

# Only An Expert Witness Can Prevent Cerebral Palsy!

No clinical obstetric policy or intervention has been shown to reduce the risk of cerebral palsy in late pregnancy. The rate of cerebral palsy (2-2.25/1000 deliveries) has remained constant over the last 40 years despite a quadrupling of caesarean section rates, the introduction of electronic fetal monitoring and better obstetric and neonatal care. Despite this, a small group of regularly commissioned plaintiff expert witnesses will often opine that, on the balance of probability, electronic fetal monitoring would have or did show clear signs of fetal compromise, which was still at a reversible stage, and that 'immediate' delivery by caesarean section about an hour earlier than occurred would have prevented the cerebral palsy outcome.

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**This is the usual plaintiff expert opinion seen in most cases of cerebral palsy litigation. This opinion is not peer reviewed, rarely undergoes public or scientific scrutiny, is not a view that has supporting data and is usually expressed by an 'expert' who has retired from obstetrics or practises in a different subspeciality. These experts have not published research in cerebral palsy causation and rarely quote recent research or consensus statements on this topic.**

Australia and the USA are the countries where cerebral palsy litigation appears to be most common. Although it is easy to blame an adversarial legal system, contingency fees and a public with high expectations of a health baby, litigation cannot be successful unless 'experts' can be found to say that the adverse outcome is preventable, that the clinical care was negligent and that the negligent care caused the adverse outcome. In most cases of cerebral palsy there are major associated contributing factors to the neuropathology such as intrauterine infection, preterm delivery, intrauterine growth restriction, complications in a multiple pregnancy, antepartum haemorrhage and congenital neurological anomalies.

The South Australian Cerebral Palsy Research Group has recently shown that there are other, often silent, antenatal factors that are associated with a cerebral palsy outcome such as hereditary

thrombophilias<sup>1</sup>, cytokine polymorphisms<sup>2</sup> and viral exposure<sup>3</sup> during the perinatal period. Other factors such as a tight nuchal cord at birth<sup>4</sup> and intrapartum pyrexia<sup>5</sup> have also been strongly associated with a cerebral palsy outcome. The latter may reflect an early intrauterine infection that may or may not have developed to a chorioamnionitis. Intrauterine or neonatal infection, especially in the more vulnerable brain of a premature infant, may initiate an inflammatory cytokine response that attacks developing neurones. There are probably many co-factors that are yet to be understood that make some fetuses or neonates more prone to such an on-going destructive process that leads to the neuropathology of cerebral palsy over the perinatal period.

Very few babies that develop cerebral palsy are born with a severe metabolic acidosis (pH <7.00 and Base Excess <-12), which is the gold standard to define hypoxia (asphyxia) at birth. Amongst these babies are many where the hypoxia is of chronic origin or is a recurrent acute hypoxia on top of chronic hypoxia or other fetal compromise such as sepsis. Only rarely does acute intrapartum hypoxia occur in a previously healthy baby and it takes a severe hypoxic sentinel event to occur in labour to render a prolonged insult severe enough to cause cerebral palsy, eg prolapsed cord, ruptured uterus, large antepartum haemorrhage, amniotic fluid embolism. To help define the very few babies, who may have suffered a *de novo* acute asphyxial event sufficient on its own to cause cerebral palsy, two international cerebral palsy consensus statements have been published, with mostly similar criteria, in 1999 and 2003<sup>6,7</sup>. Both have been endorsed by RANZCOG.

In October 2005 four senior authors from these international cerebral palsy task forces published an editorial explaining their concerns about the use of selected plaintiff experts who opine that cerebral palsy is preventable<sup>8</sup>. These experts usually imply that the neuropathology can be recognised in labour at a preventable or reversible stage in time to allow the expeditious delivery of a healthy baby. These opinions are based on scientifically unproven premises.

