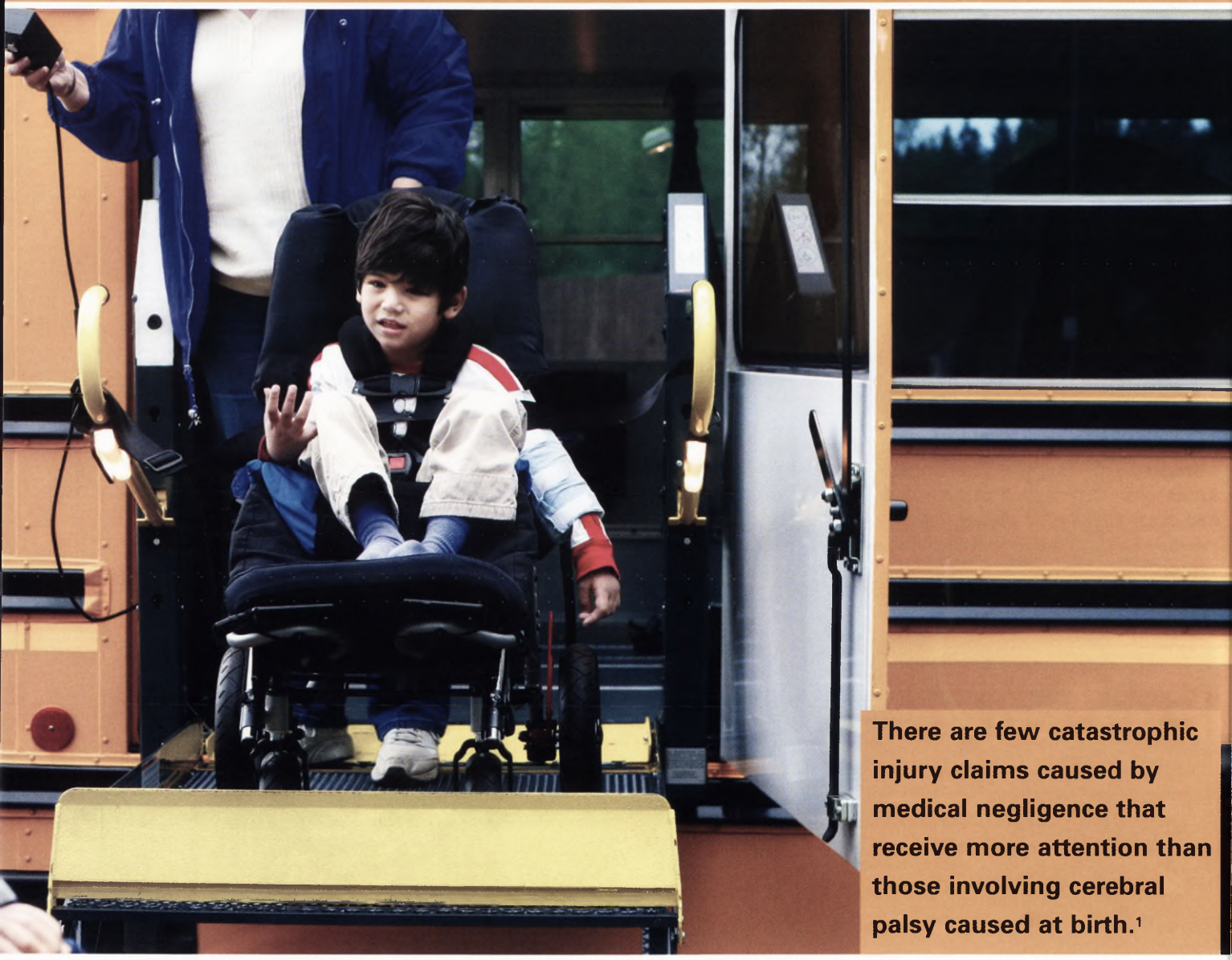
By David Hirsch

CEREBRAL PALSY CLAIMS Typical defences and how to deal with them



There are few catastrophic injury claims caused by medical negligence that receive more attention than those involving cerebral palsy caused at birth.[1]

For the family involved, significant sums are needed to ensure adequate care for the child who, depending on the quality of care provided, may have a normal or near-normal life expectancy. For doctors and their insurers, those significant sums have been blamed for increasing premiums and the resulting medical indemnity crisis that led to tort law reforms in Australia beginning in 2002. For lawyers (both plaintiff and defence), cerebral palsy (CP) litigation can be a source of significant fee income.

These last two effects have led some in the medical profession to go to great lengths to try to curtail CP litigation. Their lobbying of governments to pass tort reform legislation is well known. But the effort has also involved the use, and I would suggest also the misuse, of scientific and medical literature.

