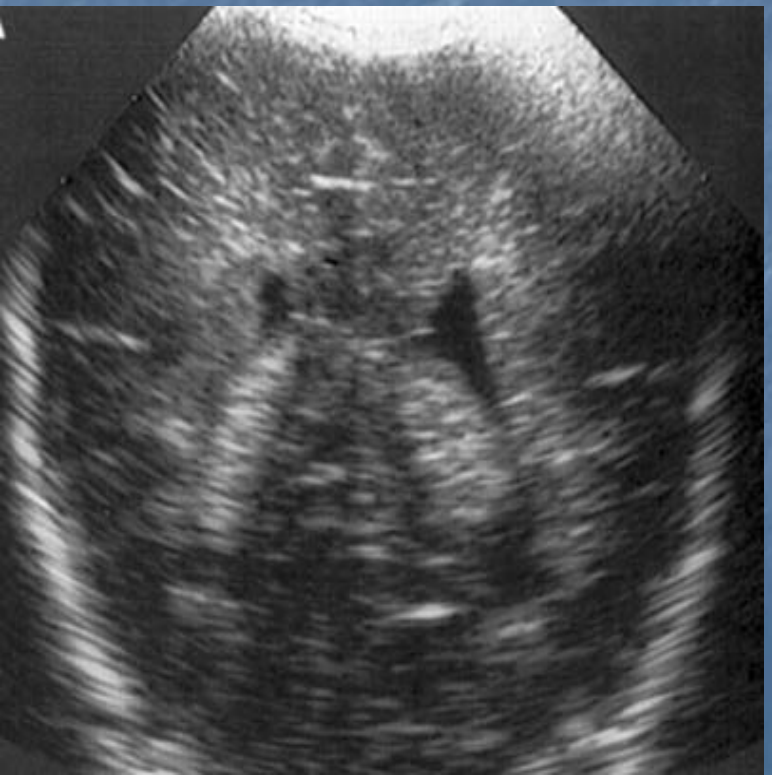
Cranial ultrasonography





Neonatology: causation

Cystic periventricular leukomalacia (PVL)

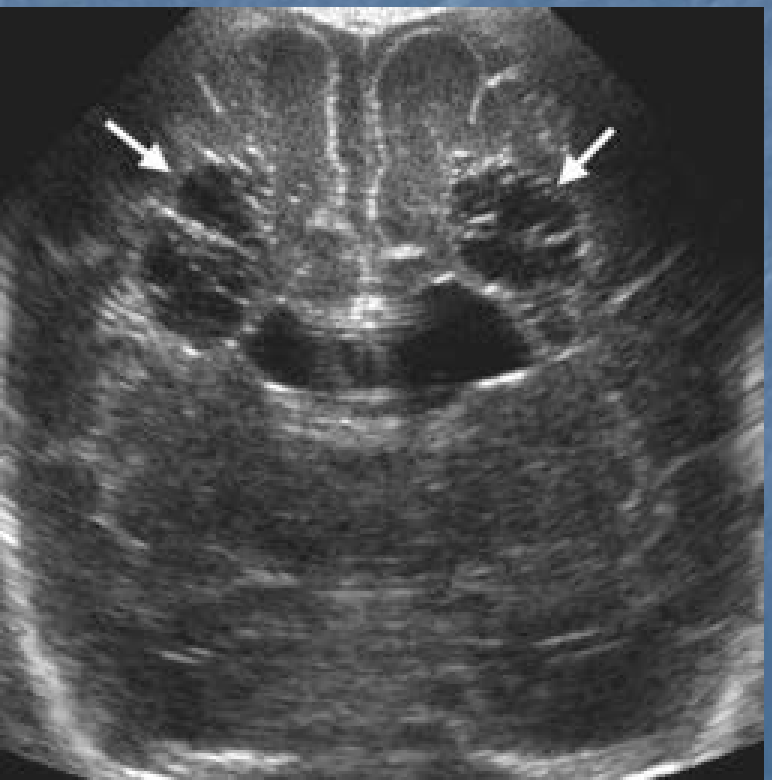


- Chorioamnionitis
- Untreated postnatal hypotension
- Postnatal collapse
- Over-ventilation with sustained hypocarbia

  $PaCO_2 < 2.67$ kPa (20mmHg.)

Neonatology: causation

Cystic periventricular leukomalacia (PVL)



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'Sustained hypocarbia'

PEDIATRICS®

OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Hypocarbia was defined as $Paco_2$ <35 mm Hg, and cumulative index of exposure (CIE) to hypocarbia was calculated as (35 − $Paco_2$) multiplied by time interval in hours for each 6-hour block in a 24-hour day.

Cumulative Index of Exposure to Hypocarbia and Hyperoxia as Risk Factors for Periventricular Leukomalacia in Low Birth Weight Infants

Seetha Shankaran, John C. Langer, S. Nadya Kazzi, Abbot R. Laptook, Michele Walsh and for the National Institute of Child Health and Human Development Neonatal Research Network

Pediatrics 2006;118;1654-1659

'Sustained hypocarbia'

Contents lists available at ScienceDirect

Early Human Development

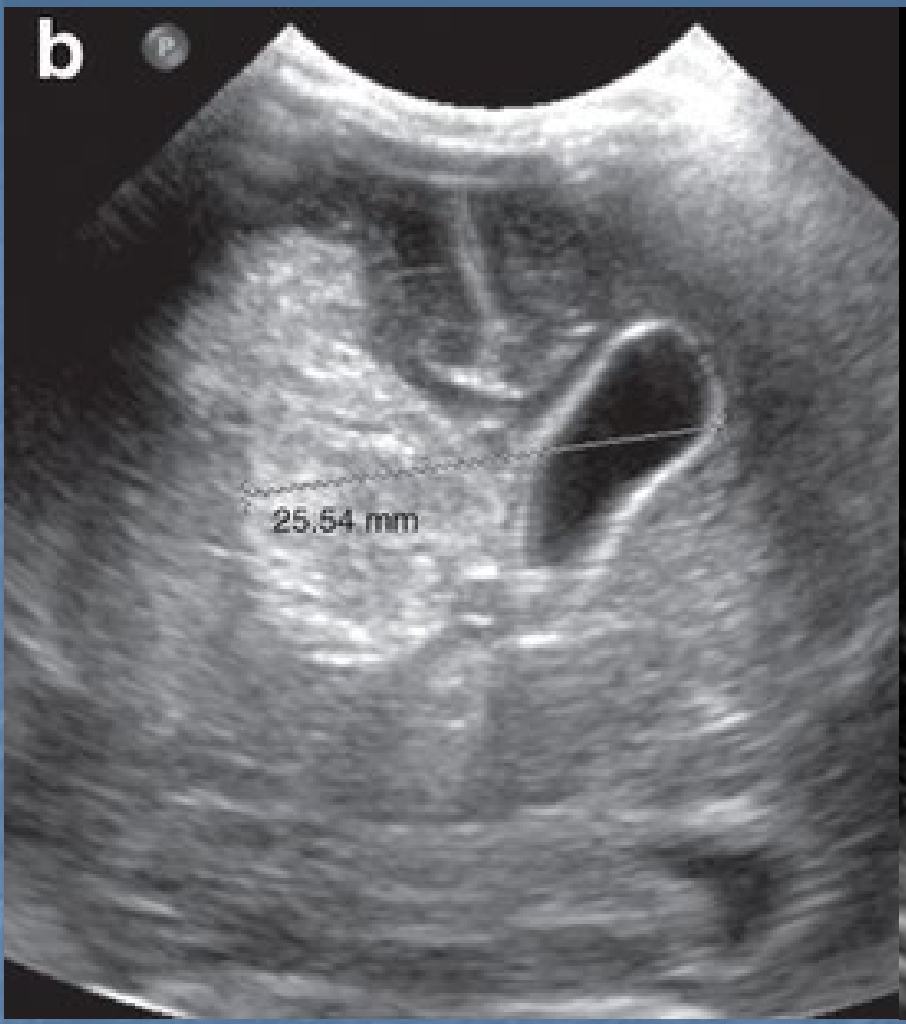
journal homepage: www.elsevier.com/locate/earlhumdev

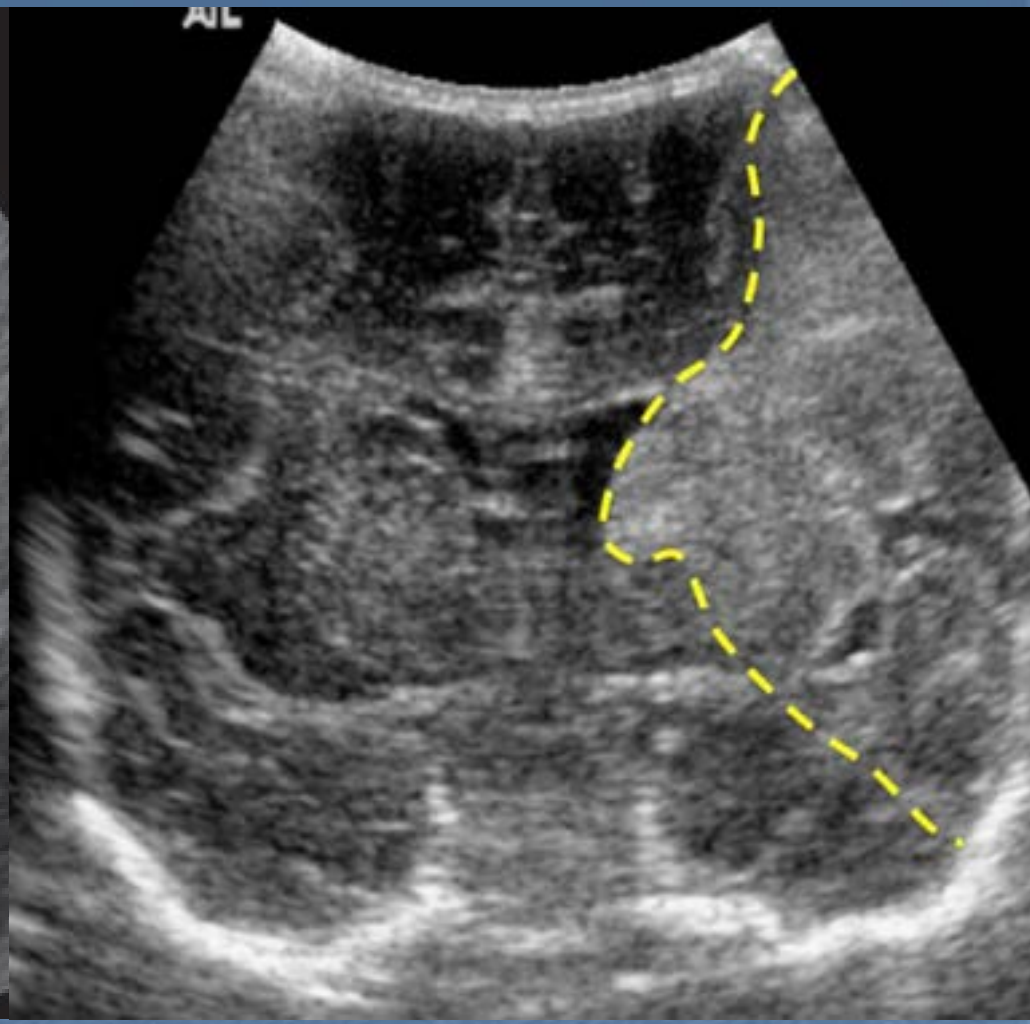
Episodes of hypocarbia and early-onset sepsis are risk factors for cystic periventricular leukomalacia in the preterm infant

B. Resch [a,b,*], K. Neubauer [a], N. Hofer [a], E. Resch [a], U. Maurer [c], J. Haas [d], W. Müller [b]

[a] Research Unit for Neonatal Infectious Diseases and Epidemiology, Medical University of Graz, Austria
[b] Division of Neonatology, Department of Pediatrics, Medical University of Graz, Austria
[c] Ambulatory of Neurodevelopmental Follow-up, Department of Pediatrics, Medical University of Graz, Australia
[d] Medical Statistics, Department of Gynecology and Obstetrics, Medical University of Graz, Austria

Hypocarbia was defined as an arterial carbon dioxide partial pressure (pCO_2) less than 35 mmHg (<4.67 kPa) for at least 1 h within the first five days of life.





