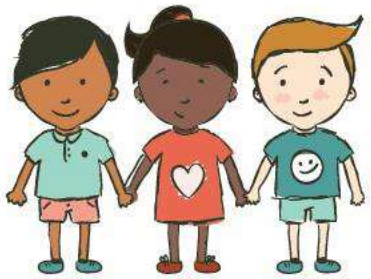


UPdate 2016
PAEDIATRIC ANNUAL CONFERENCE
UNIVERSITY OF PRETORIA
DEPARTMENT OF PAEDIATRICS
4 & 5 MARCH 2016
PROTEA HOTEL FIRE & ICE · MENLYN · PRETORIA

BIRTH ASPHYXIA and The "New" Consensus Statement

Keith Bolton

Rahima Moosa Mother & Child Hospital

THE HERD IS UNDER THREAT

HPCSA CIVIL COURTS CRIMINAL COURTS

Background

- The child with cerebral palsy, intellectual impairment and epilepsy naturally generates huge sympathy from society and the courts.
- Apart from “poor care”, the capping of the RAF, the introduction of “contingency fees” and the empowerment of the people have all resulted in a massive increase in litigation…especially for birth asphyxia!
- There is a Tsunami of cases in the courts especially in Gauteng, KZN & the Eastern Cape!

The Size of the Problem!

- A world-wide problem.
- In RSA, as with all other financial matters; Gauteng (North & South High Courts) lead the way in attracting litigation.
- South Gauteng has approximately 1000 cases pending against them – 80% are CP cases.
- The current “quantum” in these cases is between R10-30,000,000
- GHD are losing 90% of cases
- This means R10.8 billion in payouts!
- Private obstetricians and paediatricians not exempt!

‘It has never been safer to have a baby and never been more dangerous to be an obstetrician[1]’
(or paediatrician/neonatologist[2])

1. MacLennan A, Nelson KB, Hankins G, Speer M. Who will deliver our grandchildren? Implications of cerebral palsy litigation. JAMA 2005;294(13):1688-1690.
2. Bolton K. Personal opinion

Cerebral Palsy

- Was there someone to blame?

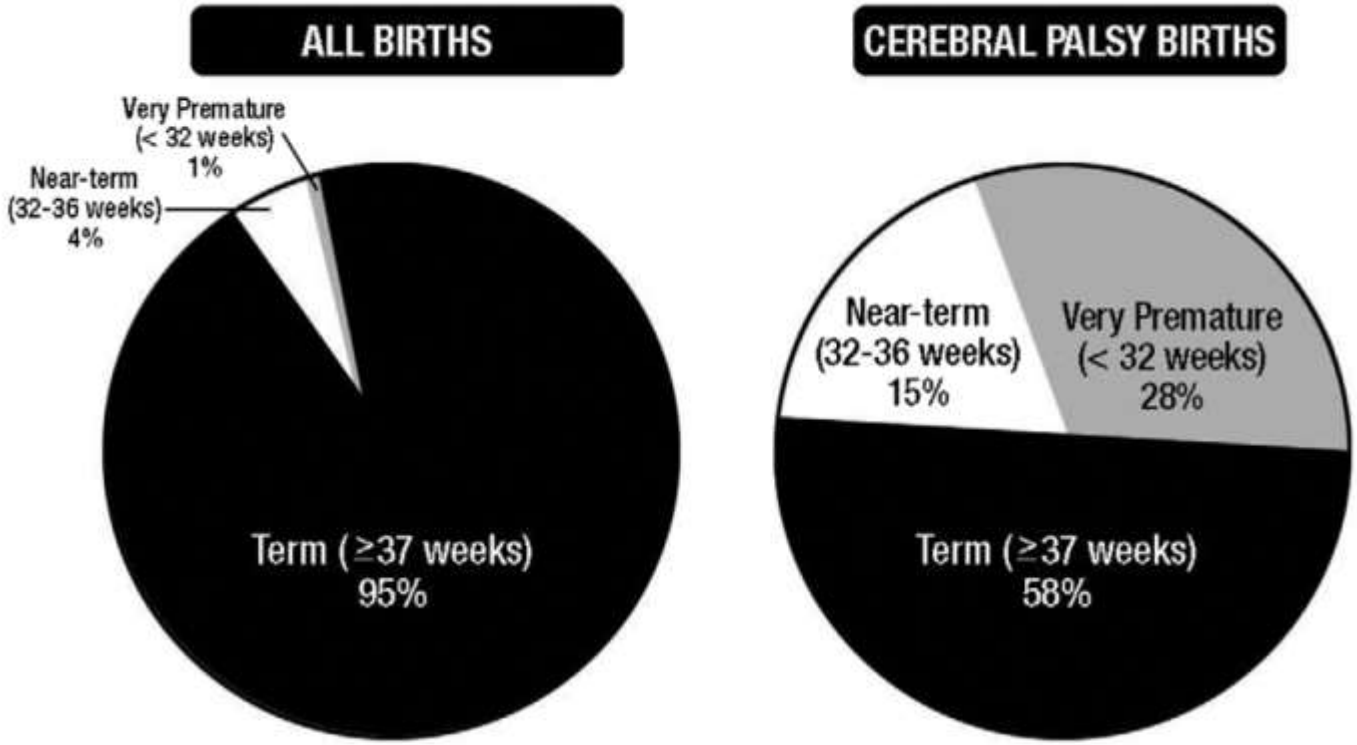
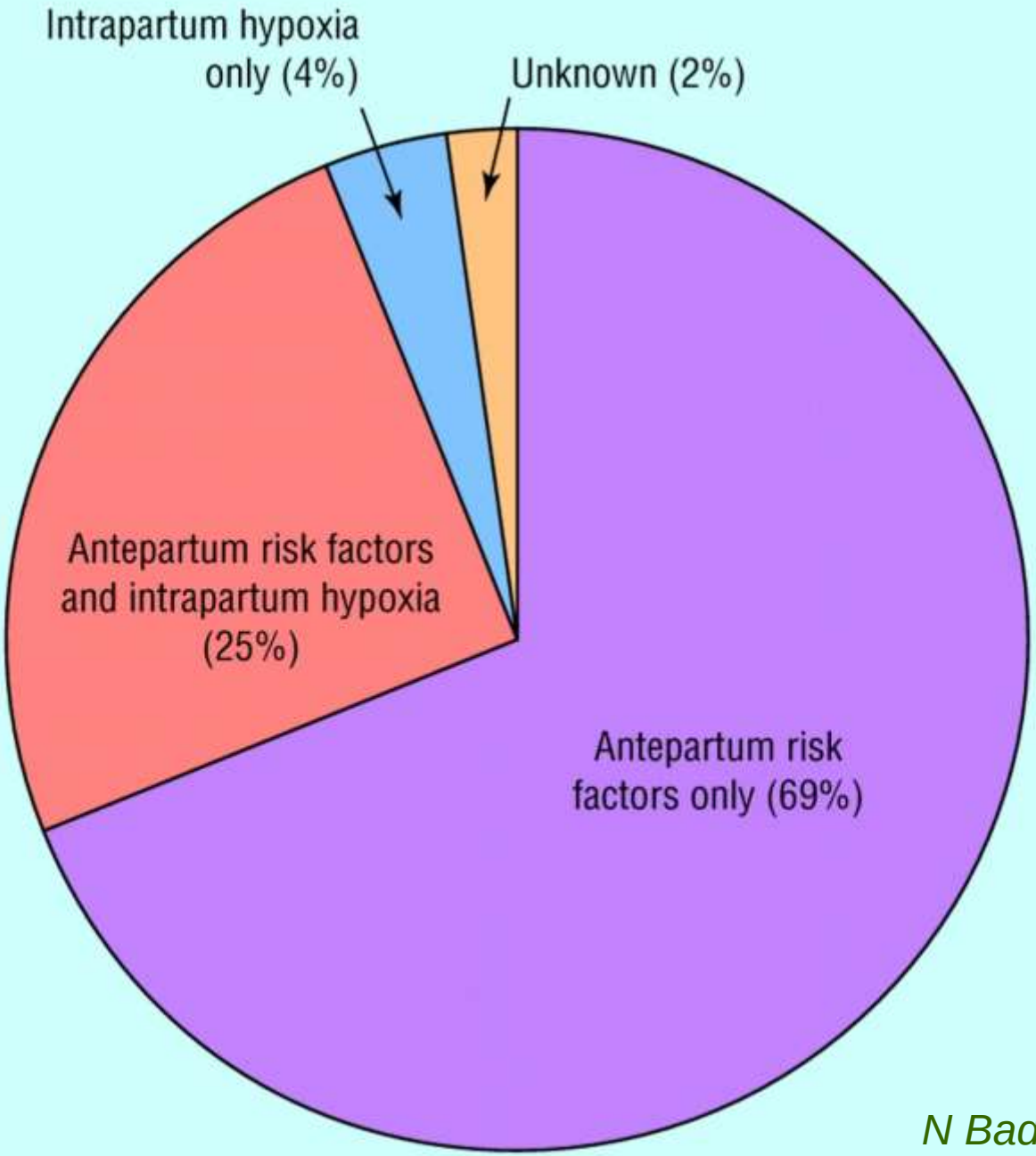


