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CEREBRAL PALSY AND CLINICAL NEGLIGENCE

**THE MEDICO-LEGAL ISSUES INVOLVED
IN ESTABLISHING AND
QUANTIFYING A CLAIM**

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CLINICAL NEGLIGENCE CLAIMS : AN OVERVIEW

NEGLIGENCE?

LEGAL REQUIREMENTS

We must demonstrate that:

- (a) The hospital owed mother/baby a duty of care;
- (b) That the hospital breached that duty of care;
- (c) That the breach caused the damage in respect of which we are claiming compensation.

THE DUTY OF CARE

There is rarely an issue about the existence of a duty of care. In particular, in these sorts of cases there will be a duty of care owed by:

- the midwife
- the obstetrician
- the paediatrician

BREACH OF DUTY

This will involve expert evidence. The experts will be asked to give an opinion on what the requirements of reasonably competent contemporary practice were in the particular circumstances of the case in question and the extent to which the treatment/management of the patient fell below that standard.

In the context of clinical negligence, the test is that set out in the Bolam case.

"Where you get a situation which involves the use of some special skill or competence... The test is the standard of the ordinary skilled man exercising and professing to have that special skill. A man need not possess the highest expert skill; it is well established law that it is sufficient if he exercises the ordinary skill of an ordinary competent man exercising that particular art.

A practitioner is not negligent if he has acted in accordance with a practice accepted as proper by a responsible body of medical men skilled in that particular area. A man is not negligent if he is acting in accordance with such a practice, merely, because there is a body of opinion which would take a contrary view."

Bolam v Friern Hospital Management Committee (1957) 2 All ER 118

Possibly the second most often quoted case in this particular area of law is Maynard v West Midlands Health Authority (1984) 1 WLR 634 in which Lord Scarman said:

"It is not enough to show that there is a body of competent professional opinion which considers that theirs was a wrong decision if there also exists a body of professional opinion, equally competent, which supports the decision as reasonable in the circumstances. It is not enough to show that subsequent events show that the operation need never have been performed if at the time the decision to operate was taken it was reasonable in the sense that a responsible body of medical opinion would have accepted it as proper"

The following year in the Siddaway case the same Judge, Lord Scarman, continued this theme when he said:

"The Bolam principle may be formulated as a rule that a doctor is not negligent if he acts in accordance with a practice accepted at the time as proper by a responsible body of medical opinion, even though other doctors adopt a different practice. In short, the law imposes the duty of care: but the standard of care is a matter of medical judgment."

*Siddaway v Board of Governors of Bethlem and Maudsley Hospital
(1985) 1 All ER 643*

In the case of Bolitho v City of Hackney Health Authority (1998) AC 232, the facts of which I shall discuss later, the House of Lords, and in particular Lord Browne-Wilkinson, refined the 'body of opinion' defence. In doing so, he referred back to the Bolam case and pointed out that the Judge in that case had prefaced the reference to a 'body of opinion' with the words 'reasonable, 'responsible'. In the Maynard case he noted that Lord Scarman had referred to a 'respectable body of opinion'.

Having made these observations, Lord Browne-Wilkinson concluded that it was open to a Judge to test the reasonableness of a 'body of opinion' and if found wanting to reject it.

"... the Court is not bound to hold that a defendant doctor escapes liability for negligent treatment or diagnosis just because he leads evidence from a number of medical experts who are genuinely of the opinion that the defendants' treatment or diagnosis accorded with sound medical practice ... The Court has to be satisfied that the exponents of the body of opinion relied upon can demonstrate that such opinion has a logical basis."

This reasoning was applied by the Judge in the case of Marriott v West Midlands Health Authority (1999) Lloyds Rep Med 23 and endorsed by the Court of Appeal.