Venous infarction - v - Arterial stroke

Arterial strokes and venous infarcts

Neonatal stroke

M A Rutherford,[1,2] L A Ramenghi,[3] F M Cowan[2,4]

Arch Dis Child Fetal Neonatal Ed 2012;**97**:F377–F384.

Risk Factors and Presentations of Periventricular Venous Infarction vs Arterial Presumed Perinatal Ischemic Stroke

Arch Neurol. 2010;67(7):842-848.

Adam Kirton, MD, MSc, FRCPC; Manohar Shroff, MD; Ann-Marie Pontigon, BScH; Gabrielle deVeber, MD, MHSc, FRCPC

Neonatology: causation

- Hypoxic ischaemic encephalopathy (HIE)

- Delay in calling paediatrician
- Standard of resuscitation
- Equipment failure
- Failure to cool?

Definitions

- Hypoxia or Anoxia: partial or complete lack of oxygen delivery to a tissue or organ
- Ischaemia: reduction or cessation of blood flow to a tissue or organ

- Hypoxic ischaemic encephalopathy: abnormal neurology following an hypoxic ischaemic event

1-2: 1000 deliveries.

Causes of neonatal HIE

Maternal
Cardiac arrest
Septic shock
Anaphylactic shock
Hypovolaemic shock
Status epilepticus

Uteroplacental
Placental abruption
Uterine rupture
Cord prolapse
Hyperstimulation
Placental failure

Fetal
Feto-maternal haemorrhage
Twin to twin transfusion
Severe Rhesus disease
Cardiac arrhythmia
Cordocentesis haemorrhage

When does hypoxic ischaemia occur?

- Before labour 20%
- During labour 35%
- Before and during labour 35%
- Following birth* 10%

*If bradycardic at birth, until heart rate >100

*Postnatal collapse requiring resuscitation

Fetal blood sampling (FBS)

Royal College of Obstetricians & Gynaecologists

FBS result (pH)	Interpretation
≥7.25	Normal FBS result. Repeat after 1 hour if CTG remains the same
7.21–7.24	Borderline FBS result. Repeat after 30 minutes
≤7.20	Abnormal FBS result. Consider delivery

Virginia Apgar score

At 1, 5 (and 10 minutes +)

	Sign	2	1	0
A	Appearance (skin color)	Normal over entire body	Normal except extremities	Cyanotic or pale all over
P	Pulse	>100 bpm	<100 bpm	Absent
G	Grimace (reflex irritability)	Sneezes, coughs, or vigorous cry	Grimaces	No response
A	Activity (muscle tone)	Active	Arms and legs flexed	Absent
R	Respirations	Good, crying	Gasping, irregular	Absent

Normal umbilical cord blood gases

Median (5^{th}-95^{th} centile ranges)	Umbilical artery n = 12,345	Umbilical vein n = 12,345
pH	7.27 (7.12-7.35)	7.35 (7.23-7.44)
$_pO2$ (kPa)	2.2 (0.8-3.7)	3.7 (2.2-5.3)
$_pCO2$ (kPa)	7.3 (5.6-9.8)	5.4 (3.8-7.1)
Base excess mmol/L	-3.0 (-9.3 to +1.5)	-3.0 (-8.3 to +2.6)
Lactate mmol/L	3.7 (2.0-6.7)	

From White et al. Australia & NZ J of Obs & Gynae 2010; 50:318-28

Check:
Are the samples both venous?
Could there be an air bubble effect?

Normal postnatal arterial blood gas

PH	7.35 - 7.45	7.35 – 7.45
pCO_2	4.7 - 6.0 kPa	35 – 45 mm Hg
pO_2	6.7 - 9.3 kPa	50 – 70 mm Hg
HCO_3	20 - 24 mmol/L	20 – 24 mEq/L
BE	+/- 5 mmol/L	± 5

pCO2 values up to 8.0 kPa are accepted in preterm infants with lung disease. A pCO2 elevation of 10 mmHg (1.33 kPa) decreases the pH by 0.08, and vice versa

Sarnat & Sarnat Staging (1976)

	Mild HIE (I)	Moderate HIE (II)	Severe HIE (III)
Level of consciousness	Hyperalert	Lethargic	Stuporose
Muscle tone	Normal	Mild hypotonia	Flaccid
Complex reflexes			
Suck	Normal/Weak	Weak/Absent	Absent
Moro	Strong	Weak/Incomplete	Absent
Seizures	Absent	Common	Frequent/difficult to control

Types of hypoxic ischaemic insult

- Chronic partial HI
  - More than one hour in duration, but can be days
  - Unlikely to be damaging if less than one hour
  - Both umbilical vessels acidotic (A&V)
  - Baby may require little resuscitation
  - Multi-organ dysfunction common
- Acute profound HI
  - Up to 25-30 minutes duration (may follow chronic partial)
  - First 10 minutes non-damaging
  - Fetal bradycardia seen if monitored
  - Discrepancy between acidosis in umbilical vessels (A>>V)
  - Baby often requires vigorous resuscitation
  - May affect the brain with little/no other organ involvement

Patterns of hypoxic ischaemic injury

- Chronic partial HI
  - white and grey matter in watershed distribution sometimes called peripheral, parasagittal, or a borderzone pattern
  - severe cases lead to cortical cysts (ulegyria)
- Acute profound HI
  - basal ganglia: putamen, globus pallidus and venterolateral thalamus
  - corticospinal tract from perirolandic cortex to posterior limb of internal capsule (PLIC)
  - Spread throughout the whole brain

Neutral Citation Number: [2023] EWHC 19 (KB)

QB-2019-004029

IN THE HIGH COURT OF JUSTICE
KING'S BENCH DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 11th January 2023

Before:

MR JUSTICE RITCHIE

- -

BETWEEN

CNZ
(SUING BY HER FATHER AND LITIGATION FRIEND MNZ)

Claimant

- and –

ROYAL BATH HOSPITALS NHS FOUNDATION TRUST (1)
THE SECRETARY OF STATE FOR HEALTH AND SOCIAL CARE (2)

Defendants

CNZ and Royal Bath Hospitals NHS Foundation Trust

- Retrospective Montgomery (2015 - 1996)
- Parental videorecording and contemporary written reflection proved crucial
- Lewis Rosenbloom's hypothesis of 5-minute aliquots of brain injury questioned
- Back calculation of acidosis not favoured by the causation experts

The Obstetrician & Gynaecologist 10.1576/toag.13.3.169.27669 http://onlinetog.org 2011;13:169–174

Review How long have we got to get the baby out? A review of the effects of acute and profound intrapartum hypoxia and ischaemia

Authors Janet Rennie / Lewis Rosenbloom

American Journal of Obstetrics and Gynecology

Founded in 1920

volume 187 *number* 1 JULY 2002

With a severe fetal bradycardia the BE can decrease by -1 mmol/L per 3 minutes

CLINICAL OPINION

Use of umbilical artery base excess: Algorithm for the timing of hypoxic injury

Michael G. Ross, MD, and Rageev Gala, MD

Neutral Citation Number: [2023] EWHC 19 (KB)

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The organs which have the highest metabolic needs are damaged first. The probable order of damage is as follows:

328.0 The deep grey matter first, including the Basal Ganglia, initially the Putamen and the Globus Pallidus then the Thalamus;

328.1 Then spreading into the cortex, the Pre and Post central Gyri.

328.2 Then spreading throughout the whole brain.

However the evidence did not prove that this progression is wholly sequential (one after the other) there is overlap (more and more structures being damaged contemporaneously). There was agreement that the damage is non linear. Each organ in the brain falls off a cliff at certain unknown times.

Therapeutic cooling criteria

Criteria A

Infants ≥ 36/40 with at least one of:

- Apgar ≤ 5 @ 10 mins after birth
- Continued need for assisted ventilation @ 10 mins after birth
- pH<7.00 within 60 mins of birth (umb/arterial/capillary)
- Base deficit ≥ 16 in umb/cap/venous/art blood sample within 60 mins of birth

Criteria B

- **Seizures**
- **Moderate to severe encephalopathy i.e.**
  - Altered state of consciousness (lethargy/stupor/coma)