FIGURE 1. Proportion of cerebral palsy and all births according to gestational age.

Clark SM et al Antenatal antecedents and the impact of obstetric care in the etiology of cerebral palsy *Clin Obstet Gynecol* 2008; 51(4):



N Badawi et al
BMJ 1998;317:1554

The Causes for Cerebral Palsy (Developed Communities)

TABLE 1. Estimates of Proportion of Cerebral Palsy in Term and Near-term Infants Attributed to Major Causes in Population-based Studies

Neuroimaging based[3]	
Perinatal ischemic stroke	22%
Congenital malformation	15
White matter disorder	12
Hypoxia-ischemia	5
Clinical studies	
Intrauterine exposure to inflammation[3,4]	11%-12%
Birth asphyxia[5]	6
Complications of multiple birth[6]	5

Nelson KB. Causative factors in cerebral palsy. *Clin Obstets Gynec* 2008; 51(4): 749-62

The Causes for Cerebral Palsy (Developed Communities)

Relationship between intrapartum asphyxia and cerebral palsy: Term infants

Country	Years of Birth	% related to asphyxia
USA	1959-66	12%
Australia	1975-80	17%
Finland	1978-82	24%
Ireland	1981-1983	23%
England	1984-87	17%
Sweden	1987-90	17%
Sweden	1991-94	24%

Volpe JJ. Neurology of the Newborn 5th Edition, 2008; ISBN: 978-1-4160-3995-2

The Causes for Cerebral Palsy (Less Developed Communities)

TABLE I. CLINICAL SPECTRUM OF CEREBRAL PALSY

Developed countries	Developing countries
Most cases of cerebral palsy attributable to events before labour[2,13]	Higher prevalence of postneonatal-acquired cerebral palsy cases[7] CNS infections [6,7] +++ Bilirubin toxicity [4,6] ++
Intrapartum asphyxia less than 10%[2]	Birth asphyxia [14,15] ++++
Spastic diplegia and hemiplegia the predominant types [1]	Spastic quadriplegia the predominant type [4,6]

Van Toorn R et al. Aetiology of cerebral palsy in children presenting at Tygerberg Hospital *SAJ Child Health* 2007; 1(2): 74-77

The Causes for Cerebral Palsy (Less Developed Communities)

TABLE III. ORIGIN OF INSULT CAUSING CEREBRAL PALSY

	N (%)
Antenatal period	70 (28.9)
Perinatal period	92 (38.0)
Acquired	51 (21.1)
Undetermined	29 (12.0)
Total	242 (100)

Van Toorn R et al. Aetiology of cerebral palsy in children presenting at Tygerberg Hospital *SAJ Child Health* 2007; 1(2): 74-77

The First Consensus Statement 1999

A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement

Alastair MacLennan for the International Cerebral Palsy Task Force*

BMJ 1999;319:1054–9

Box 1—Supporters of the consensus statement

American College of Obstetricians and Gynecologists
American Gynecological and Obstetrical Society
Australian College of Midwives
Hong Kong Society of Neonatal Medicine
Institute of Obstetrics and Gynaecology of the Royal College of Physicians of Ireland
International Society of Perinatal Obstetricians
New Zealand College of Midwives
Paediatric Society of New Zealand
Perinatal Society of Australia and New Zealand
Royal Australasian College of Physicians, Paediatric Division
Royal Australian College of General Practitioners
Royal Australian College of Obstetricians and Gynaecologists
Royal College of Obstetricians and Gynaecologists
Royal College of Pathologists of Australasia
Royal New Zealand College of Obstetricians and Gynaecologists
Society of Obstetricians and Gynaecologists of Canada

Box 2—Criteria to define an acute intrapartum hypoxic event

Essential criteria

1 Evidence of a metabolic acidosis in intrapartum fetal, umbilical arterial cord, or very early neonatal blood samples (pH < 7.00 and base deficit ≥ 12 mmol/l)
2 Early onset of severe or moderate neonatal encephalopathy in infants of ≥ 34 weeks' gestation
3 Cerebral palsy of the spastic quadriplegic or dyskinetic type

Criteria that together suggest an intrapartum timing but by themselves are non-specific

4 A sentinel (signal) hypoxic event occurring immediately before or during labour
5 A sudden, rapid, and sustained deterioration of the fetal heart rate pattern usually after the hypoxic sentinel event where the pattern was previously normal
6 Apgar scores of 0-6 for longer than 5 minutes
7 Early evidence of multisystem involvement
8 Early imaging evidence of acute cerebral abnormality

Neonatal Encephalopathy 1

Definition

Neonatal encephalopathy is a clinically defined syndrome of disturbed neurologic function in the earliest days of life in an infant born at, or beyond 35 weeks of gestation, manifested by a subnormal level of consciousness or seizures, and often accompanied by difficulty with initiating and maintaining respiration and depression of tone and reflexes.

