Cerebral palsy—medicolegal aspects

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In 1862 William James Little, a London orthopaedic surgeon wrote:

> 'The object of this communication is to show that the act of birth does occasionally imprint upon the nervous and muscular systems of the nascent infantile organism very serious and peculiar evils. Nearly twenty years ago . . . I showed that premature birth, difficult labours, mechanical injuries during parturition to head and neck, where life had been saved, convulsions following the act of birth, were apt to be succeeded by a determinate affection of limbs of new-born children, spastic rigidity from asphyxia neonatorum, and assimilated it to the trismus nascentium and the universal spastic rigidity sometimes produced at later periods of existence[1].'

This condition was known as Little's disease until William Osler coined the term cerebral palsy in 1888. He too noted the association with difficult deliveries and with asphyxia requiring prolonged resuscitation[2]. In the 1890s Sigmund Freud was the first to recognize that antepartum and postpartum factors could cause a similar condition. He postulated that most cases arose from difficult birth but speculated that the birth difficulty might have been caused by some underlying condition[2]. The asphyxia theory was subsequently given impetus when research in monkeys showed that perinatal asphyxia could cause brain damage[3].

In the past 30 years, great increases in the use of fetal monitoring and caesarean section have been driven by the belief that early detection of asphyxia and speedy delivery will prevent brain damage. In parallel with these changes there has been an unprecedented rise in malpractice litigation. Surprisingly, over the same period the incidence of cerebral palsy in term infants has not changed[4,5]. This lack of impact prompted epidemiological studies which showed that asphyxia accounts for less than 10% of cases[6]. In 1999 an international consensus statement was published to provide an agreed reference for use by the courts and expert witnesses in birth injury litigation[7]. The intention was to provide a template that could be modified as new knowledge became available[8].

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ESSENTIAL CRITERIA

How can the cause of cerebral palsy be established many years after the event? This is achieved by starting with the clinical condition and working backwards. The only type of cerebral palsy associated with intrapartum hypoxia is spastic quadriplegia, especially if accompanied by dyskinesia[9]. Mental retardation, epilepsy and learning disorders are not caused by birth asphyxia unless also accompanied by spastic quadriplegia. A statement of severity should not be made before 3–4 years, because mild to moderate cerebral palsy improves in the early years and dyskinesia is not always evident before then. Abnormal tone will, however, have been noticed earlier if the dyskinesia is caused by hypoxia. Furthermore, speech and cognitive development cannot be accurately assessed before age 3–4. There are several neurodegenerative and metabolic conditions that are slowly progressive and in their early phases may mimic cerebral palsy. Where there is doubt the child may need to be seen again after an interval. Syndromes such as Lesch–Nyhan, Rett and glutaric aciduria type 1 are examples.

Cerebral palsy caused by intrapartum hypoxia is always associated with a neonatal encephalopathy and seizures[10,11]. Newborn encephalopathy is defined by Nelson and Leviton as 'a clinically defined syndrome of disturbed neurological function in the earliest days of life in the term infant, manifested by difficulty with initiating and maintaining respiration, depression of tone and reflexes, subnormal level of consciousness, and often by seizures'[12]. This definition is applicable only to term infants because feeding difficulty and abnormality of tone and reflexes are common in preterm infants.

The incidence of neonatal encephalopathy in a large study in Western Australia was 3.8/1000 term births[13]. That study identified certain preconceptional and antepartum risk factors for neonatal encephalopathy[14], shown in Box 1. In 29% there were both antepartum and intrapartum risk factors while only intrapartum factors occurred in 4.9%. Spastic quadriplegia develops in about 10% of cases of neonatal encephalopathy, all with seizures[11].

The inclusion of fetal acidaemia as an essential criterion in the consensus statement has been criticized, as pH measurements are often not available. The onus is now on maternity units to obtain that information at delivery. Acidaemia is defined as a pH <7 or base deficit >12[15,16]. A normal pH excludes hypoxic encephalopathy. By

Box 1 **Western Australian risk factors for newborn encephalopathy**
[reproduced by permission, Refs 13, 14]

Preconceptional factors*	Antepartum factors*
Increasing maternal age	Maternal thyroid disease
Unemployed, unskilled labourer or housewife	Severe pre-eclampsia
No private health insurance	Bleeding in pregnancy
Family history of seizures	Viral illness during pregnancy
Family history of neurological disorders	Post-dates pregnancy
Infertility treatment	Growth restriction in the fetus
	Placental abnormalities

*Significantly and independently associated with newborn encephalopathy in multiple logistic regression analysis (Ref. 13)

contrast, a pH <7 is associated with encephalopathy in only 10–20%[15]. The majority of severely acidotic infants, born with a base deficit >16, are also normal[16].