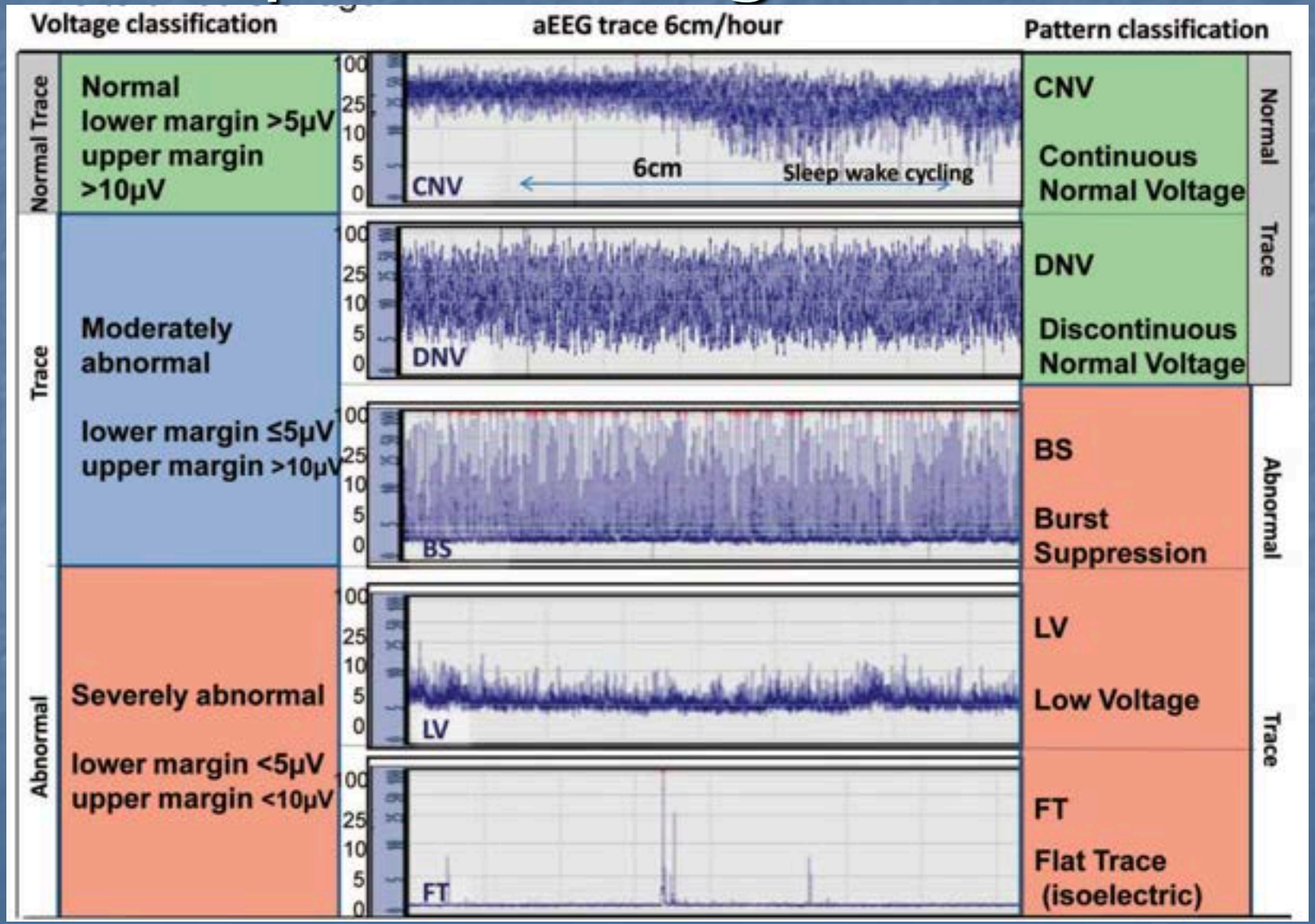
  AND
  - Abnormal tone (focal/generalised hypotonia/flaccid)
  - Abnormal primitive reflexes Suck and Moro (startle)

Gestational cut off now 35 weeks; occasionally used at 34 and 33 weeks
Can cause or exacerbate coagulopathy and persistent pulmonary hypertension

72

HOURS

Cerebral function monitoring (CFM)
Amplitude integrated EEG



Cochrane

Trusted evidence.
Informed decisions.
Better health.

Cooling: outcome

2013 meta-analysis

- Reduction in the combined outcome of mortality or major neurodevelopmental disability to 18 months of age, number needed to treat for an additional beneficial outcome (NNTB) 7 (95% CI 5 to 10)
- Reduction in mortality NNTB 11 (95% CI 8 to 25)
- Reduction in neurodevelopmental disability in survivors NNTB 8 (95% CI 5 to 14)

School-age outcomes of children without cerebral palsy cooled for neonatal hypoxic–ischaemic encephalopathy in 2008–2010

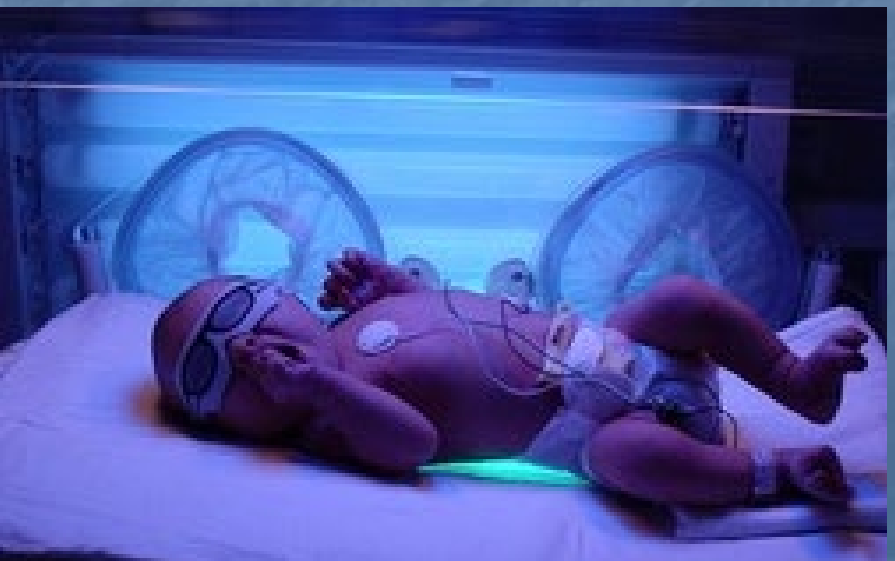
Richard Lee-Kelland,[1] Sally Jary,[1] James Tonks,[1,2] Frances M Cowan,[1,3] Marianne Thoresen,[1,4] Ela Chakkarapani[1]

- **Conclusions:** "School-age children without Cerebral Palsy cooled for HIE still have reduced cognitive and motor performance and more emotional difficulties than their peers."

Lee-Kelland R, *et al*. *Arch Dis Child Fetal Neonatal Ed* 2019;**0**:F1–F6.

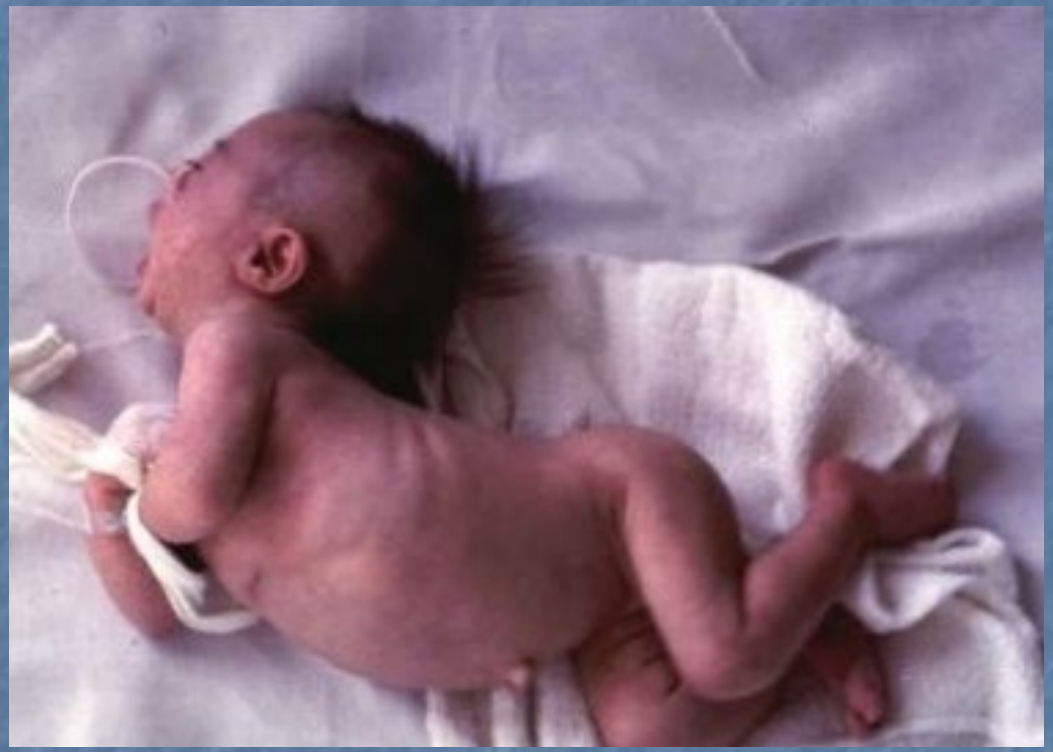
Neonatology: causation

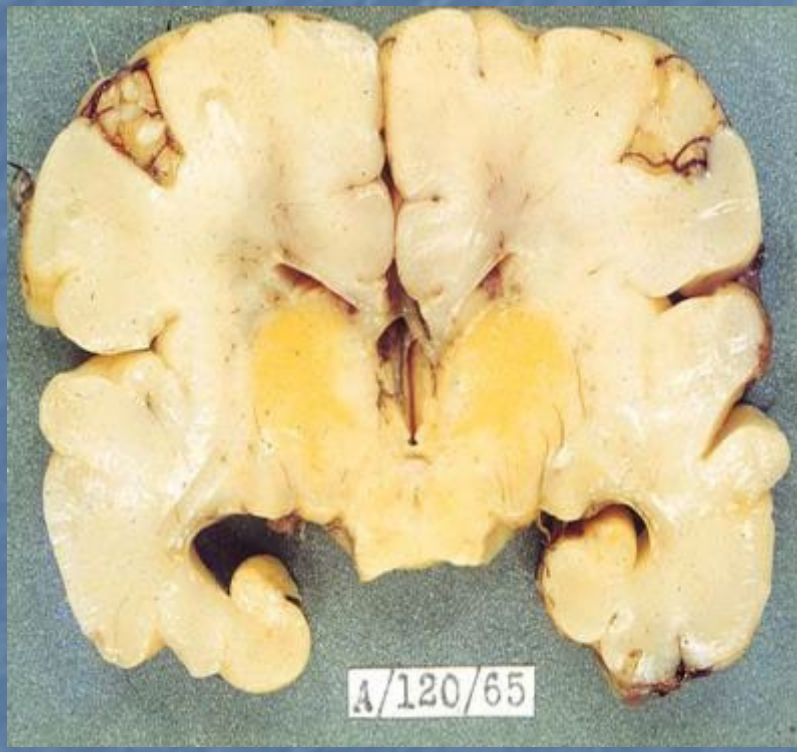
- Jaundice
- Kernicterus
- Deafness
- Biliary atresia



- Failure to detect significant jaundice
- Failure to treat with phototherapy or exchange transfusion
- Failure to diagnose biliary atresia

Kernicterus should be a ‘never event’