Firstly there is no good evidence that electronic fetal monitoring and appropriate reactions to its results reduces cerebral palsy rates. Secondly there is no evidence that by the time there are non-reassuring fetal heart rate patterns the process leading to the neuropathology of cerebral palsy has not already become established or that it is reversible. Thirdly, the plaintiff expert often opines that delivery could have occurred within minutes of the first signs of possible non-reassuring fetal status without acknowledging the high rate of false positive signs, the maternal risks of high caesarean section rates and, in particular, the median and 90th percentile times taken in hospitals with varying facilities to perform urgent caesarean sections. Most plaintiff experts advocate abdominal delivery within 15-30 minutes and ignore international and Australian audits of decision-to-delivery times for "urgent" emergency caesarean sections, median and (90th percentile) of 42 (86), 54 (94) and 69 (114) for Level 3, 2 and 1 hospitals respectively<sup>9</sup>.

Fourthly, the plaintiff expert provides no evidence that quicker delivery in such circumstances by up to an hour reduces cerebral palsy risk and indeed there is some evidence to the contrary<sup>10,11</sup>. Lastly, the plaintiff expert often makes the incredulous jump from details in the management of labour, that he or she thinks were inappropriate or negligent, to the opinion that this management was the cause of the cerebral palsy. If the labour was induced or augmented uterine hyper stimulation is sometimes claimed, often without evidence of this, and even labours of above average length or the forces of normal labour are blamed for presumed hypoxia. It is usually not explained that the healthy fetus is well protected from the normal 'forces' of labour and hypoxia with fetal haemoglobin, preferential fetal circulation to the brain, relatively low oxygen requirements, head moulding, etc.

Few plaintiff experts acknowledge the multiplicity of antenatal causative factors, the fact that many can be silent during pregnancy and labour, that many are probably still unknown and that some are almost impossible to recognise many years later on review of the hospital and medical case notes. A few selected plaintiff radiologists may try to opine that on the basis of neuro-imaging months and years after delivery that they can determine an asphyxial cause from the imaging patterns and that over the many weeks of the perinatal period they can determine it occurred during the minutes before birth. It has never been published that neuroradiologists, blinded to the paediatric outcome, have verified the ability to predict, in retrospect, the timing and cause of the neuropathology, eg pre- or post-partum hypoxia or infection.

Cerebral palsy is not a preventable disorder and has largely congenital or antenatal pathological causes. A few occur during childhood. The major types of cerebral palsy are usually associated with long-term hardship for the child and its family. They usually receive or out to receive a weekly disability pension sufficient for their particular needs without having to go through a prolonged and distressing litigation lottery which can take 20 or more years. Estimates of the costs of cerebral palsy litigation are that up to 70 per cent of the costs are spent on the legal process. Clearly a 'no fault' system for neurologically disabled children should be considered soon in Australia. This could be simply to legislate that cerebral palsy should not be the subject of litigation. Whether all cerebral palsy children deserve a bigger disability pension than other disabled children is a separate question. The current situation uses obstetricians, midwives and the maternity services as an inefficient and expensive *de facto* extra social welfare system for a few of these unfortunate children and their families. The cerebral palsy litigation industry is gradually destroying the Australian

maternity services. General practitioner obstetricians are disappearing, country and smaller city maternity hospitals are closing and our 2001 survey of Australian obstetricians suggested that the rate of retirement expected this decade would greatly exceed the numbers of graduates in training<sup>12</sup>. This survey also showed that 'fear of litigation' was a major reason for stopping obstetric practice.

Legislative changes, especially if there is a strong legal lobby against them, are slow to enact. However, our College can effectively act by policing non-evidence based opinion and rogue expert witnesses. Its list of fellows accredited to give opinion on obstetric cases should include only experienced currently practising obstetricians and/or those researching in causation of the adverse event in question eg cerebral palsy. Those giving opinion should be trained in giving medico-legal opinion, should keep up to date in their areas of professed expertise and should not assume that deviations from their retrospective choice of clinical care were the cause of the child's disability. Cerebral palsy causation is a very complex area where many of the individual causes have yet to be found. An experienced obstetrician who delivers 5000 babies in a lifetime may deliver about 12 babies that are later diagnosed with cerebral palsy. Unfortunately, few obstetricians are informed about such outcomes and it may only be those who initiate legal proceedings that come to their attention. The extent of such personal experience is not an effective scientific audit to determine the causes and prevention of cerebral palsy and does not make the experienced obstetrician an expert in causation. Paediatric neurologists can be even more in the dark about the possible antenatal causes of cerebral palsy but one or two do regularly offer plaintiff opinion on causation and even obstetric management!