Neonatal Encephalopathy 2

"If an intrapartum insult has caused permanent brain damage in an infant of more than 34 weeks' gestation there will be abnormalities of behaviour in the neonatal period, usually of at least moderate severity and noted within 24 hours of delivery. However, *moderate to severe encephalopathy after a non-reassuring intrapartum cardiotocograph is very uncommo*n, occurring in around 7 per 1000 such births—just twice the rate in the background population. Conversely, *many cases of severe neonatal encephalopathy are not associated with intrapartum hypoxaemia.* Cerebral palsy associated with intrapartum events in infants born beyond 34 weeks' gestation is only rarely an outcome associated with milder grades of encephalopathy. Infants with severe encephalopathy frequently have an adverse outcome."

BMJ 1999;319:1054–9

Sarnat & Sarnat staging (1976)

	Stage 1	Stage 2	Stage 3
Consciousness	hyperalert	Lethargic of obtunded	Stupor or coma
Activity	Normal	Decreased	Absent
Neuromuscular control			
Muscle tone	Normal	Mild hypotonia	Flaccid
Posture	Mild distal flexion	Strong distal flexion	Intermittent decerebration
Strech reflexes	Overactive	Overactive	Decreased or absent
Primitive reflexes			
Suck	Weak	Weak or absent	Absent
Moro	Strong	Weak, incomplete	Absent
Tonic neck	Slight	Strong	Absent
Autonomic function			
Pupils	Normal	Miosis	Mydriase or variable, unequal
Heart rate	Tachycardia	Bradycardia	Variable
Seizures	None	Common	Uncommon

Stage 0 = Normal

Sarnat HB & Sarnat MS. Neonatal encephalopathy following fetal distress. A clinical and electroencephalographic study. *Arch Neurol.* 1976 Oct;33(10):696-705

Neonatal Encephalopathy & Outcome

Table 2. Classifying the Degree of Encephalopathy to Establish the "Pretest" Probability of Poor Outcome

Grade	Mental Status	Need for Ventilator	Feeding Problems	Tone	Seizures	Probability of Severe Handicap or Death*
Mild (Sarnat 1)	Hyperalert	No	Mild	Jittery	No	<1%
Moderate (Sarnat 2)	Lethargy	No	Moderate	High	Yes	25%
Moderate to severe**	Lethargy	Yes	Moderate	High	Yes	50%
Severe (Sarnat 3)	Coma	Yes	Severe	Flaccid	Yes (early)	75%

*See Levene MI et al. *Lancet*. 1986

**Moderate-to-severe encephalopathy: Suggested grade for infants who require respiratory assistance but are not flaccid and definitely comatose. The probability of severe handicap is an approximation based on the author's experience.

Thompson Score* for Neonatal Encephalopathy

Sign	0	1	2	3
Tone	normal	hyper	hypo	flaccid
LOC	normal	hyperalert, stare	lethargic	comatouse
Fits	none	< 3 per day	> 2 per day	
Posture	normal	fisting, cylcing	strong distal flexion	decerebrate
Moro	normal	partial	absent	
Grasp	normal	poor	absent	
Suck	normal	poor	absent ± bites	
Respir	normal	hyperventilation	brief apnea	IPPV (apnea)
Fontanell	normal	full, not tensetense		

*Thompson CM et al: **The value of a scoring system for hypoxic ischaemic encephalopathy in predicting neurodevelopmental outcome.** *Acta Paediatr* 1997, **86**(7)**:**757-761.

The 2nd Consensus Statement 2003

Defining the Pathogenesis and Pathophysiology of Neonatal Encephalopathy and Cerebral Palsy

Gary D. V. Hankins, MD, and Michael Speer, MD

VOL. 102, NO. 3, SEPTEMBER 2003

Essential Criteria

1. Evidence of metabolic acidosis
2. Early onset moderate or severe NE
3. Spastic quadriplegic/dyskinetic CP

AND:

4**. Exclusion of other identifiable etiologies, such as trauma, coagulation disorders, infectious conditions, or genetic disorders.**

Criteria suggestive of intrapartum timing

5. Sentinel Event
6. Sudden fetal brady **CTG Criteria**
7. Apgar Scores **0-3 > 5 mins**
8. Multisystem involvement < 72 hours
9. Early Imaging criteria

The 3rd Consensus Statement 2014

Neonatal Encephalopathy and Neurologic Outcome, Second Edition

Report of the American College of Obstetricians and Gynecologists' Task Force on Neonatal Encephalopathy

PEDIATRICS Volume 133, Number 5, May 2014

- American Academy of Pediatrics
- American College of Nurse-Midwives
- American Gynecologic and Obstetrical Society
- American Society for Reproductive Medicine
- Association of Women's Health, Obstetric and Neonatal Nurses
- Australian Collaborative Cerebral Palsy Research Group
- Child Neurology Society
- Japan Society of Obstetrics and Gynecology
- March of Dimes Foundation
- Royal Australian and New Zealand College of Obstetricians and Gynaecologists
- Royal College of Obstetricians and Gynaecologists
- Society for Maternal-Fetal Medicine
- Society of Obstetricians and Gynaecologists of Canada

The 3rd Consensus Statement 2014; Motivation for a "second edition"

- The Task force recognised that a broader perspective was necessary.
- Based on "the sober recognition that knowledge gaps still preclude a definitive test or set of markers that accurately identifies, with high sensitivity and specificity, an infant in whom neonatal encephalopathy is attributable to an acute intrapartum event."
- As a comprehensive etiologic evaluation is not possible, the term hypoxic–ischemic encephalopathy should best be replaced by neonatal encephalopathy because neither hypoxia nor ischemia can be assumed to have been the unique initiating causal mechanism.