In response to asphyxia there is an increase in fetal bloodflow to the heart, brain and adrenals at the expense of the kidney, liver, intestines and lung. Severe ischaemia frequently causes major organ dysfunction. Evidence of organ dysfunction provides confirmation of intrapartum hypoxia. It is not, however, an essential criterion, because there are instances of intrapartum hypoxia without evidence of organ dysfunction[11,17].

NON-SPECIFIC FACTORS SUGGESTIVE OF HYPOXIA

Although meconium staining of the amniotic fluid is associated with increased risk of quadriplegic cerebral palsy, most children born with meconium in the liquor are normal[18,19]. The Apgar score, developed in 1952 by Virginia Apgar, remains a useful means of predicting neonatal survival, particularly when used in conjunction with pH values[20]. It is not, however, a sensitive method for predicting neurological outcome and was never intended for that purpose. Over 90% of infants with a 5-minute score of 0–3 will be normal[21,22]. Even among infants who do not breathe spontaneously for 20 minutes three-quarters of survivors will be normal[23]. It seems that there is a fine threshold between normality and death from asphyxia.

Fetal heart rate monitoring became established in the 1970s without proper evaluation, and led to a dramatic increase in emergency caesarean sections for fetal distress. According to a recent Cochrane review, the only benefit of electronic heart rate monitoring was a reduction in neonatal seizures[24]. Fetal heart rate monitoring lacks specificity. For every case of encephalopathy with an abnormal trace there are 83 normal babies with an abnormal trace[25]. In malpractice litigation, heart trace changes consistent with asphyxia frequently give rise to the claim that an earlier caesarean section would have prevented brain damage. There is no evidence to support such a contention[6,9]. The time between the decision to perform a caesarean section and delivery has assumed importance in the medicolegal arena. A 30-minute interval is regarded as the 'gold standard'. Some hospitals have difficulty meeting that arbitrary standard, which seems to be based on what is generally achievable rather than evidence of potential harm[26].

SENTINEL HYPOXIC EVENTS

Sentinel hypoxic events are episodes of ischaemia that cause hypoxic brain injury in a neurologically intact fetus. For accurate timing and a judgment on possible sequelae, clear clinical signs are required. In addition, the fetal response to the event should be demonstrable by heart trace and pH evidence consistent with asphyxia. Such events, which seldom result in cerebral palsy, are cord prolapse, placental abruption and uterine rupture[27].

OTHER CAUSES

Certain factors such as prematurity, intrauterine growth restriction and microcephaly at birth suggest a cause other than intrapartum asphyxia[7]. If two siblings have cerebral palsy, particularly the same type, a genetic cause is likely. Multiple pregnancy is associated with an excess risk of cerebral palsy, the risk being highest if one of the fetuses had died *in utero*. Fetal coagulation disorders and maternal autoimmune disorders have been linked with cerebral palsy. The coagulopathy induced by these disorders would explain reports of placental thrombi and brain thrombi in stillbirths and neonatal deaths[28].

Infection (chorioamnionitis) is now known to be an important factor for cerebral palsy[29]. It can mimic all the essential and non-specific criteria of intrapartum birth asphyxia. In the past, many cases of cerebral palsy caused by infection were wrongly attributed to birth asphyxia[9]. There may be no history of prolonged rupture of membranes or clinical evidence of infection in the infant. Cytokines which are neurotoxic are generated by the fetus in response to infection[30]. Ultrasonography early in the neonatal period shows evidence of brain injury caused by infection[31]. The reason why the adverse effects are confined to a small minority of infants is not clear.

INVESTIGATIONS

Imaging shortly after birth is useful in that it may reveal evidence of cerebral oedema—which confirms that the cerebral insult is of recent onset. Oedema develops in 6–12 hours and clears in 4 days[7]. Radiologists do not always agree on the interpretation of CT scans: the signs of cerebral oedema may not be clearcut. Ultrasonography, which is

widely used in neonatology, is likewise open to differences in interpretation. In a recent follow-up study of normal babies, 20% had neonatal ultrasound abnormalities[32].