This article will examine three strategies regularly advanced by doctors in their defence of CP claims:

- the Consensus Statement on the cause of cerebral palsy;
- the utility of intrapartum cardiotachograph (CTG) monitoring; and
- decision-to-delivery time to perform a caesarean section.

BACKGROUND

A typical CP claim alleges that the child suffered a period of hypoxia (inadequate oxygen) and that this has caused brain damage. In medical terms this kind of damage is 'hypoxic ischaemic encephalopathy' or HIE. The plaintiff typically asserts that the labour was not properly managed and that signs of foetal distress were not recognised and acted upon in a timely manner.[2] The allegedly unrecognised signs of distress usually involve a failure to observe and interpret foetal heart rate abnormalities seen on CTG monitoring.[3] Ultimately, the plaintiff argues that had the abnormalities been recognised an urgent caesarean delivery would have been performed before the hypoxia became so severe as to cause HIE.

'Cerebral palsy' is often used synonymously with 'brain damage', especially in the minds of lawyers and the general public. Brain damage can, however, by caused by many things including infection, genetic abnormality, developmental abnormality, trauma and the effects of prematurity. While brain damage from these causes may include the kind of clinical picture seen in CP caused by HIE, they often do not.[4] Where CP is caused by HIE the typical presentation involves spastic quadriplegia – a movement disorder. This is caused by damage to certain parts of the brain (the basal ganglia) that are highly susceptible to injury due to acute hypoxic/ischaemic events.

THE CONSENSUS STATEMENT

The first iteration of the Consensus Statement appeared in 1995.[5] It was the product of a campaign by certain doctors, spearheaded by South Australian obstetrician and gynaecologist, Professor Alastair MacLennan, to promulgate the view that CP claims against doctors were not scientifically justified and should not be allowed.[6] Professor MacLennan's antipathy to plaintiff lawyers is well known, and the 1995 Consensus Statement unabashedly sought to direct judges how to decide cases.[7]

The second iteration of the Consensus Statement appeared in 1999.[8] This provided a 'template' to be used to determine whether an acute hypoxic/ischaemic event during labour was the cause of CP in any particular case. It was generally accepted that about 10 per cent of CP could be ascribed to acute intrapartum events.[9] The purpose of the Consensus Statement was to define further the narrow circumstances in which a case could fall within this 10 per cent.

The Consensus Statement lists three 'essential criteria' for cerebral palsy caused at birth:

1. **Metabolic acidosis**: The umbilical cord or very early neonatal blood samples should have a pH<7.0 and base deficit ≥ -12.
2. **Neurological signs**: Early onset of severe or moderate encephalopathy (like seizures) in infants of ≥ 34 weeks gestation.
3. **Cerebral palsy:** This must be either of the spastic or athetoid (dyskinetic) type.[10]

There are five further, 'non-specific' criteria that suggest birth-related cerebral palsy:

1. **A sentinel hypoxic event:** For example, uterine rupture, placental abruption or cord prolapse.
2. **Severe bradycardia:** A sudden, rapid and sustained deterioration in foetal heart rate (commonly seen with a sentinel event).
3. **Low APGAR score[11]:** Scores of six or less for more than five minutes.
4. **Early multi-system involvement:** Usually cardiac, renal or respiratory complications.
5. **Early imaging evidence:** Ultrasound or CT imaging showing brain swelling.

Metabolic acidosis

Proof of metabolic acidosis sufficient to establish the first essential criterion can be problematic. Proof requires evidence from umbilical cord blood analysis, and sometimes cord blood is not taken.

The Consensus Statement bravely asserts that *'if blood gas data are not available, it cannot be assumed from other signs that hypoxia was present at birth since these signs lack specificity either individually or as a group'*. One would be suspicious of the failure to obtain cord blood where a baby is born severely compromised since this omission would, based on the Consensus Statement, afford a strong defence against any CP claim. In practice, even if cord blood is not obtained, arterial blood gases will be – albeit at a time following resuscitation when metabolic acidosis has been treated. Those arterial blood gas results usually show pH and base excess levels that allow an expert neonatologist to infer that had cord bloods been taken, they probably would have demonstrated marked metabolic acidosis.

Of greater concern are the 'non-specific' criteria of a sentinel hypoxic event and severe bradycardia.