…..but has never gone away

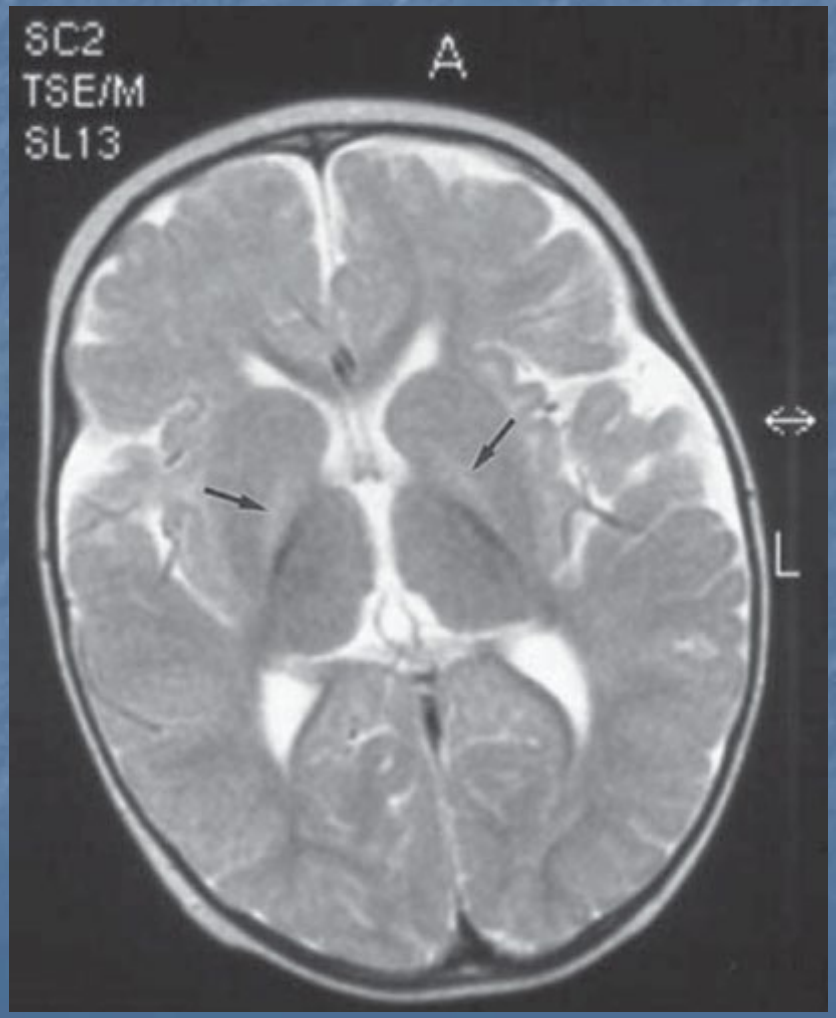
Bilirubin Neurotoxicity

- Acute bilirubin encephalopathy
  - *hypotonia/hypertonia*
  - *retrocollis, opisthotonus*
  - *drowsiness, poor feeding, fever*
  - *irritability, high pitched cry*
  - *apnoeas, seizures, coma*
- Can be asymptomatic

Bilirubin Neurotoxicity

- Chronic bilirubin encephalopathy or kernicterus
  - *dystonia, athetosis, CP*
  - *auditory neuropathy, deafness*
  - *auditory dyssynchrony*
  - *oculomotor paresis of upgaze*
  - *dental enamel dysplasia*
  - *cognitive abilities usually intact*

Neuroimaging: MRI Abnormalities



Globus pallidus (90%) (arrowed)

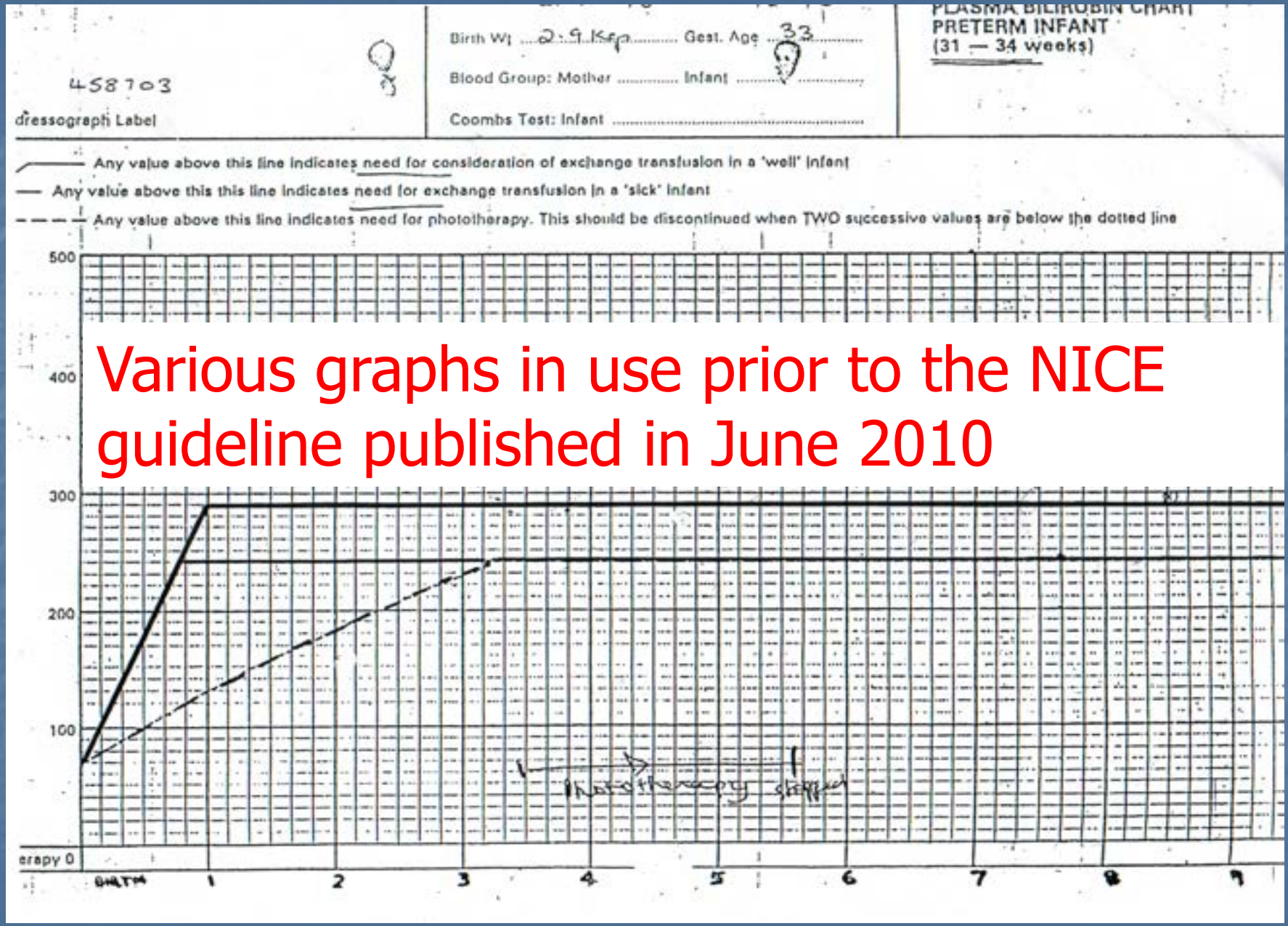
Subthalamic nucleus (40%)

Hippocampus (5%)

MRI can be normal

So, what goes wrong?

- Failure to identify the at-risk infant
- Failure to detect jaundice
- Failure to measure the bilirubin level
- Failure to adhere to local and NICE guidelines
- Failure to heed acute signs of neurotoxicity
- Failure to use phototherapy properly
- Failure to perform timely exchange transfusion
- Complications of exchange transfusion