The 3rd Consensus Statement 2014 - Causal Pathways to CP in Term Infants

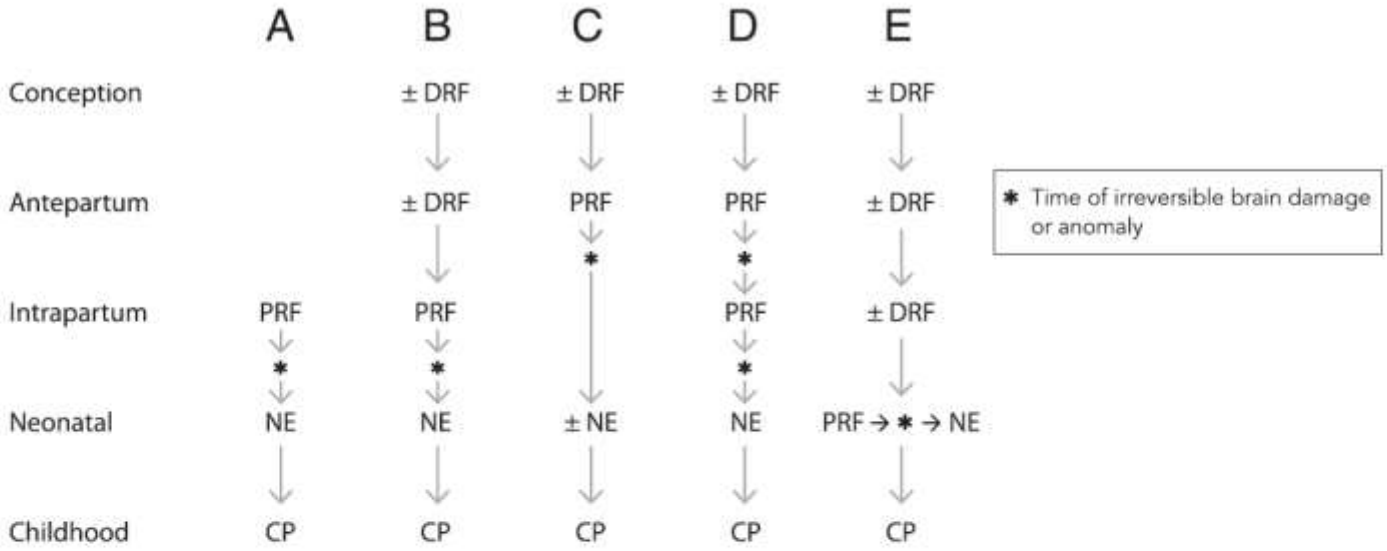
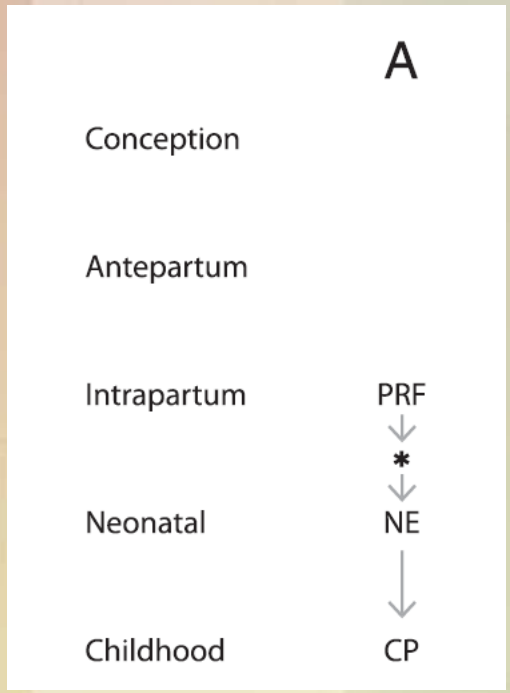


FIGURE 1

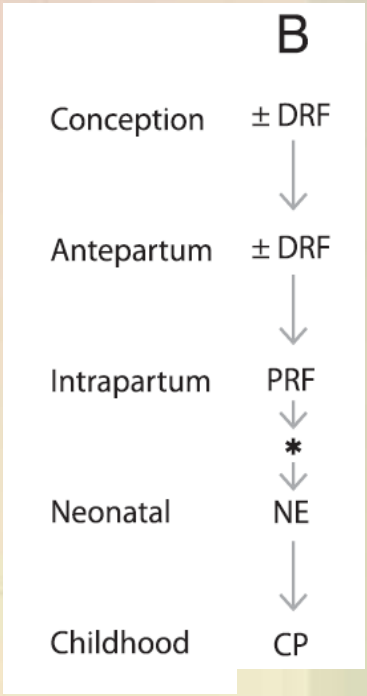
Prenatal and perinatal causal pathways to cerebral palsy in term infants. Distal risk factors exert a pathogenic effect on fetal brain development starting at a time that is remote from the onset of irreversible brain injury. Examples include genetic abnormalities, environmental and sociodemographic factors, and some placental abnormalities. Proximal risk factors exert pathogenic effects on fetal brain development at a time that closely predates or coincides with the onset of irreversible brain injury. Examples include abruptio placentae, chorioamnionitis, and twin–twin transfusion. There are multiple potential causal pathways that lead to cerebral palsy in term infants, and the signs and symptoms of neonatal encephalopathy may range from mild to severe, depending on the nature and timing of the brain injury. **A**. Intrapartum brain injury that is due to a proximal risk factor may lead to neonatal encephalopathy and subsequent cerebral palsy. **B**. Intrapartum brain injury may be the result of both distal and proximal risk factors that predispose the fetus to brain injury and cerebral palsy. **C**. Brain injury or anomaly may occur in the antepartum period as a result of distal and proximal risk factors. When brain injury or anomaly occurs at a time that is remote from the delivery process, neonatal encephalopathy may or may not be seen after birth. **D**. Brain injury may occur at multiple points during gestation. **E**. Proximal risk factor and brain injury may occur in the neonatal period following predisposing distal risk factors. Abbreviations: DRF, distal risk factor; PRF, proximal risk factor.

The 3rd Consensus Statement 2014 Clinical Examples of Causal Pathway A



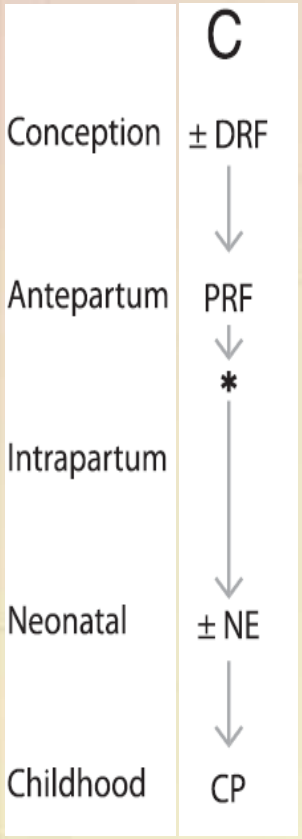
- Causal Pathway A – PRF is a sentinel event. Examples: Abruptio, Prolapsed cord, Ruptured uterus etc
- Causal Pathway A – PRF is not a sentinel event. Examples: Pregnancy induced hypertension, Antepartum haemorrhage, Fetal growth retardation etc.