In the United States of America some colleges have introduced a 'yellow card, red card' system where their fellows have been found to be offering inappropriate non-scientific medico-legal opinion, which is to the detriment of the future practice of medicine<sup>13</sup>. The 'yellow card' is a warning to stop offering such rogue opinions and the 'red card' is issued if a complaint is upheld that the expert has erred again. This means loss of Fellowship and presumably loss of face and credibility in court. Several of us have encouraged our College to introduce such a system before the impossible standards of non-practising obstetricians take precedence over scientific systematic reviews or commonsense! Legislation will be necessary to avoid 'hired gun' expert witnesses being commissioned from abroad. Where appropriate Australian or New Zealand expert witnesses are unavailable the courts should choose the overseas experts.

The recent JAMA editorial entitled, 'Who will deliver our grandchildren? - Implications of Cerebral Palsy Litigation', outlines the problems that are often common to the United States of America and Australia and included scientific argument for the lack of evidence that cerebral palsy can be prevented by 'better' intrapartum care<sup>8</sup>. More importantly, it proposed solutions (Box 1) to the obstetric litigation crisis.

#### BOX 1

##### Suggested Solutions to Cerebral Palsy Litigation

1. A 'no fault' system for cerebral palsy. With or without extra pension.
2. Health courts and better dispute resolution process for children with cerebral palsy.
3. Better college and medical board policing of rogue medico-legal opinions eg with a yellow card/red card system.
4. Better internal audit, review and feedback to parents of babies born with early neurological disabilities eg neonatal encephalopathy.

## BOX 2

## Protocol On Delivery Of A Baby With Neonatal Complications

1. Clear contemporaneous notes about all decisions made before and during labour and delivery.
2. Retain all cardiotocographic records, partograms etc.
3. Obtain arterial cord gases or if not possible neonatal arterial gases within one hour of birth.
4. Send the placenta to pathology with a specific request for a perinatal pathologist to look for chorioamnionitis, funisitis, villitis etc.
5. Document all antenatal risk factors, infections, intrapartum pyrexia etc especially if GP/private notes are not to be retained in the hospital notes.
6. Request that the cord and neonatal bloods are examined for nucleated red blood cells and hereditary thrombophilias (FVL, PGM, MTHFR).
7. Request that neonatal ultrasound or other brain imaging is performed in the first four days of life.
8. Keep in close sympathetic communication with the parents.
9. Discourage statements that infer blame, culpability or preventability and have a case discussion with staff involved to offer support and education.
10. Keep all records for 25 years and ask to be informed of any long-term adverse outcome.

Finally, obstetricians and midwives can help themselves and their patients by following evidence based protocols and if they deliver a baby that requires resuscitation or admission to neonatal care they should make sure that all the suggestions in Box 2 are clearly documented in the notes<sup>14</sup>. Such documentation can often elucidate then, and years later, some of the common associations with a cerebral palsy outcome. This helps research into the causes of cerebral palsy and can help protect those involved with the infant's delivery, from non-evidence-based advocacy that 'the cause of the cerebral palsy was due to recognisable, reversible brain pathology, due only to acute asphyxia in a previously healthy baby.' 'It progressed to cerebral palsy because of the negligent inability of the accoucheur to deliver the baby quicker.' The standards of obstetric care are being arbitrarily set by those who do not have to work to those standards, without evidence for their safety and efficacy, in ignorance or defiance of better scientific evidence and without concern about their effect on the Australian health system and the women and children who must find obstetric care.

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