458703
dressograph Label
Birth Wt 2.9 Kgs
Gest. Age 33
Blood Group: Mother
Infant
Coombs Test: Infant
PLASMA BILIRUBIN CHART
PRETERM INFANT
(31 — 34 weeks)
Any value above this line indicates need for consideration of exchange transfusion in a 'well' infant
Any value above this this line indicates need for exchange transfusion in a 'sick' infant
Any value above this line indicates need for phototherapy. This should be discontinued when TWO successive values are below the dotted line
500
400
300
200
100
Various graphs in use prior to the NICE guideline published in June 2010
BIRTH
1
2
3
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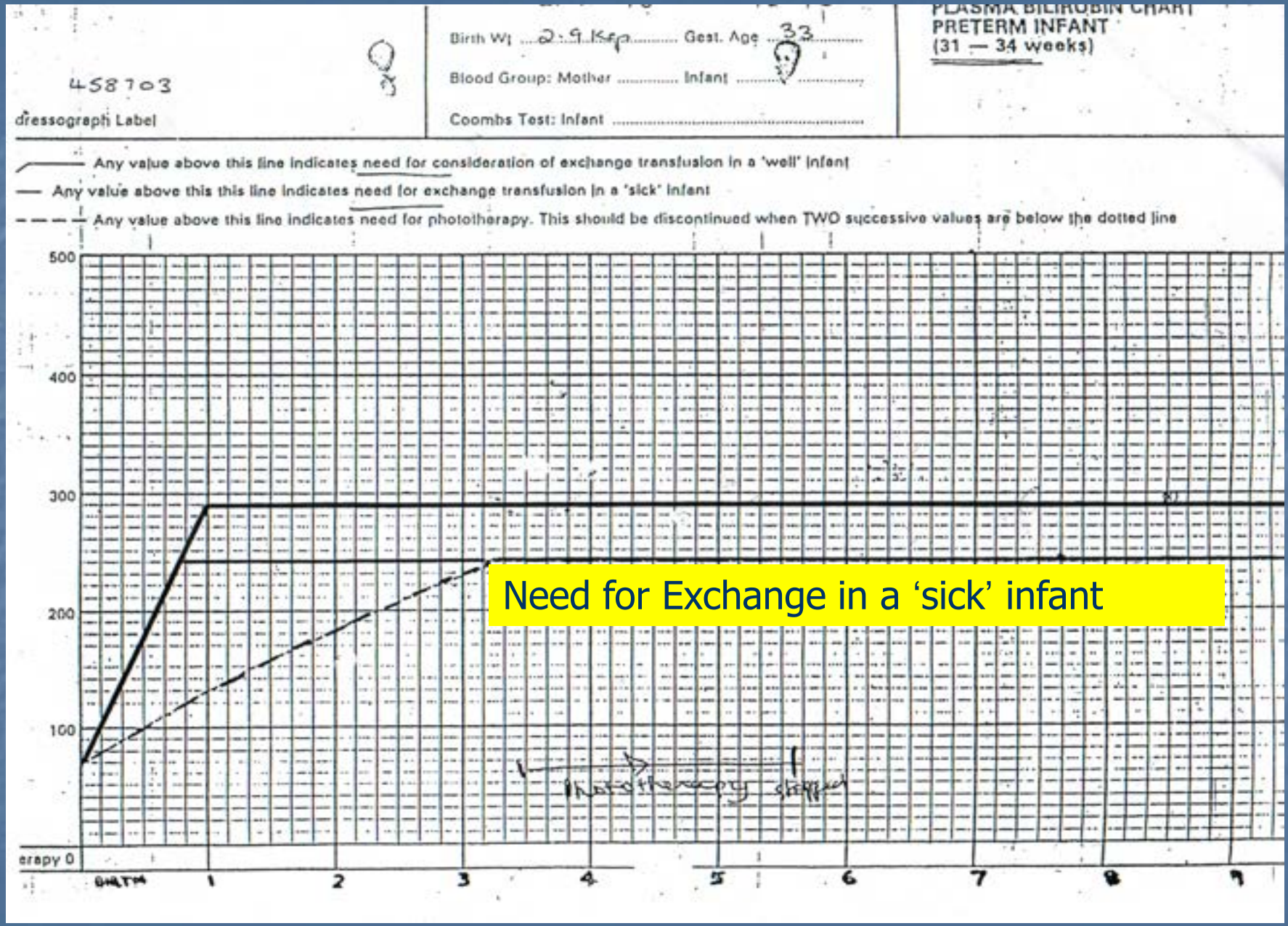
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400
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Need for Exchange in a 'sick' infant
BIRTH
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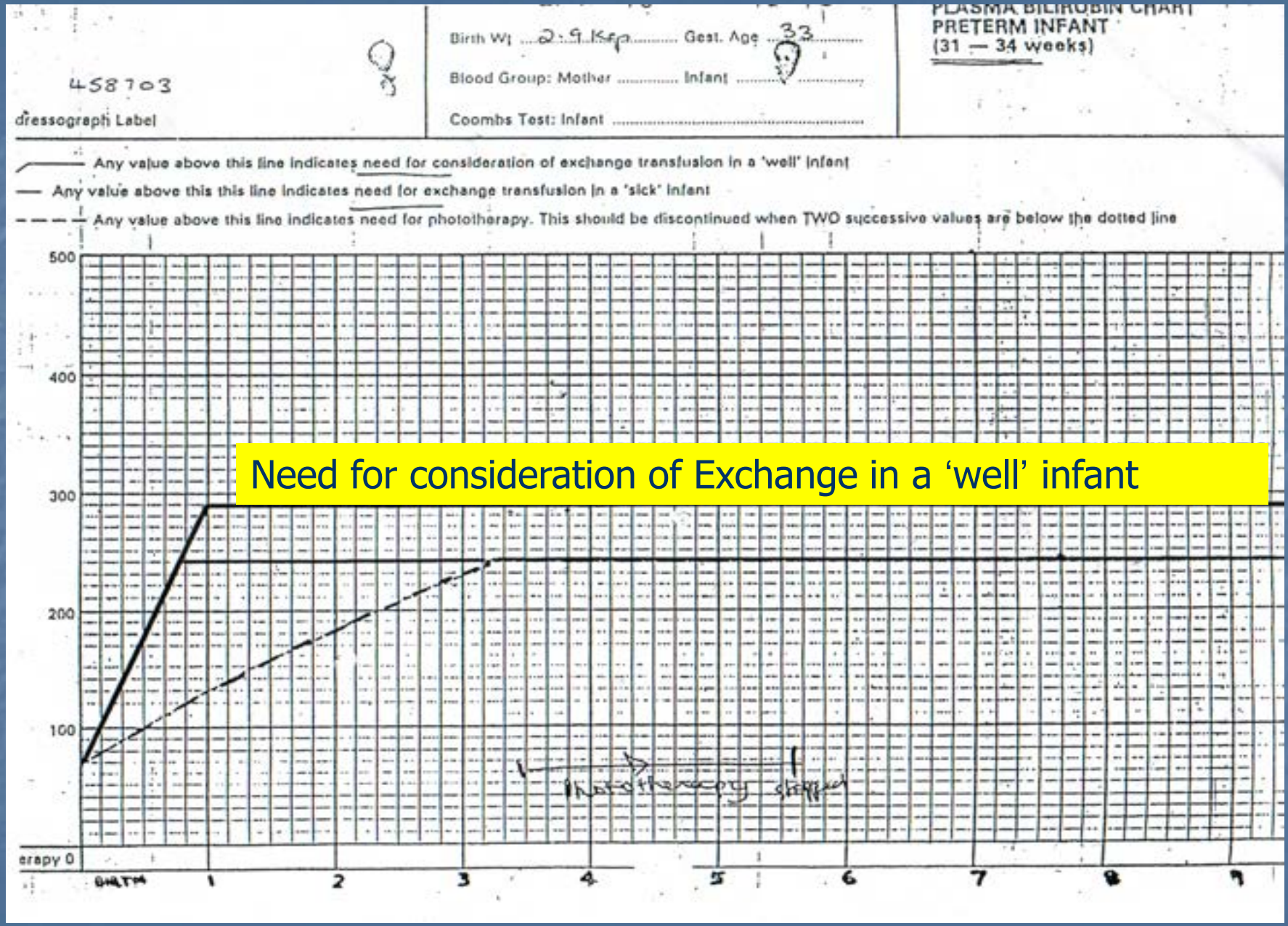
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500
400
300
200
100
Need for consideration of Exchange in a 'well' infant
Phototherapy started
erapy 0
BIRTH
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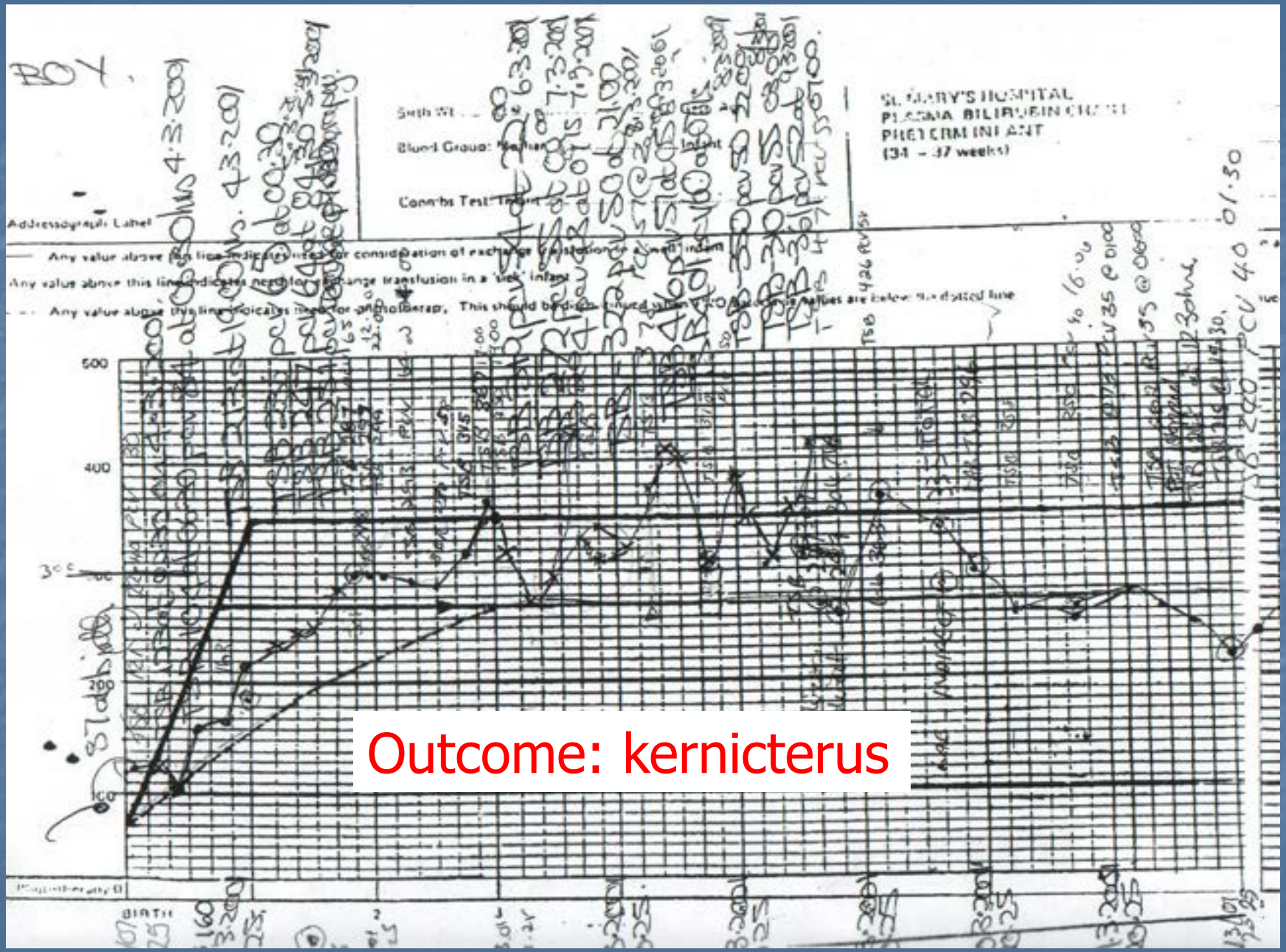
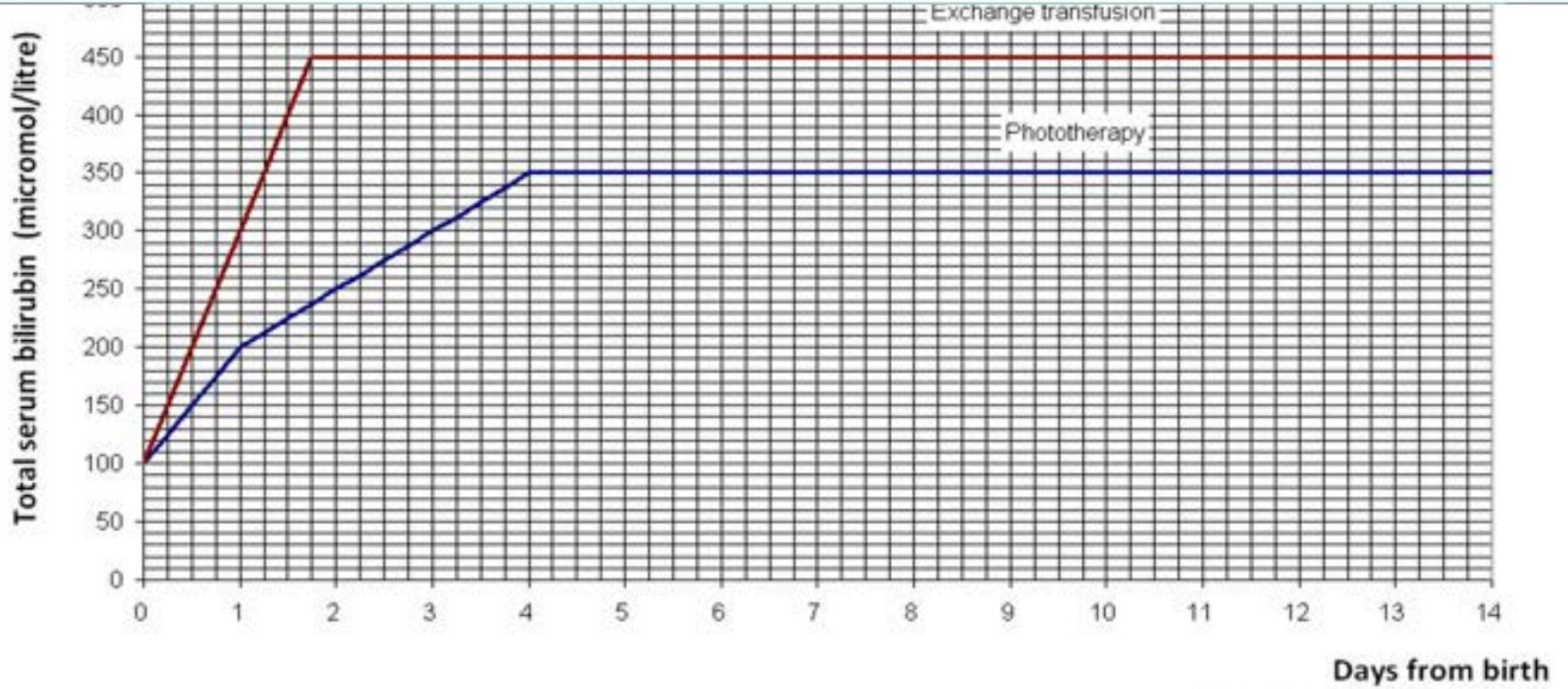


