The Role of the Obstetrician in preventing Cerebral palsy and protecting oneself from litigation.

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The Facts about Cerebral Palsy

- The overall incidence of cerebral palsy is about 0.2% (2 in every 1000 births)
- The cause can be genetic, a problem during the pregnancy (which may or may not be recognizable), a problem during labour (particularly hypoxia), or a problem occurring after delivery usually within the first 1-2 years of life (cerebral haemorrhage, infection, infarction etc).
- It is generally accepted that intrapartum hypoxia is the cause in about 10% of cases, with some of this hypoxia not being preventable, some not recognized although it should have been, and some not acted on as quickly as it should have been.

The Facts about Cerebral Palsy

- High payouts are now common where inadequate care has been defined by the Courts, and many specialists and GP's are ceasing to deliver babies because of the litigation risk and high pay-outs.
- The largest Obstetrics and Gynaecology claim to date in Australia was a CP claim, finalised for $16.2 million in 2004. In Malaysia a recent award was about 4.5 million MR (about 1.5 million A$).

Matters covered in this presentation

- Causes of Neonatal Encephalopathy as defined by Badawi in 1998.
- Epidemiologic Associations with Cerebral Palsy as defined by the Australian Collaborative Research Group in 2011.
- International consensus statements regarding intrapartum hypoxia.
- Methods to be used to protect yourself from litigation.

Genetic risk factors for neonatal encephalopathy. (Badawi, BMJ)

• Family history of siezures	2.55	(5.1/!000)	
• Family history of nervous disorders	2.77	(5.5/1000)	
• Family history of coagulopathy	??		*

The Relative Risk (RR) is quoted.

*This data is not in the Badawi publication
317: 1549 – 1558 (1998)

Antenatal risk factors for neonatal encephalopathy. (Badawi, BMJ)

• Viral illness	2.97		
• Moderate/severe APH	3.57		
• IUGR (3rd to 9th percentile)	4.37	(8.7/1000)	
• IUGR (<3rd percentile)	38.23	(76/1000)	
• Previous infertility treatment	4.43		
• Severe pre-eclampsia	6.30		
• Maternal thyroid disease (± treat)	9.70		
• Death of a dichorionic twin	????		*
• Death of a monochorionic twin	?250.00	(500/1000)	*
• Reduced or no fetal movements	????		*

Intrapartum risk factors for neonatal encephalopathy. (Badawi, BMJ)

• Intrapartum fever (infection)	3.82	
• OP presentation in labour/delivery	4.29	
• Acute intrapartum event	4.44	
• No labour or elective CS	0.17	
• Emergency Caesarean section	2.17	
• Instrumental delivery	2.34	
• GA for delivery	3.08	
• Premature delivery	????	*

Risk of cerebral palsy following premature delivery

- Term delivery (2/1000)
- Delivered < 28 weeks 76/1000
- Birth weight < 1500g 50-80/1000

- Delivered at 24 weeks ?200/1000

Incidence of CP in multiple pregnancy.

- BMJ 1993;307:1239-43.
- WA Research Institute of Child Health.
- 10 year intake (1980-1989).
- 228,329 singleton births
- 5288 births from twin pregnancies.
- 237 births from triplet pregnancies.

Multiple pregnancy outcome (BMJ 1993; 307: 1239-43)

	Death rate per 1000 births	CP rate per 1000 births
Singleton pregnancy	15.6	1.6
Twin pregnancy	70.5	7.4
Triplet pregnancy	92.8	26.7

Risk of CP in multiple pregnancy

- Number of fetuses
- Gestation at delivery.
- Birth weight.
- Evidence of IUGR.
- Chorionicity- MCMA bad. ?MCDA worse, especially if TTTS or FDIU of one twin (resulting in release of thromboplastins from dead twin, or exsanguination of live twin into dilated vascular system of dead one).

Does the vanishing twin (VT) pose a risk of Cerebral Palsy to the live one?

- The incidence of VT has been reported recently.
- Some reported series indicate the chance of an initially diagnosed twin pregnancy finishing as a singleton pregnancy by 12 weeks of gestation, as being 85%.
- These numbers are probably inflated because many of the twins having very early US (<6 weeks) are as a result of IVF. Need more studies to provide accurate figures.
- Is chorionicity important? Most are probably DCDA, and in MCDA both twins would be lost.

Epidemiologic Associations with Cerebral Palsy.