The 3rd Consensus Statement 2014 Clinical Examples of Causal Pathway B



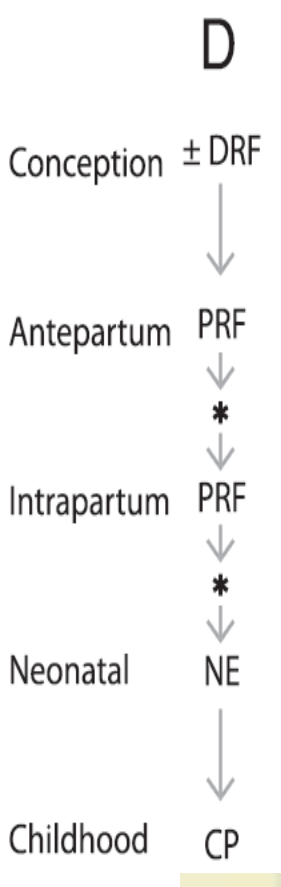
- Causal Pathway B – DRF present at time of conception eg Family history of CP
- Causal Pathway B – DRF present during pregnancy eg Oligohydramnios
- Causal Pathway B – PRF present during labour eg Meconium stained liquor

The 3rd Consensus Statement 2014 Clinical Example of Causal Pathway C



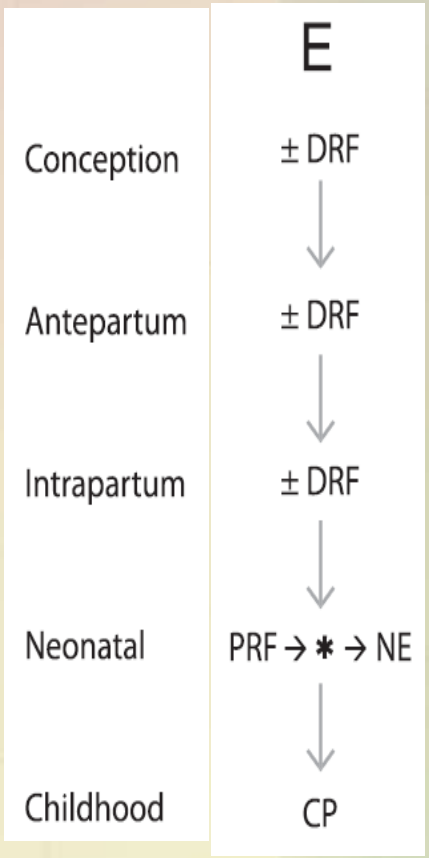
- Causal Pathway C DRF present at conception eg Advanced maternal age
- Causal Pathway C PRF occurs early intrapartum eg Chorioamnionitis
- Causal Pathway C – Neonatal Encephalopathy may be absent

The 3rd Consensus Statement 2014 Clinical Example of Causal Pathway D



- Causal Pathway D – DRF present at time of conception eg maternal seizure disorder
- Causal Pathway D – 1st PRF intrapartum eg chorioamnionitis
- Causal Pathway D – 2nd PRF intrapartum eg tight nuchal cord

The 3rd Consensus Statement 2014 Clinical Example of Causal Pathway E



- 1st DRF – eg Maternal Thyroid disease
- 2nd DRF – eg Oligohydramnios
- 3rd DRF – eg Prolonged rupture membranes
- PRF – Neonatal eg Meningitis

New Consensus Statement
I: Case Definition

I. CASE DEFINITION*

- **Both a + b must be present for any further consideration**

	CONSENSUS STATEMENT	THIS CASE
a	Beyond 35 weeks gestation	*Yes, Junior was a term baby*
b	At least Sarnat II NE With/without Convulsions	*Yes, Junior demonstrated HIE Grade III with early convulsions*

New Consensus Statement: II Neonatal Signs

NEONATAL SIGNS CONSISTENT WITH AN ACUTE PERIPARTUM OR INTRAPARTUM EVENT

	CONSENSUS STATEMENT	THIS CASE
a	APGAR Score < 5 @ 5 mins &	*Yes, the 5-min Apgar was 3/10*
	APGAR Score < 5 @ 10 mins	*Yes; the 10-min Apgar was 5/10*
b	UA pH < 7.0 or	*Borderline- pH was 7.1*
	Base deficit => 12mmol/l	*Yes, the BD was 19.3*
c	Early compat. Neuroimaging	*Early Sonar*
	MRI	
	CT & Sonar	*Early sonar revealed cerebral oedema and periventricular echodensities*
d	Suggestive Multiorgan Failure	
	Kidney	*Yes. Urea and creatinine were transiently elevated*
	Liver	
	Other	*Respiratory Distress*

New Consensus Statement: III Time & Timing of events suggestive of intrapartum/peripartum role

I. TYPE AND TIMING OF CONTRIBUTORY FACTORS THAT ARE CONSISTENT WITH AN ACUTE PERIPARTUM OR INTRAPARTUM EVENT

	CONSENSUS STATEMENT	THIS CASE
a	Sentinel H/I Event	*No details are available to me*
	Ruptured Uterus	
	Major Abruptio placentae	
	Umbilical Cord Prolapse	
	Amniotic Fluid Embolus	
	Maternal CVS Collapse	
	Fetal exsanguination	
	Other	
b	Fetal Heart Rate Patterns	*No details are available to me*
	Category I or II without asph	
	Initial vs Labour CTG Abn	
	Cat II Initially	
	Cat I > Cat III	
	Cat I > Other CTG Abn	
c	Imaging & Timing of insult	
	Cranial Ultrasonography	*Early Sonar was compatible with intrapartum asphyxia*
	Early MRI	*Not done. An MRI is essential at this stage.*
	Patterns of Damage HI	
	Patterns of Damage *not* HI	
d	Proximal contributing factor	*Yes. Evidence of Chorioamnionitis was present at birth*
	Distal contributing factor/s	*None apparent*

New Consensus Statement: IV Developmental Outcome Compatible

I. DEVELOPMENTAL OUTCOME IS SPASTIC QUADRIPLEGIA OR DYSKINETIC CEREBRAL PALSY

	CONSENSUS STATEMENT	THIS CASE
a	Spastic/Dystonic Quad	*Apparently "mixed" spastic / dystonic quadriplegic CP is present. Neurological examination is required*
b	Other Subtypes CP	
c	Other development disorder	*? Epilepsy, Intellectual impairment Neurological examination is required*

Apgar Scores

- Low Apgar scores at 5 and10 minutes clearly confer an increased relative risk of CP and the degree of Apgar abnormality at 5 & 10 minutes correlates with the risk of CP.
- BUT, most infants with low Apgars will not develop CP!
- If the Apgar score at 5 minutes is > 6/10, then it is highly improbably that peri-partum hypoxia-iscaemia played major role in causing neonatal encephalopathy.