Outcome: kernicterus

Jaundice in newborn babies under 28 days

Clinical guideline [CG98] Published date: May 2010 Last updated: October 2016



Baby's blood group _____ Mother's blood group _____

NHS National Institute for Health and ClinicalExcellence

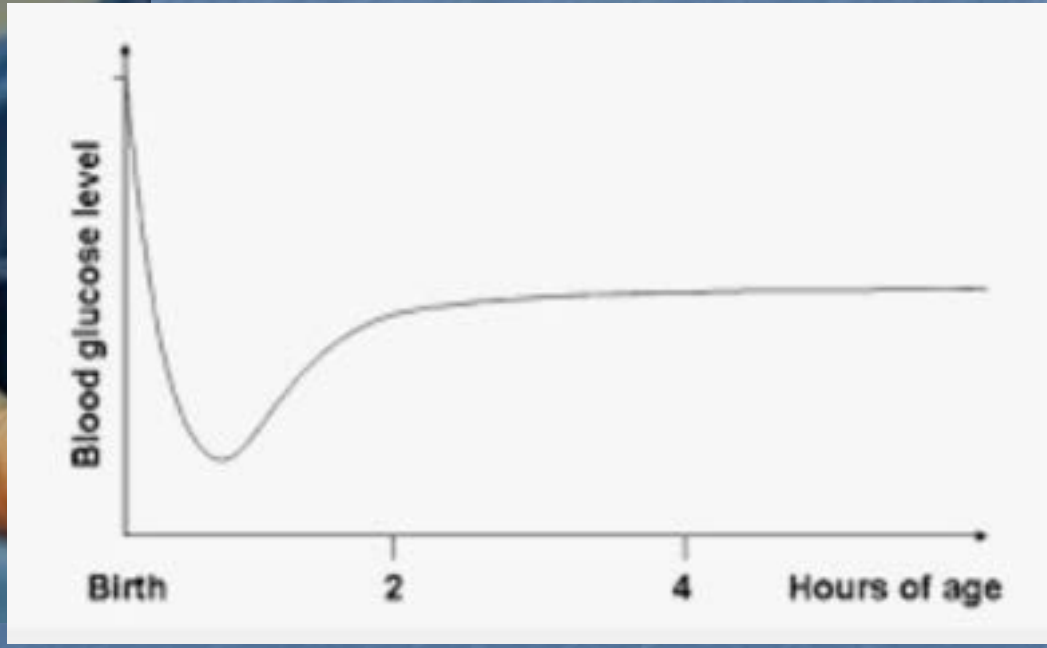
Neonatology: causation

Hypoglycaemia

Blood sugar < 2mmol/L

- Lack of monitoring for at risk groups
- Failure to recognise signs & symptoms
- Failure to treat promptly

Cutting the umbilical cord



Postnatal metabolic adaptation starts.....

Term infants at risk of impaired metabolic adaptation

1. *Infants of diabetic mothers*
2. *Infants whose mothers are on beta-blockers (impairs glycogenolysis)*
3. *Infants with intrauterine growth restriction: weight <2nd centile for gestation & sex*
4. *Clinically wasted or scrawny*

.....

Birth asphyxia, sepsis

Hyperinsulinaemia syndromes

Scrawny, despite > 2nd centile

Birth weight on 2nd centile / kg		
Gestational age / weeks	Boys	Girls
37	2.10	2.00
38	2.30	2.20
39	2.50	2.45
40	2.65	2.60
41	2.80	2.75
42	2.90	2.85

British Association of Perinatal Medicine

Identification and Management of Neonatal Hypoglycaemia in the Full Term Infant – A Framework for Practice

April 2017

Blood glucose to be measured if…

- Perinatal acidosis (cord arterial or infant pH <7.1 and base deficit ≥ -12mmol/l)
- Hypothermia (<36.5°C) not attributed to environmental factors
- Suspected / confirmed early onset sepsis
- Cyanosis
- Apnoea
- Altered level of consciousness
- Seizures
- Hypotonia
- Lethargy
- High pitched cry

Danger signs: not waking for feeds, or sucking effectively HAVING PREVIOUSLY FED WELL

……think hypoglycaemia and think SEPSIS

Original article

OPEN ACCESS

Neonatal hypoglycaemia: learning from claims

Jane M Hawdon,[1] Jeanette Beer,[2] Deborah Sharp,[3] Michele Upton,[4] On behalf of NHS Improvement Patient Safety Programme 'Reducing Term Admissions to Neonatal Units'

Hawdon JM, et al. Arch Dis Child Fetal Neonatal Ed 2016;0:F1–F6.

- *The NHS Litigation Authority (NHS LA): injury secondary to neonatal hypoglycaemia, between 2002 and 2011.*
- *25 claims for which damages were paid, with a total financial cost of claims to the NHS of £162,166,677*
- *Individual claims, inclusive of costs, were £2.5m–£12.6m, median £6.3m*
- *ALL potentially avoidable. Thematic failures*

Seminars in Fetal & Neonatal Medicine (2005) 10, 363–368

Seminars in
FETAL & NEONATAL MEDICINE

www.elsevierhealth.com/journals/siny

Neonatal hypoglycaemia: Clinical and legal aspects

A.F. Williams

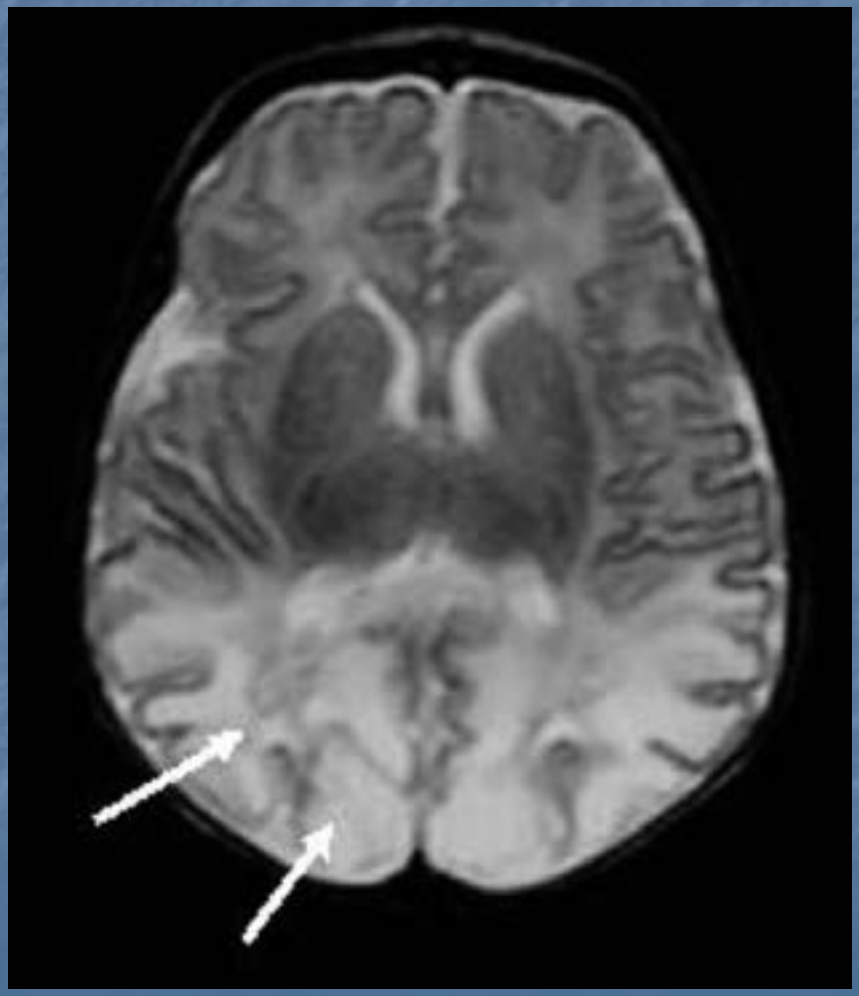
Whipple's triad

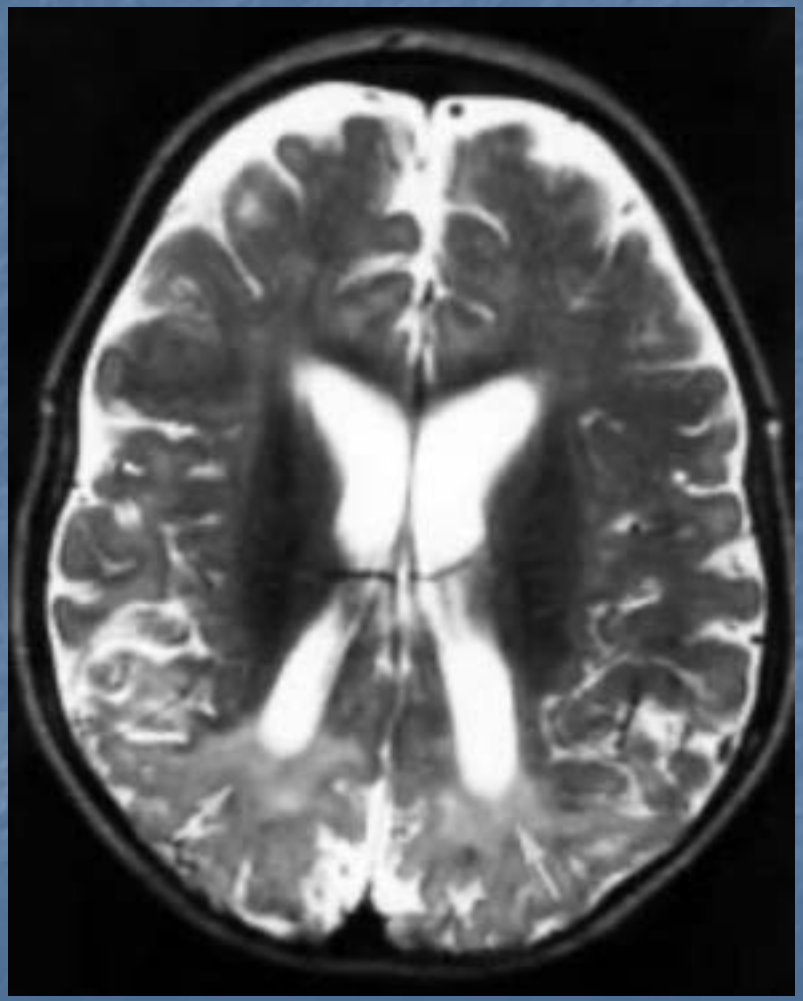
1. clinical features are typical of hypoglycaemia, *and*
2. a low plasma glucose concentration is documented concurrently using accurate and precise methods (q.v.), *and*
3. clinical features resolve within minutes to hours once normoglycaemia is regained unless brain injury has already occurred (q.v.).

Link to causation

- Whipple's triad is satisfied, demonstrating that neurological abnormalities noted in the neonatal period were on the balance of probability attributable to hypoglycaemia, *AND* that
- the pattern of brain injury revealed by diagnostic imaging is characteristic of that described in the neuropathological literature cited above.