After the neonatal period the main value of neuroimaging is to determine whether the cerebral palsy is caused by a developmental brain abnormality, intrauterine infection or some other congenital abnormality[33,34]. Since many children with cerebral palsy have brain malformations, neuroimaging is an essential part of legal proceedings[35]. MR imaging in the infant is now a good predictor of future neurological status, but when used many years later is not reliable in determining the cause or timing of a brain insult[8].

LIFE EXPECTANCY

The most important factors determining life expectancy are the degree of mental retardation, mobility and the ability to feed. Population-based cerebral palsy registers have been used to gather information about life expectancy. Differences in populations, data collection methods and definitions have resulted in wide variation between studies. The median survival for children who are immobile and tube-fed is about 7 years[36]. 80% of children with severe cognitive and ambulatory impairment survive to 18, most to 35 and beyond[37]. Life expectancy can be most accurately assessed by an individualized statistical assessment rather than by simply matching degree of disability with outcome in the published studies (Strauss D, personal communication).

CONCLUSION

From Little's observations in the middle of the 19th century, a childbirth litigation industry has been born. We now know that birth asphyxia accounts for only a small percentage of this poorly understood condition. Meanwhile the cost of medical negligence payments has risen sharply[38]. Payments to children with cerebral palsy are some of the largest. These payments now regularly exceed £3 million, and in some cases lawyers' fees exceed the award. In Ireland a child was recently awarded £2.1 million, with legal fees almost double at £4 million[39]. With the National Health Service currently facing a medical negligence bill of £2.6 billion, would not a no-fault compensation scheme be kinder to families and better for the Treasury?[40]

REFERENCES

1 Little WJ. On the influence of abnormal parturition, difficult labours, premature births, and asphyxia neonatorum, on the mental and physical condition of the child, especially in relation to deformities. *Trans Obstet Soc Lond* 1862;**3**:293–344

2 Schifrin B, Longo L. William John Little and cerebral palsy. A reappraisal. *European J Obstet Gynecol* 2000;**90**:139–44

3 Myers R. Two patterns of perinatal brain damage and their conditions of occurrence. *Am J Obstet Gynecol* 1972;**112**:246–76

4 Colver A, Gibson M, Hey E, *et al*. Increasing rates of cerebral palsy across the severity spectrum in north-east England 1964–1993. *Arch Dis Child Fetal Neonatal Edn* 2000;**83**:F7–F12

5 Hagberg G, Hagberg G, Beckung E, Uvebrant P. Changing panorama of cerebral palsy in Sweden. VIII. Prevalence and origin in the birth year period 1991–94. *Acta Paediatr* 2001;**90**:271–7

6 Stanley F, Blair E, Alberman E. Pathways to cerebral palsy involving birth asphyxia. *Clin Devel Med* 2000;**151**:98–108

7 MacLennan A. A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement. *BMJ* 1999;**319**:1054–9

8 MacLennan A. A guest editorial from abroad: medicolegal opinion—time for peer review. *Obstet Gynecol Surv* 2001;**56**:121–3

9 Nelson K. The neurologically impaired child and alleged malpractice at birth. *Neurolog Clin* 1999;**17**:283–93

10 Levene M, Sands C, Grindulis H, Moore J. Comparison of two methods of predicting outcome in perinatal asphyxia. *Lancet* 1986;**1**:67–9

11 Evans K, Rigby A, Hamilton P, *et al*. The relationships between neonatal encephalopathy and cerebral palsy: a cohort study. *J Obstet Gynaecol* 2001;**21**:114–20

12 Nelson K, Leviton A. How much of neonatal encephalopathy is due to birth asphyxia? *Am J Dis Child* 1991;**145**:1325–31

13 Badawi N, Kurinczuk J, Keogh J, *et al*. Antepartum risk factors for neonatal encephalopathy: the Western Australian case control study. *BMJ* 1998;**317**:1549–53

14 Badawi N, Keogh J. Antepartum and intrapartum risk factors for newborn encephalopathy. *Contemp Ob/Gyn* 2001;**2**:26–54