Apgar Scores – Policy Statement AAP & ACOG[1]

- It is inappropriate to use an Apgar score alone to establish the diagnosis of asphyxia.
- An Apgar score assigned during resuscitation is not equivalent to a score assigned to a spontaneously breathing infant.
- An Apgar score of 0-3 at five minutes is associated with a *slightly* increased risk of CP. Conversely 75% of children with CP have had normal scores at 5 minutes.
- A five minute score of 7 to 10 is considered normal.
- The risk of poor neurological outcome increases when the Apgar score is 3 or less at 10, 15 and 20 minutes.
- [Apart from asphyxia] the Apgar score is affected by gestational age, maternal medications, resuscitation, cardiorespiratory and neurological conditions.

1. AAP & ACOG. The Apgar Score. *Pediatrics* 2006; 117(4): 1444-1447

Three-Tier Fetal Heart Rate Interpretation System

- **Category I**
  - Baseline rate 110-160 beats/min
  - Moderate variability
  - Absence of any late or variable decelerations
  - Early decelerations may or may not be present
  - Accelerations may or may not be present

Require routine observations without any specific action required

Three-Tier Fetal Heart Rate Interpretation System

Category II

- *Baseline Rate*

  Tachycardia or Bradycardia not with absent baseline variability

- *Baseline FHR Variability*

  Minimal, Absent or Marked baseline variability

- *Absence of Induced Accelerations* (eg scalp stimulation)
- *Periodic or Episodic Decelerations*

  Recurrent variable decels with min or mod baseline variability

  Prolonged decels >2min but < 10min

  Recurrent late decels with mod baseline variability

  Variable decels with other characteristics such as slow return to baseline, “overshoots” or “shoulders”.

Indeterminate tracings; require continued surveillance & re-evaluation

Three-Tier Fetal Heart Rate Interpretation System

Category III

- *Absent baseline FHR variability with any of:*
  - Recurrent late decels
  - Recurrent variable decels
  - Bradycardia
- *Sinusoidal Pattern*

Abnormal tracings predictive of fetal acidemia. Require prompt actions.

CTG

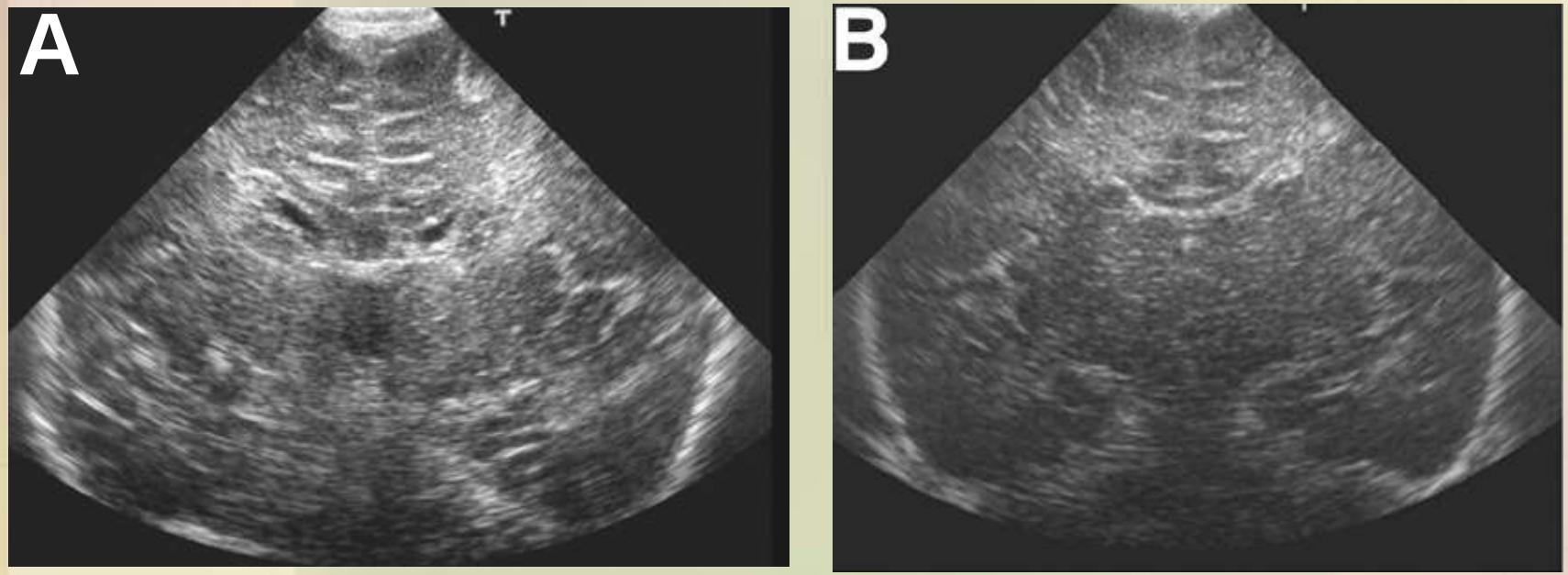
1. A Category I or Category II fetal heart rate tracing when associated with Apgar scores of 7 or higher at 5 minutes, normal umbilical cord arterial blood (± 1 standard deviation), or both is not consistent with an acute hypoxic–ischemic event.
2. There is a great distinction to be made between a patient who initially presents with an abnormal fetal heart rate pattern and one who develops an abnormal fetal heart rate pattern during labour.
   a) A category II fetal heart rate pattern lasting 60 minutes or more that was identified on initial presentation with persistently minimal or absent variability and lacking accelerations, even in the absence of decelerations, is suggestive of a previously compromised or injured fetus.
   b) The patient who presents with a Category I fetal heart rate pattern that converts to Category III as defined by the Eunice Kennedy Shriver National Institute of Child Health and Human Development guidelines is suggestive of a hypoxic–ischemic event.
   c) Additional fetal heart rate patterns that develop after a Category I fetal heart rate pattern on presentation, which may suggest intrapartum timing of a hypoxic–ischemic event, include tachycardia with recurrent decelerations and persistent minimal variability with recurrent decelerations.