Hypoglycaemia, causation: characteristic posterior parietal and occipital lobe injury





PEDIATRICS®

OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

CONCLUSIONS. Patterns of injury associated with symptomatic neonatal hypoglycemia were more varied than described previously. White matter injury was not confined to the posterior regions; hemorrhage, middle cerebral artery infarction, and basal ganglia/thalamic abnormalities were seen, and cortical involvement was common.

Patterns of Cerebral Injury and Neurodevelopmental Outcomes After Symptomatic Neonatal Hypoglycemia

Charlotte M. Burns, Mary A. Rutherford, James P. Boardman and Frances M. Cowan

Pediatrics 2008;122;65
DOI: 10.1542/peds.2007-2822

35 cases: congenital infection, developmental brain abnormalities and hypoxic ischaemic encephalopathy ‘had been excluded’. Early brain MRI showed general white matter injury (94%; severe in 43%), basal ganglia and thalamic injury (40%). Posterior white matter damage seen in 29%.

Causation: exacerbating insults

THE JOURNAL OF PEDIATRICS • www.jpeds.com

July 2012

ORIGINAL ARTICLES

Hypoglycemia is Associated with Increased Risk for Brain Injury and Adverse Neurodevelopmental Outcome in Neonates at Risk for Encephalopathy

Emily W. Y. Tam, MDCM, MAS, FRCPC[1,2,*], Laurel A. Haeusslein, BA[1,*], Sonia L. Bonifacio, MD[2], Hannah C. Glass, MDCM, MAS[1,2], Elizabeth E. Rogers, MD[2], Rita J. Jeremy, PhD[4], A. James Barkovich, MD[1,2,3], and Donna M. Ferriero, MD, MS[1]

> JAMA. 2003 Nov 26;290(20):2677-84. doi: 10.1001/jama.290.20.2677.

Chorioamnionitis and cerebral palsy in term and near-term infants

Yvonne W Wu [1], Gabriel J Escobar, Judith K Grether, Lisa A Croen, John D Greene, Thomas B Newman

Learning from claims

Original article

OPEN ACCESS

Neonatal hypoglycaemia: learning from claims

Jane M Hawdon,[1] Jeanette Beer,[2] Deborah Sharp,[3] Michele Upton,[4] On behalf of NHS Improvement Patient Safety Programme 'Reducing Term Admissions to Neonatal Units'

Hawdon JM, et al. Arch Dis Child Fetal Neonatal Ed 2016;0:F1–F6.

Original article

OPEN ACCESS

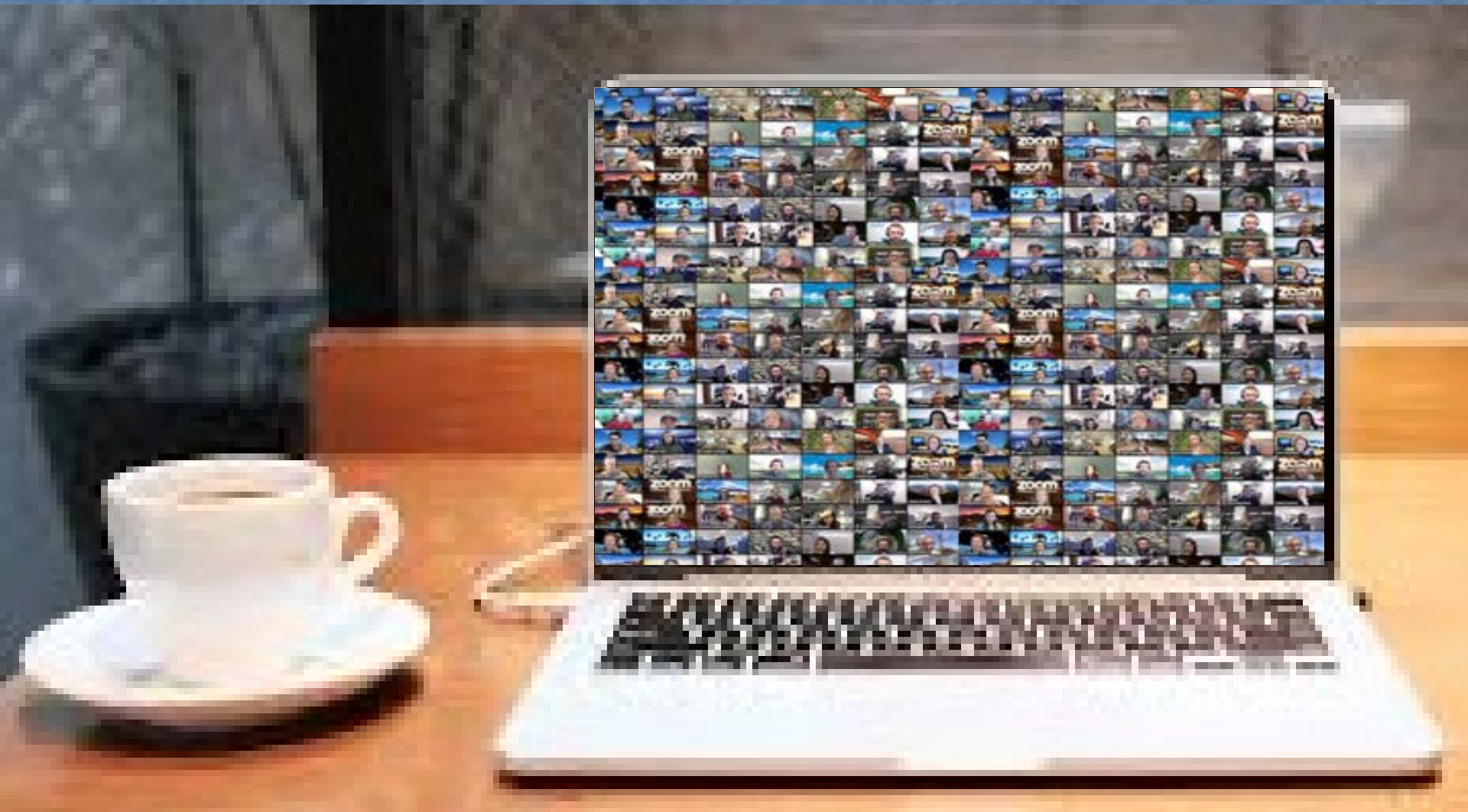
Learning from claims: hyperbilirubinaemia and kernicterus

Janet M Rennie,[1] Jeanette Beer,[2] Michele Upton[3]

Arch Dis Child Fetal Neonatal Ed 2018;**0**:F1–F3

Mutual respect and Learning

Thank you for your attention



What is pH?

- **pH** = pK + log (HCO3/H2CO3) (Henderson-Hasselbach eqn) pK= constant, it is the pH value at which H2CO3 is 50% dissociated i.e. concentration of HCO3- and carbonic acid in body are equal. PK=6.1 for H2CO3.
- Normal ratio HCO3/H2CO3 = 20/1 and hence
- pH = 6.1 + log 20
  = 6.1 + 1.3 = 7.4 pH Normal 7.35-7.45
- 7.4 $\pm$ 2 S.D. Alkalosis >7.5 Acidosis < 7.3
- Severe acidosis <7.2

What is base excess?

- **Base excess (BE)** is calculated from blood pH and PaCO2
- **Base excess** refers to variance (above or below) the total buffer base, which is made up of bicarbonate (50%), haemoglobin (25%) and a combination of proteins, phosphate and sulphate (25%).
- Normal buffer base is a value of around 48mmol/L

Examples:

- If buffer base is 30 it means that the buffer base is reduced by 18mmol/L, BE is -18 (this may also be called a positive base deficit {BD}).
- If buffer base is 60 it means that the buffer base is increased by 12 mmol/L, BE is +12 (this may also be called a negative base deficit {BD}).

12
King's Bench Walk

Causation in Birth Injury claims: *CNZ v Royal Bath Hospitals NHS Foundation Trust*

Speakers:

John-Paul Swoboda

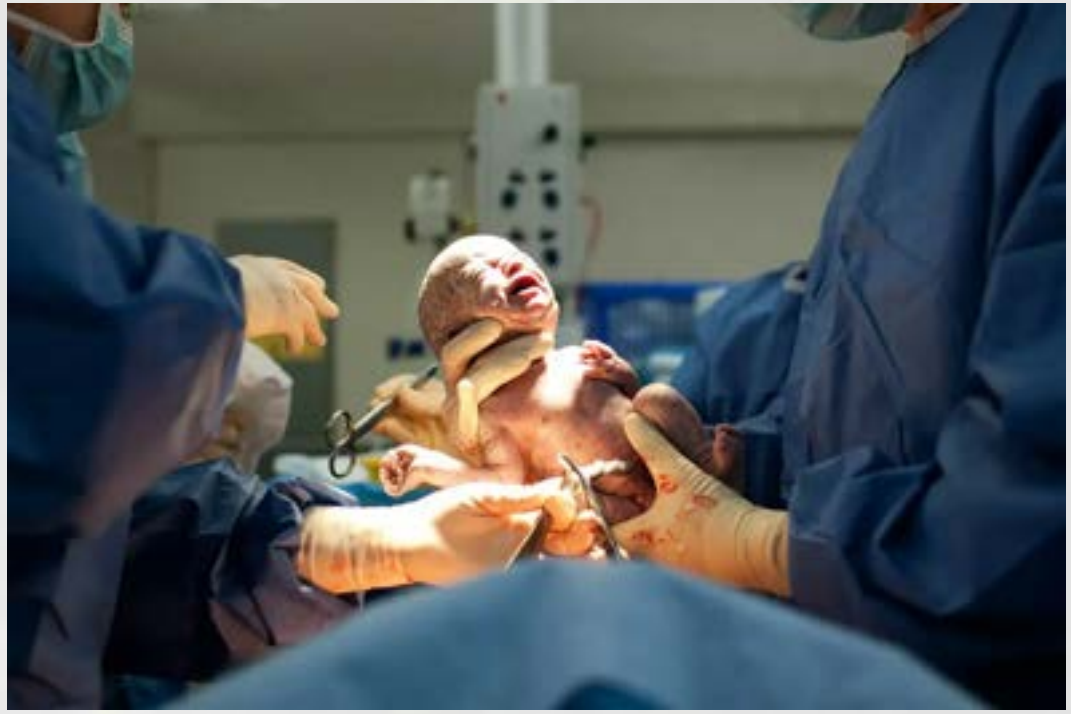
swoboda@12kbw.co.uk

Thea Wilson

wilson@12kbw.co.uk

The Facts

- C was born in 1996.
- C suffered from quadriplegic cerebral palsy caused by hypoxic brain injury immediately prior to and following birth.
- Her twin was born vaginally approximately 1 hour before her birth by C-Section.
- C was born in poor condition and it took approximately 3 minutes 30 seconds for her heartrate to be restored post-birth.
- After C reached adulthood, expressed a desire to bring a claim and her father acted as her litigation friend.



The Proceedings

April 2018	Letter of claim (draft Particulars of Claim)
June 2019	Letter of Response
November 2019	Issue
March 2020	Service
September 2020	Defence
May 2022	First trial (adjourned with amendment of pleadings)
December 2022	Trial
January 2023	Judgment

The Issues

- Antenatal issues:
  - Did M request a caesarian antenatally?
  - Was M offered an elective caesarian antenatally?
  - If not, should M have been offered an elective caesarian?
  - Did M agree to vaginal birth having had the pros and cons properly discussed with her?
  - Was M's right to make an informed choice breached?
- Labour issues:
  - Did the obstetricians fail to grant the parents' choice for a caesarian?
  - Did the obstetricians negligently fail to perform the caesarian quickly enough?