15 Goodwin T. Clinical implications of perinatal depression. *Obstet Gynecol Clin Am* 1999;**26**:711–23

16 Low J. Intrapartum Fetal Surveillance. Is it worthwhile? *Obstet Gynecol Clin N Am* 1999;**26**:725–39

17 Phelan J, Ahn M, Korst L, *et al*. Intrapartum fetal asphyxial brain injury with absent multiorgan system dysfunction. *J Maternal-Fetal Med* 1998;**7**:19–22

18 Naeye R. Can meconium in the amniotic fluid injure the fetal brain? *Obstet Gynecol* 1995;**86**:720–4

19 Yeomans E, Gilstrap L, Leveno K, Burris J. Meconium in the amniotic fluid and fetal acid–base status. *Obstet Gynecol* 1989;**73**:175–8

20 Casey B, McIntyre D, Leveno K. The continuing value of the Apgar score for the assessment of newborn infants. *N Engl J Med* 2001;**344**:467–71

21 Nelson K, Ellenberg J. Apgar scores as predictors of chronic neurologic disability. *Pediatrics* 1981;**68**:36–44

22 Moster D, Lie R, Irgens L, *et al*. The association of Apgar score with subsequent death and cerebral palsy: A population-based study in term infants. *J Pediatr* 2001;**138**:798–803

23 Scott H. Outcome of very severe birth asphyxia. *Arch Dis Child* 1976;**51**:712–16

24 Thacker S, Stroup D, Chang M. Continuous electronic heart rate monitoring for fetal assessment during labour (Cochrane Review). In: *The Cochrane Library*, Issue 2. Oxford: Update Software, 2001

25 Spencer A, Badawi N, Burton P, *et al*. The intrapartum CTG prior to neonatal encephalopathy at term: a case–control study. *Br J Obstet Gynaecol* 1997;**104**:25–8

26 James D. Caesarean section for fetal distress. The 30 minute yardstick is in danger of becoming a rod for our backs. *BMJ* 2001;**322**:1316–17

27 Nelson K, Grether J. Potentially asphyxiating conditions and spastic cerebral palsy in infants of normal birth weight. *Am J Obstet Gynecol* 1998;**179**:507–13

28 Nelson K, Grether J. Causes of cerebral palsy. *Curr Opin Pediatr* 1999;**11**:487–91

29 Wu Y, Colford J. Chorioamnionitis as a risk factor for cerebral palsy. A meta-analysis. *JAMA* 2000;**284**:1417–24

30 Yoon B, Romero R, Park J, *et al.* Fetal exposure to an intra-amniotic inflammation and the development of cerebral palsy at the age of three years. *Am J Obstet Gynecol* 2000;**182**:675–81

31 De Felice Toti P, Laurini R, *et al.* Early neonatal brain injury in histologic chorioamnionitis. *J Pediatr* 2001;**138**:101–4

32 Haataja L, Mercuri E, Cowan F, Dubowitz L. Cranial ultrasound abnormalities in full term infants in a postnatal ward: outcome at 12 and 18 months. *Arch Dis Child Fetal Neonatal Ed* 2000;**82**:F128–33

33 Sugimoto T, Woo M, Nishida N, *et al.* When do brain abnormalities in cerebral palsy occur? An MRI study. *Devel Med Child Neurol* 1955;**37**:285–92

34 Hoon A, Reinhardt E, Kelley R, *et al.* Brain magnetic resonance imaging in suspected extrapyramidal cerebral palsy: observations in distinguishing genetic–metabolic from acquired causes. *J Pediatr* 1997;**131**:240–5

35 Croen L, Grether J, Curry C, Nelson K. Congenital abnormalities among children with cerebral palsy: more evidence for prenatal antecedents. *J Pediatr* 2001;**138**:804–10

36 Eyman R, Grossman H. Living with cerebral palsy and tube feeding. *J Pediatr* 2001;**138**:47

37 Hutton J, Colver A, Mackie P. Effect of severity of disability on survival in northeast England cerebral palsy cohort. *Arch Dis Child* 2000;**83**:468–74

38 Dyer C. NHS faces rise in negligence payments. *BMJ* 2001;**323**:11

39 Birchard K. No-fault awards for babies with cerebral palsy in Ireland? *Lancet* 2000;**356**:664

40 Ferryman A. NHS faces medical negligence bill of £2.6bn. *BMJ* 2001;**322**:108