Cerebral Palsy Litigation:
Change Course or Abandon Ship

Sartwelle TP & Johnston JC. *Journal of Child Neurology* 2015, Vol. 30(7) 828-841

- One of the cardinal drivers of birth injury claims is electronic fetal monitoring
- The scientific foundation for it's use is almost non-existent
- Its false-positive rate exceeds 99%
- It does not predict cerebral palsy
- In the last 40 years monitoring has harmed more mothers and babies than it ever helped
- Birth is a dangerous journey and monitoring doesn't help

Early Ultrasound



Early Ultrasound in Term Asphyxia

Table 2 Comparison of early cranial ultrasound findings between asphyxiated and non-asphyxiated term infants

Ultrasound findings	Asphyxiated infants $n = 104$ (%)	Non-asphyxiated infants $n = 76$ (%)	P values
General increase in echodensity of cerebral parenchyma	38.7	1.3	< 0.0001*
Increased periventricular echodensity	61.5	34.2	0.0005*
Loss of normal sulcoparenchymal differentiation	26.0	0.0	< 0.0001*
Slit-like lateral ventricles	44.2	9.2	< 0.0001*
Increased subependymal echogenicity	1.0	1.3	1.0
Intraventricular haemorrhage	26.9	6.6	0.001*
Echodense thalamus	30.8	2.6	< 0.0001*
Echodense brainstem	51.9	42.1	0.39
Echodense cerebellum	22.1	6.5	0.02*

*Statistical significance.

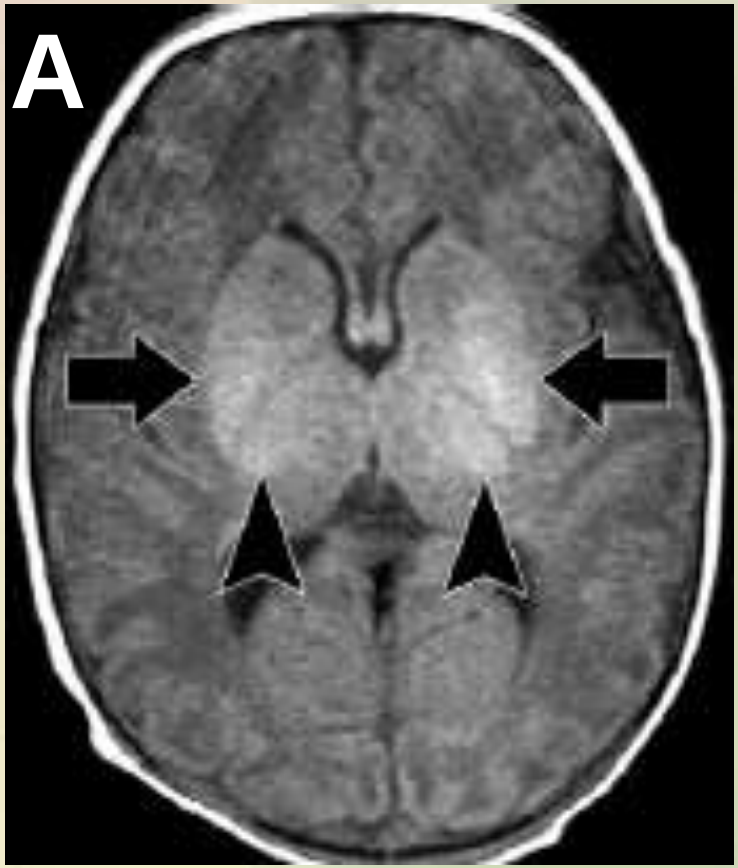
BOO NY, CHANDRAN V, ZULFIQAR MA. Early cranial ultrasound changes as predictors of outcome during first year of life in term infants with perinatal asphyxia. *J. Paediatr. Child Health* (2000) 36, 363–369

Imaging; the MRI

- An MRI is the best modality for demonstrating the nature and extent of cerebral injury.
- Cranial ultrasonography and CT lack sensitivity needed to define the injury.
- The optimal time to do the MRI is 10 days (7-21days)
- If an MRI done anytime after 24 hours shows no injury, then it is unlikely that peripartum or intrapartum H-I brain injury was a significant factor in NE.

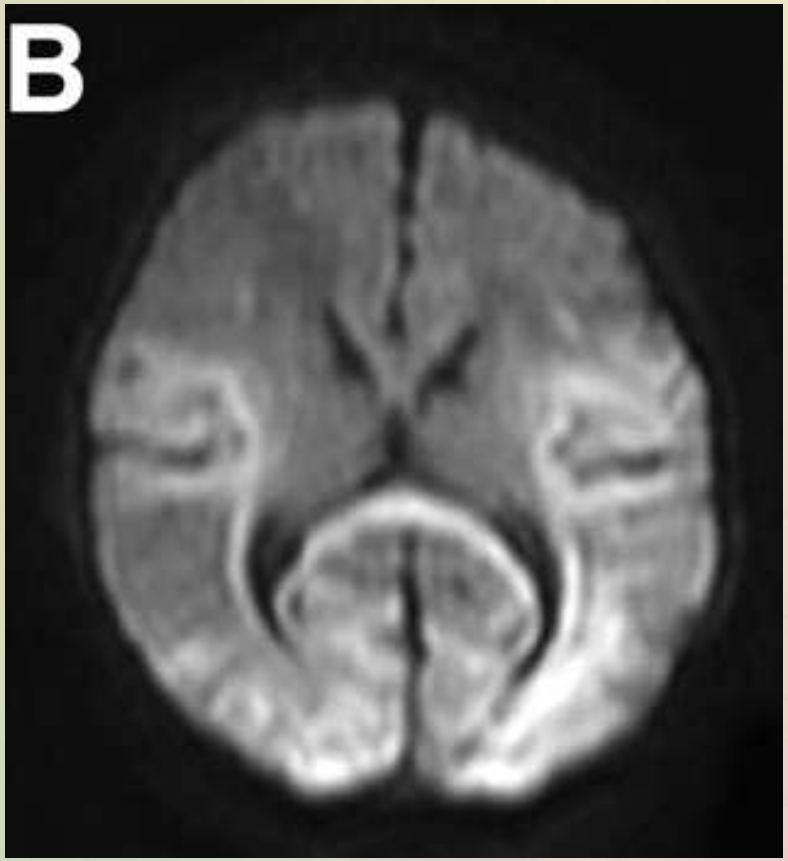
Classic MRI Patterns of an Intrapartum Aetiology