Factual Findings

- Ritchie J held in respect of the antenatal issues:
  - CS was a reasonable treatment option in 1996, even though it would not have been recommended by obstetricians in these circumstances other than as a fall back.
  - M was properly counselled against CS and accepted that counselling.
  - Antenatally, M wanted a natural child birth with minimal instrumental involvement.

Factual Findings (cont)

- Ritchie J held in respect of the labour issues:
  - M entered hospital for a properly planned and chosen induction of labour.
  - Twin 1 was born vaginally at 00:01 and CS was not requested prior to her birth.
  - The CTG suggested C was doing reasonably well until 00:40, but there were three clear decelerations. The first two were of equivocal significance, but the last at 00:16-17 was un-reassuring.
  - The normal, aimed-for, birth window between two twins was 30 minutes or less, after which it was necessary to make plans with urgency to deliver the baby.
  - M's treatment was reasonable up to 00:25.
  - After 00:25, there were two options for artificial rupture of membrane a low ARM was easy, but a high one was risky; M probably could not have an epidural and did not want a rupture of membranes; Urgent transfer to theatre was the only safe option for C and M.
  - Following this, CS under general anaesthetic was the best reasonable treatment unless the baby had descended during transfer.

Findings on Breach

- Antenatal process:
  - The consent process for the birth plan was not unreasonable or a breach of duty

- Labour:
  - The obstetrician failed to properly discuss the options with the parents or confirm their choice when she should have.
  - From 00:25, and particularly from 00:40, there was a negligent delay in M's treatment.
  - The obstetrician failed to inform her Consultant when she discussed the case that: epidural had been attempted and had failed; M did not want ARM; and the parents had already chosen CS. The Consultant's recommendation to try ARM in theatre was therefore not adequately informed.
  - The consent for ARM in theatre was not properly obtained.
  - There were negligent delays in: setting off for theatre; transfer to theatre; the induction of anaesthetic; and the performance of the CS.

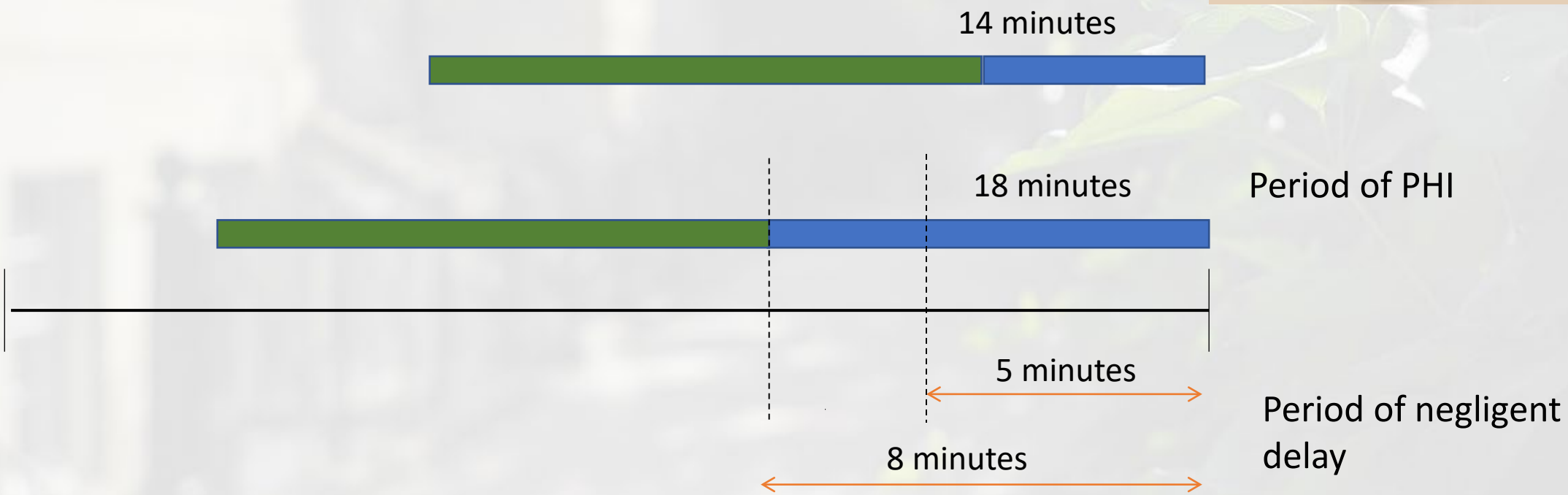
Factual Causation

- But for the negligence, there would have been a full discussion of options with the parents at 00:26 and their views obtained.
- A plan to take to theatre and discussion with the consultant should have followed.
- M should have been taken to theatre earlier than she was.
- The CS should have taken 6-10 rather than 13 minutes.

- C should have been delivered by no later than between 00:55 and 00:58 and resuscitated in 1-2 minutes, rather than born at 01:03 and resuscitated at 01:06.

- C suffered bradycardia starting 00:48 to 00:52 and continuing until resuscitation.

Factual Causation (cont)



Legal Causation

- C won on ‘but for’ test
  - By 30 secs
  - 16 mins bradycardia
  - 6 mins irreversible PHI
  - 6.5 mins (range of 5 to 8) of neg delay

- Alternatively: functional outcome of CP is indivisible
  - Apportionment not appropriate
  - C still recovers 100% damages

Legal Causation

- BUT
  - Permission granted by trial judge to appeal on one ground
  - Permission sought by D

- NOT end of this story

Legal Causation

- A reminder of whether injury divisible/ indivisible

- Is injury 'triggered'
  - Malaria (single trigger); cancer (threshold trigger)
  - Indivisible injuries
  - 100% damages

- OR dose related
  - NIHL; asbestosis
  - Divisible injuries
  - Aliquot of damages apportioned to negligent dose

Legal Causation

- So is brain injury caused by PHI a divisible injury?
  - Yes according to Ritchie J (para 361)
  - Brain damage is *'wholly dose dependent'*
  - BUT what about if the function outcome is indivisible
    - Impossible to determine functional outcome in CP cases on basis of extent of brain injury?

Legal Causation

- The normal approach in divisible injury cases

- *Thompson v Smiths* [1984] Q.B. 405
- *"If we know … that a substantial part of the impairment took place before the defendants were in breach why in fairness should they be made to pay for it? The fact the precise quantification is impossible should not alter the position"*

Legal Causation

- So in CNZ was
  - any quantification impossible (full damages)
  - Precise quantification impossible (apportion damages)

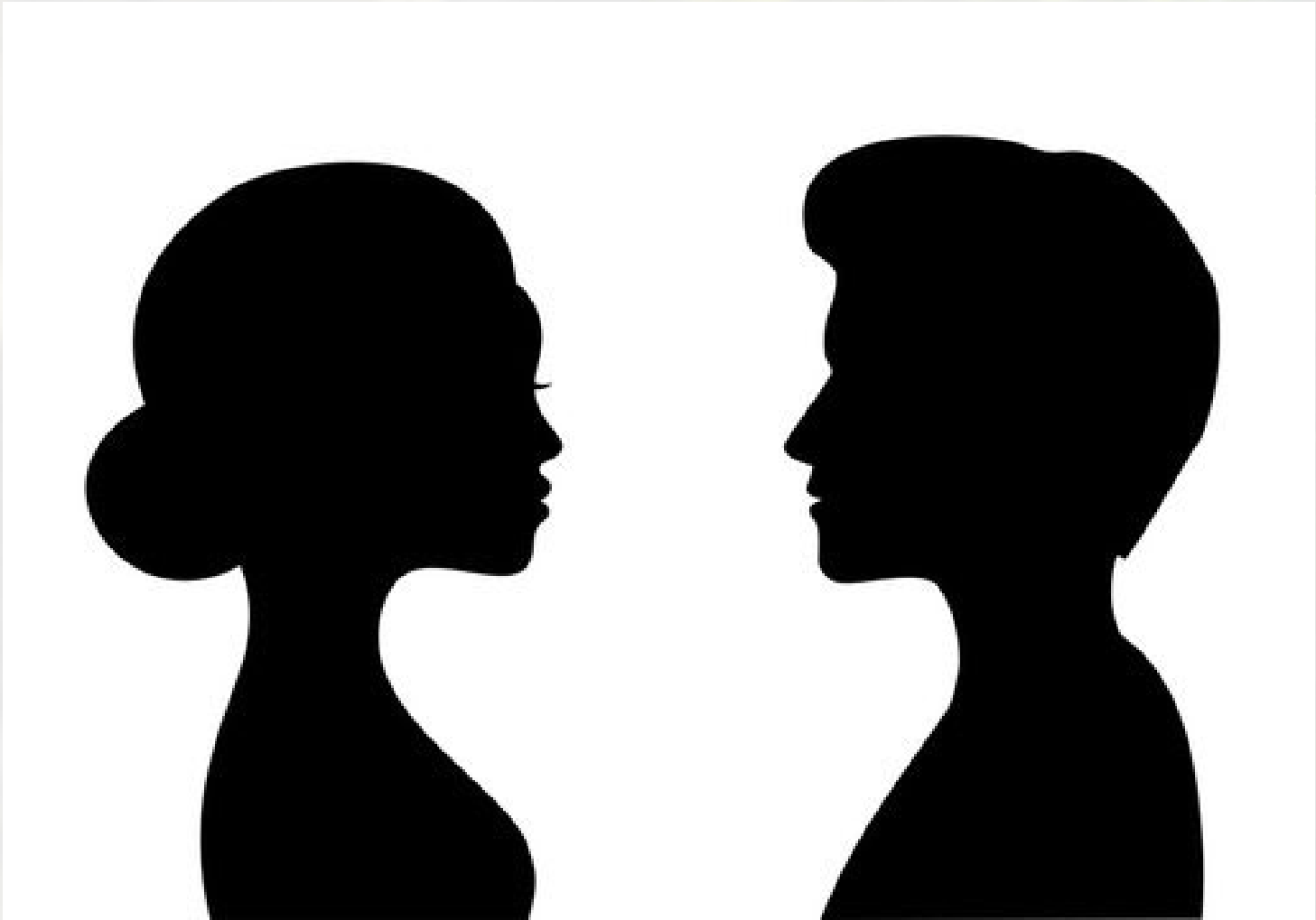
- Dr Rosenbloom (paediatric neurologist for D)
  - Argued rough and ready apportionment was possible
  - 5 min chunks (mild, moderate, severe)

- BUT rejected by Ritchie J

Legal Causation

- What will happen on appeal???
- All to play for
- Despite winning at 1st instance
  - Finding HPI divisible makes C vulnerable
  - Rosenbloom model not perfect but doesn't need to be
- D needs to knock 30 secs of neg delay
  - *Montgomery* to be applied in 1996 (NB Before GMC guidance in 1998)

Discussion



Questions?

Thank You