- 587 cases of CP and 1154 controls.
- Data published in Obstetrics & Gynecology 118:576-82 (2011)
- Types of CP in cases-
  - Hemiplegia 191 (33.4%)
  - Diplegia 149 (26%)
  - Quadriplegia 145 (25.3%)
  - Other types 70 (12.2%)

Maternal infection and CP

Infection type	Odds ratio (95% CI)	P test result
Any infection	1.71 (1.39-2.11)	<0.001
URTI (0-20W)	1.26 (0.90-1.77)	.18 NS
Gastrointestinal (0-20W)	1.74 (0.84-3.59)	.13 NS
Herpes (0-20W)	2.00 (1.01-3.94)	.04
Fever (20-40W)	5.05 (2.21-11.54)	<0.001
Infection in labour & del	2.95 (1.65-5.26)	<0.001
Urinary tract infection	1.53 (0.91-2.57)	.11 NS
Other infections (21-40W)	3.98 (2.20-7.22)	<0.001

Birth weight and CP (weight compared with 40-60th centile).

Weight Category	Odds Ratio (95% CI)	P test result
Less than 3rd centile	11.5 (6.25-22.08)	<0.001
4th -5th centile	2.63 (1.22-5.68)	.01
6th -10th centile	2.13 (1.26-3.62)	.004
10th -19th centile	1.67 (1.09-2.54)	.02
20th -29th centile	1.27 (0.81-13.97)	.30 NS
30th -39th centile	1.52 (1.02-2.28)	.04
< 40th centile	2.02 (1.50-2.73)	<0.001

Other associations with CP – 1.

Association	Odds Ratio (95% CI)	P test result
Deliv < 32 W, cf >36W	70.62 (34.38-145.04)	<0.001
Deliv 32-36W, cf >36W	5.02 (3.49-7.21)	<0.001
Twins cf Singletons	6.62 (4.00-10.94)	<0.001
Breech cf cephalic	2.48 (1.76-3.49)	<0.001
Vag Breech deliv cf NCVD	8.36 (3.30-21.19)	<0.001
Emerg CS breech cf NCVD	4.48 (2.62-.65)	<0.001
Elective CS breech cf NCVD	1.32 (0.75 – 2.34)	.34 NS

Other Associations with CP - 2

Association	Odds Ration (95% CI)	P test result
Disappearing twin on US	1.98 (0.82-4.79)	0.12 NS
Bleeding during pregnancy	2.04 (1.61-2.58)	<0.001
Any Miscarriages cf none	1.25 (1.00-1.57)	0.05
Three or more misc. cf none	2.30 (1.38-3.82)	0.001
Drug use in pregnancy	2.22 (1.14-4.30)	0.02
Smoking in pregnancy	1.37 (1.02-1.85)	0.04
Male sex of baby cf female	1.68 (1.38-2.06)	<0.001

Other Associations with CP – 3.

Association	Odds Ratio (95% CI)	P test result
Apgar score at 1 minute <4 cf >8	20.27 (11.29-36.42)	<0.001
Apgar score at 5 minutes <4 cf >8	51.27 (12.20-215.47)	<0.001
Emergency CS delivery cf NVD	2.42 (1.88-3.12)	<0.001
Elective CS delivery cf NVD	1.39 (1.00-1.94)	0.05

International consensus statements regarding cerebral palsy.

- Two such statements have been published
- First was by a group of international experts BMJ 1999: 319; 1054-1059, A. MacLennan et al.
- Second by the ACOG and American Academy of Paediatrics, and published by them in January 2003.
- The statements are similar but not identical

International Consensus Statements concerning intrapartum cause of CP.

- Essential Criteria for diagnosis of intrapartum hypoxia (MacLennan et al. criteria)
  - Evidence of metabolic acidosis- scalp sample, cord arterial blood or early neonatal blood (pH<7.00, base deficit >12mmol/L*). (ACOG/AAP only allows cord blood results)*
  - Early onset of severe or moderate encephalopathy.
  - Cerebral palsy of spastic quadriplegic or dyskenetic type.
  - *Exclusion of other aetiologies- trauma, coagulation disorders, infectious conditions, genetic disorders.(ACOG/AAP criteria, in addition)*

International Consensus Statement concerning intrapartum cause of CP.

- Non-specific Criteria for diagnosis of intrapartum hypoxia
  - Sentinel hypoxic event immediately before or in labour- APH, cord prolapse.
  - Sudden, rapid and sustained deterioration in FHR pattern, usually after a sentinel event, prior to which the FHR was normal. *(Sudden & sustained bradycardia, or absense of FHR variability,in presence of persistent late or variable decelerations ACOG/AAP).*
  - Apgar scores of 0-6 for longer than 5 minutes. *(Scores of 0-3 for more than 5 minutes ACOG/AAP)*
  - Evidence of multisystem involvement *(within 72 hours of birth-ACOG/AAP)*
  - Early imaging evidence of acute cerebral abnormality. *(Needs to be non-focal ACOG/AAP)*

CTG abnormalities and Cerebral palsy (Parer et al. J.MFNMed vol 19)

- The facts.
- Moderate FHRV was strongly associated with an umbilical artery pH of >7.15
- Undetectable or minimal FHRV, in the presence of late or variable decelerations was the commonest predictor of newborn acidaemia (but only 23%)
- There was a positive relationship between the degree of acidosis and depth of deceleration or bradycardia
- Except for that due to a sudden sustained bradycardia, newborn acidaemia associated with decreased FHRV develops over about 1 hour.