Acute Profound HI

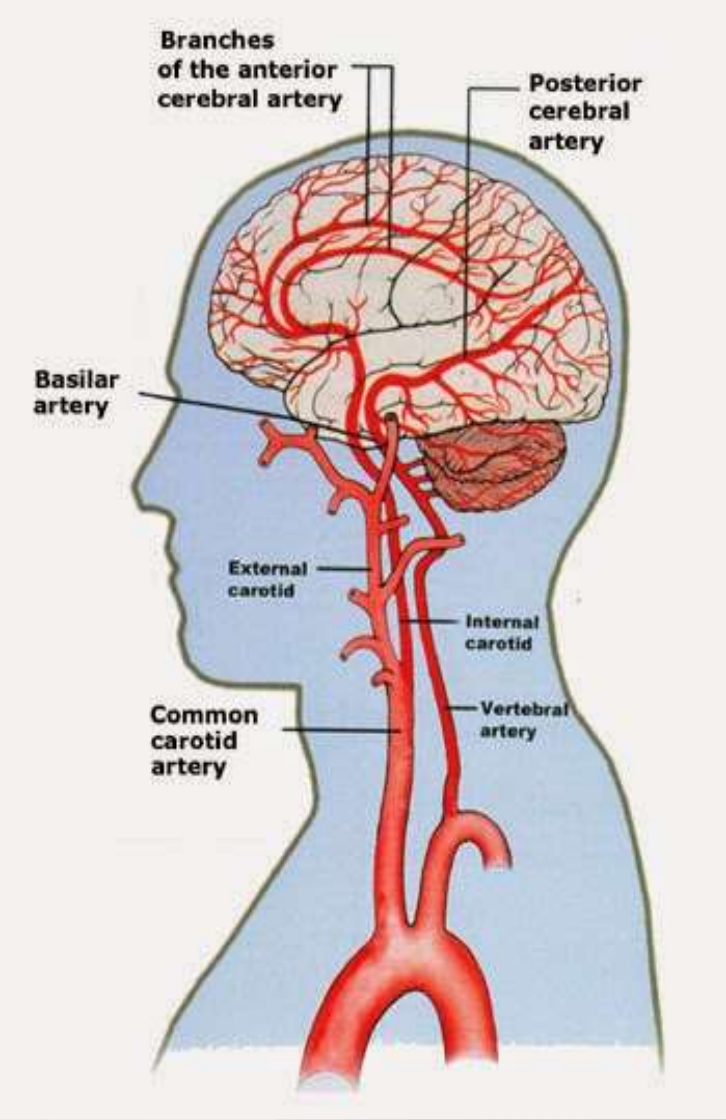


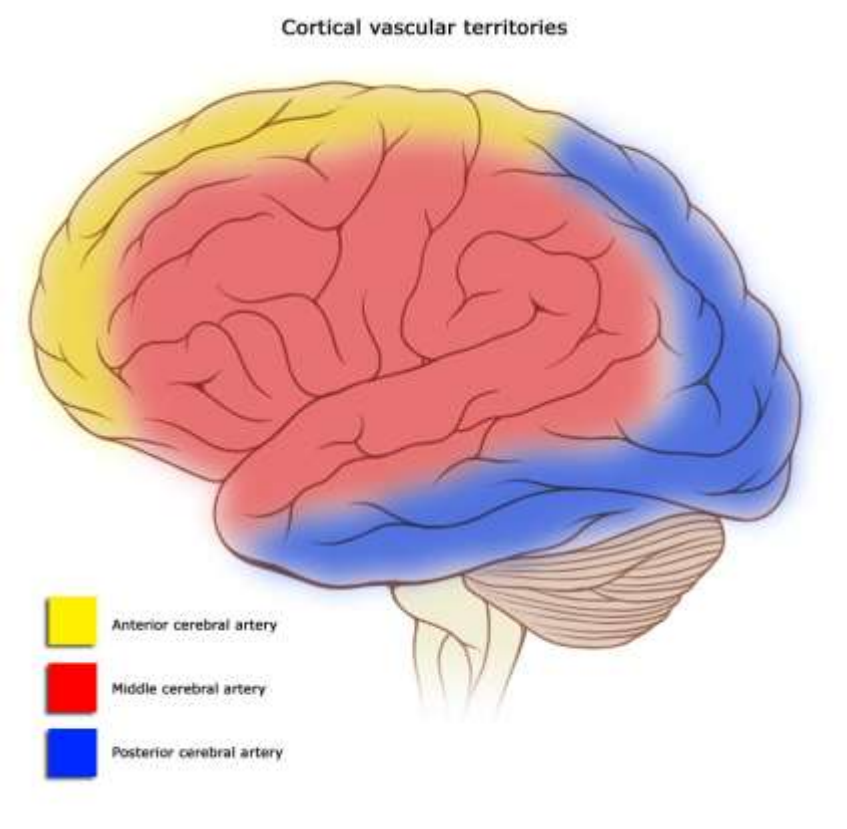
Predominantly Basal Ganglia

Partial Prolonged Asphyxia



Predominant “watershed” White Matter


Branches of the anterior cerebral artery
Posterior cerebral artery
Basilar artery
External carotid
Internal carotid
Vertebral artery
Common carotid artery


Cortical vascular territories
Anterior cerebral artery
Middle cerebral artery
Posterior cerebral artery

Summary

- The “epidemic” in litigation for alleged negligence in obstetric & neonatal care has unsustainable economic and morale consequences in both the private and public health sectors
- There are many areas which need to be tackled to minimise this problem. These involve medical, legal and societal remedies.
- Understanding the complex nature of the role of birth asphyxia in aetiology of irreversible brain damage and the resultant cerebral palsy is important if we are to justly compensate where negligence occurred but equally, to vigorously defend healthcare professionals against unjust prosecution.

"..the salient question being – 'who, if anyone, will be performing deliveries in private practice by the end of the decade?' If the answer to the question is 'nobody', the consequences will extend beyond private healthcare. There are also serious implications for the state sector that will require addressing[1]".

1. Howarth GR. Obstetric risk avoidance; will anyone be offering obstetrics in private practice by the end of the decade? *S Afr Med J* 2013;103(8):513-514.

Thank you!

- Keith Bolton declares that he has referenced any work that was not his own.
- The visuals used are either referenced or common property.
- He has received precious little incentive, perverse or otherwise, for this talk.
- He declares no existing or potential conflicts of interest other than giving numerous “expert paediatric opinions” to assist the Court in negligence cases.