Sentinel events

The sentinel events referred to in the Consensus Statement include uterine rupture, placental abruption and cord prolapse. All are sudden, catastrophic events that lead to loss of blood supply to the baby. In the case of uterine rupture and placental abruption, one sees a sudden drop in maternal blood pressure. In cord prolapse, the umbilical cord typically falls through the cervix and out of the vagina, where it gets caught between the baby's head and the mother's pelvis, cutting off the blood supply to the baby. According to the Consensus Statement, *'it is only when it is apparent and detectable that it helps to define the probable timing of the event* >>

The argument that CTG monitoring does not prevent CP ignores the steady increase in the survival of premature babies, the group at highest risk of CP.

and the determination of whether its sequelae might have been preventable'.

Severe bradycardia

While sentinel events are easily observable, severe bradycardia is detectable only when one is paying attention to the foetal heart rate.

The Consensus Statement accepts that extreme abnormalities (including absence of baseline variability and persistent late or variable decelerations) are associated with metabolic acidosis, HIE and consequent CP. But it also questions the utility of CTG monitoring as a predictor of CP.

CTG monitoring

When a CP claim asserts a negligent failure to perform or interpret CTG monitoring, the defence typically argues that CTG monitoring does not prevent CP. Statistics will be presented showing that the incidence of CP in the general population has not declined since the advent of CTG monitoring over 40 years ago. This proves (according to the defence) that CTG monitoring does not prevent CP.

The argument has two fundamental flaws.

First, the conclusion is false because the argument deliberately fails to note that improvements in obstetric care have led to a steady increase in the survival of premature babies, and it is this group that is at highest risk for CP. The absolute numbers of CP cases may not have changed much, but there is now a very high number of iatrogenically caused CP in babies who would have died in earlier times.[12] Improvements in obstetric care, including regular CTG monitoring, have reduced the number of stillbirths and early neonatal deaths for term births and it is these babies (not the very premature ones) who used to survive but with CP.

Second, no sensible obstetrician believes that CTG monitoring does not identify early signs of foetal distress or is useless in preventing CP.

Those who question the utility of CTG monitoring cite a retrospective study of births in California between 1983 and 1985. The study confirmed a statistically significant association between certain foetal heart rate abnormalities (multiple late decelerations and decreased variability) and CP. But it also showed that the records of most of the normal children in the control group also showed foetal heart rate abnormalities of some kind. Extrapolation of the numbers (95 children with CP out of a population study of over 155,000 births) was said to support a finding that 99.8 per cent of foetal heart abnormalities detected during labour are false positive for CP.[13]

This study had significant limitations. First, there was no uniformity in the records reviewed and CTG monitoring was inconsistent. Many of the babies with CP did not have any CTG monitoring. Second, it was unknown whether the abnormalities seen occurred early or late in labour or how long they lasted. Third, there was no information on why the babies without CP were delivered when they were. It may be that they were delivered because foetal heart rate abnormalities were detected and action was taken. Finally, there was no information on the duration of severe bradycardia, a matter that the authors admitted was '*especially regrettable*'.

This article is a good example of how the findings of a limited study can be taken out of context and used by defendants in CP litigation to their advantage. A more rigorous examination of the article itself should counter this. Consideration of general guidelines on foetal monitoring would be useful as well.[14]

Decision to delivery time

Where a CP claim alleges a negligent failure to perform a caesarean section in a timely manner, the question of a reasonable 'decision to delivery time' becomes important.

It is no small wonder that the Consensus Statement is prepared to concede that 'sentinel' events like uterine rupture, placental abruption and cord prolapse can cause CP. What all of these catastrophic events have in common is that they are unexpected and afford the obstetrician little time to deliver the baby before irretrievable hypoxic damage occurs. In other words, where causation can be proven, breach of duty cannot.

Where CTG abnormalities (prolonged late or variable decelerations of bradycardia) are acknowledged to exist and where a good argument can be advanced for the need for a caesarean delivery, the plaintiff will be met with the objection that there is an inevitable delay between recognising the need for a caesarean and actually performing one. If, by the time a caesarean should have been done the hypoxic brain damage has already occurred, then breach of duty can be proven but causation cannot.

Defendants regularly cite a study performed by Professor MacLennan and his South Australian colleagues that describes 'decision to delivery times' for caesarean section of around 60 minutes for 'urgent' cases.[15]

The purpose of the study was to 'debunk the myth' promoted by 'rogue plaintiff experts' that an urgent caesarean delivery can and therefore should be achieved within 30 minutes of the decision being made (the '30-minute rule').

In this study, however, 'urgent' was defined to include not just unquestionably urgent situations like the sentinel events described in the Consensus Statement, but also cases of questionable urgency like the vaguely defined 'non-reassuring foetal heart rate'. As it turned out, 75 per cent of the 'urgent' caesarean deliveries in his study were for 'non-reassuring

foetal heart rate' and the average median decision to delivery time for those cases was around 56 minutes. But for truly urgent cases like cord prolapse, the average median decision to delivery time was just 22 minutes. Far from undermining the '30-minute rule', this study actually vindicated it.