CTG abnormalities and Cerebral palsy (Nelson et al, NEJM 1996;334:613-8)

- Only CTG abnormalities associated with CP (Nelson et al) were those of recurrent late or prolonged decelerations or profound loss of short term FHR variability.
- False positive rates of FHR abnormalities were very high.
- Almost all labours have some FHR abnormalities.

IF INTRAPARTUM HYPOXIA IS DEFINED, ASSESS THESE …

- Was there a sentinel hypoxic event? - cord prolapse, APH
- What was the duration of probable hypoxia?
- Was there an intervention available proven to shorten this?
- Could signs of fetal compromise in labour have been detected earlier?
- Was there an avoidable delay in expediting delivery once fetal distress was defined?

IF INTRAPARTUM HYPOXIA IS DEFINED, ASSESS THESE …

- Would quicker delivery of the baby have compromised the mother's health or life?
- Would an earlier delivery, if practical, have prevented or ameliorated the outcome?
- Are there any additional investigations or assessments required? - MRI, inherited thrombophilias etc

What are the general criticisms levelled against the obstetrician?

- Inadequate fetal monitoring.
- Inappropriate mode of delivery.
- Delay in expediting delivery.
- Inadequate resuscitation, including absence of a paediatrician at delivery.

AS AN OBSTETRICIAN, WHAT IS YOUR ROLE IN PROVIDING SPECIAL ANTENATAL CARE IN HIGH RISK CASES.

- Ensure adequate care in high risk cases.
  - Inhibit premature labour.
  - Look for evidence of chorioamnionitis after PPROM.
  - ?timing of delivery after severe IUGR identified.
  - Adequate fetal monitoring in at risk cases- US, CTG, Doppler studies.
  - Lean towards CS delivery in high risk cases.
  - Specialized care of multiple pregnancies.
  - Use of antenatal IV MgSO4 (in addition to glucocorticoid therapy) prior to pre-term birth (<30W) - 4g loading dose, then 1g/hr for 4-24 hours. (Cochrane review indicated RR =0.68)

AS AN OBSTETRICIAN, WHAT IS YOUR ROLE IN PROVIDING APPROPRIATE CARE IN LABOUR IF I/P HYPOXIA IS TO BE PREVENTED?

- Ensure CTG monitoring is used in labour, if indicated- PET, previous APH, GDM, clinical IUGR, meconium, use of syntocinon/PG, use of epidural anaesthesia.
- PG inductions are not free of risk- get consent.
- Turn off syntocinon if CTG becomes abnormal.
- If S2 becomes prolonged, monitor fetus with CTG.
- Ensure minimal delay when delivery is required.
- Assess fetal scalp pH or lactate in S1 of labour, if possible hypoxia is defined.
- If anticipate a “problem” fetus, have paediatrician there for the delivery.Don’t try to find one later.

DELAY IN EXPEDITING DELIVERY

- Caesarean Section (SA data) Decision/delivery interval in metropolitan Adelaide, where urgent delivery was required. (Spencer & MacLennan, ANZJOG 2001;41:7-11)
  - Level 3: Median= 42 minutes (17-86 range)
  - Level 2: Median= 54 minutes (28-94 range)
  - Level 1: Median= 69 minutes (37-114 range)

DELAY IN EXPEDITING DELIVERY

Reason for CS	level-1	level-2	level-3
Non-reassuring FHR	69(9)	57(67)	43(130)
Abnormal pH	-	30(5)	33(19)
Failed instr.deliv	15(1)	53(3)	21(7)
Cord pres/prolapse	-	17(3)	28(2)
APH	-	53(11)	40(16)

The initial figure is the decision/delivery in minutes.
The figure in brackets is the number of deliveries.

DELAY IN EXPEDITING DELIVERY

- Forceps delivery- delay due to need for PV to assess position of head, insertion of anaesthetic, accurate placement of forceps on fetal head, rotation between contractions, and traction during contractions.
- Ventouse delivery- delay due to time taken to create the “vacuum”, traction only with contractions, always takes longer than forceps, often cup becomes dislodged.

AS AN OBSTETRICIAN, WHAT IS YOUR ROLE AT THE TIME OF DELIVERY, IF I/P HYPOXIA IS LIKELY?

- Resuscitation of the baby, if no paediatrician in attendance.
- Quickly clamp (x2) the cord.
- If Apgar scores are low, collect cord blood for measurement of pH, and base excess levels.
- If baby shows evidence of IUGR or low Apgar scores at birth, get the paediatricians involved to assess, define further care, including diagnostic tests, glucose levels etc. **AND**
- Use of early U/S and MRI for diagnostic and timing considerations.