CONCLUSION

A typical CP claim alleging a failure to recognise and act on signs of foetal distress and delay in effecting a timely delivery by caesarean section will be met with typical defences.

The Consensus Statement was designed to limit the class of 'permissible' CP cases to those of acute intrapartum events leading to brain damage caused by HIE. The common feature of these is that the 'sentinel event' is unpredictable, sudden and catastrophic and therefore (so the argument goes) not preventable, even with the exercise of reasonable care.

For those cases where there is no obvious sentinel event but rather foetal heart rate abnormalities on CTG monitoring that mandate urgent delivery, it is not reasonable (so the argument goes) to deliver the baby for about 60 minutes, by which time it would be too late to prevent damage caused by the hypoxic/ischaemic event that provoked the CTG signs of foetal distress.

Practitioners need to be alert to these arguments and the paucity of literature that actually supports them. A thorough understanding of the literature, together with evidence by a fair-minded neonatology expert, should assist in obtaining compensation in deserving CP claims. ■

Notes: **1** For simplicity, I will refer to all cases involving cerebral palsy allegedly caused by intrapartum events (events that took place at or around the time of birth) as 'CP claims'. **2** Mismanagement of labour is not limited to failure to recognise signs of foetal distress. Some claims, for example, involve the injudicious use of labour augmenting drugs (Oxytocin or Syntocinon) which can provoke foetal distress. Others involve negligent forceps delivery and/or failure to perform urgent caesarean after a failed forceps delivery (See *Simpson v Diamond* [2001] NSWSC 925; *Ren v Mukerjee* [1996] ACTSC 119). **3** The foetal heart rate alone can be assessed through auscultation (hearing the baby's heart with a listening device). CTG monitoring shows not only heart rate but how this changes compared to maternal contractions. The CTG pattern can demonstrate actual and emerging distress. The passing of meconium can be another sign of distress, but it is non-specific and cannot be relied on alone to demonstrate actual distress. **4** For example, deafness, epilepsy, learning disabilities and autism are features usually associated with infection, genetic or developmental problems. **5** A MacLennan,' The origins of cerebral palsy – a consensus statement', *MJA*, 1995; 162: 85-90. **6** Professor MacLennan's publications include 'Who will deliver the next generation', *MJA*, 1993; 159: 261-3 and 'Only an expert witness can prevent cerebral palsy!', *O&G* Vol. 8 No. 1, Autumn 2006, 28-30. **7** The Consensus Statement was considered in the case of *Grimsey v Southern Regional Health Board* (1997) 7 Tas R 67, where Wright J observed: "A good deal of the paper is taken up with legal issues and concern for insurance ramifications leading to increased premiums from claims that cerebral palsy may be attributable to intrapartum carelessness. I have the suspicion that the document may lack the pure objectivity of painstaking scientific analysis." **8** A MacLennan, 'A template for defining a causal relation between acute intrapartum events and cerebral palsy; international consensus statement', *BMJ*, 1999; 319: 1054-9 **9** F Stanley, 'Pathways to cerebral palsy involving signs of birth asphyxia'. In: 'Cerebral palsies; epidemiology and causal pathways', *Clin Develop Med*, 2000; 151:98-108. **10** In the American version of the Consensus Statement, the spastic cerebral palsy must be quadriplegic rather than either quadriplegic or diplegic and there is a fourth essential criteria: 'Exclusion of other identifiable etiologies such as trauma, coagulation disorders, infectious conditions, or genetic disorders': http://www.acog.org/from_home/Misc/neonatalEncephalopathy.cfm. **11** An assessment of neonatal condition with a maximum score of 10/10. **12** For example, CP rates in infants under 1500gm rose from 12.1 in 1968 to 64.9 in 1985. See F Stanley, 'Trends in perinatal mortality and cerebral palsy in Western Australia, 1967 to 1985', *BMJ*, 1992 Jun 27; 304 (6843): 1658-63. **13** K Nelson, 'Uncertain value of electronic fetal monitoring in predicting cerebral palsy', *NEJM*, 1996: 334: 613-18. **14** For example, *Intrapartum Fetal Surveillance Clinical Guidelines* – Royal Australian & NZ College of Obstetricians and Gynaecologists (RANZCOG), 2006. **15** MK Spencer, AH MacLennan, 'How long does it take to deliver a baby by emergency Caesarean section?', *Aust NZ J Obstet Gynaecol*, 2001 Feb; 41(1): 7-11. The actual times were 69, 54 and 43 minutes for Level 1, 2 and 3 hospitals respectively.

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