In that case, Mr Marriott had fallen down some stairs suffering a head injury. He was taken to hospital for x-rays and neurological observations and released home the next day. He remained lethargic with no appetite and had headaches. One

week later, with no improvement in his condition, the GP was called out. The GP took a history, examined Mr Marriott and advised Mrs Marriott that if her husband deteriorated she should call him (the GP) back. Four days later, Mr Marriott's condition suddenly deteriorated. He was rushed to hospital where he was found to have suffered a linear fracture of the skull and bleeding from an artery in the brain. Had he been admitted to hospital when the GP called, the problems would, more likely than not, have been identified and treated. The sudden collapse (which caused hemiplegia, dysarthria and severe disablement) would have been avoided.

The GP's defence was that a body of opinion supported his decision to leave him at home. The Judge refused to accept the GP's expert evidence on that point, saying that she was entitled to find it could not be a reasonable exercise of a GP's discretion to leave a patient at home in these circumstances.

Penney v East Kent Health Authority (2000) Lloyds Rep Med 41. Given that it was agreed by all the experts that the cervical smear slides contained abnormalities that ought to have been observed there could be no logical justification for the Defendants' experts view that the cytoscreener was nonetheless justified in classifying the slides as negative.

EXAMPLES OF BREACH:

- **Midwife**
 - **Failure to interpret** (eg: not correctly identify potential problem on CTG)
 - **Failure to communicate** (eg: on realising the problem, did not pass on to obstetrician)
- **Obstetrician:**
 - **Failure to deliver baby at right time**
 - **CTG traces**
 - **Fetal blood sampling**
 - **Trial of labour:** Previous birth had been very difficult – a long labour converted to Caesarean section. Mother noted to have small pelvic outlet (recorded in notes). Birth Plan detailed Trial of Labour. In fact, labour allowed to go on for several hours with no real progress. Baby became

distressed and eventually converted to a Caesarean section but too late to avoid serious damage.

- **Failure to manage labour correctly**
 - **Abuse of Syntocinon:** Quite often, labour will not progress as fast as hoped. This can be augmented by Syntocinon, administered via a drip, at a rate which normally increases until uterine activity is stimulated. Potential problems:
 - Baby squeezed by uterus but if cervix remains stubbornly closed, in extreme cases can cause brain damage.
 - Can risk hyper-stimulation of the uterus and, in certain cases, lead to rupture of the uterus with devastating consequences for both mother and baby.
 - **Prophylactic antibiotics.**
- **Paediatrician:**
 - Failure to incorrectly intubate
 - Failure to transfer to ITU

CAUSATION

It is not sufficient to establish that there was a breach of duty: that breach of duty must have caused the relevant damage.

It is often a very difficult issue in birth injury cases. Indeed, I have had several cases in which breach of duty is admitted and causation has been the main issue in dispute.

A defendant may well accept that the baby should, for example, have been delivered earlier but at the same time argue that delivery at that earlier time would not have seen a happier outcome.

The issue of causation is also often a matter for expert evidence. The Court must determine:

- what would the condition of the baby have been but for the negligence?
- what would the condition of the baby have been if proper management had taken place?

Sometimes, the causation question can be factual: what treatment would have been provided or what management would have been pursued if there had been no breach of duty. In such a case, the Court will be obliged to make a finding of fact in relation to the conduct of those likely to have been involved; and if it be the case that the treatment/management likely to have been provided/pursued would have made no difference to the outcome, causation will not be established unless such course of treatment/management would itself have been a breach of duty.

Perhaps the most frustrating cases of all are those where there is an established/admitted breach of duty but no evidence that the outcome for the baby would have been any different.

To try and explain why establishing causation can be so difficult, we need to understand a little more about the cause of brain damage to the newborn and why, despite increased levels of care and higher rates of intervention, the numbers of children with cerebral palsy remain much as they ever have done.

HOW IS THE NEWBORN BRAIN DAMAGED?

Brain damage leading to cerebral palsy and neurological impairment in later life may be caused by a number of different basic mechanisms. These include:

➤ Congenital malformation of the brain

This appears to be a relatively common form of brain injury although it remains very poorly understood. The highly complex process of brain development (which is particularly concentrated in the first three months of pregnancy) may be disturbed leading to a permanent abnormality of the brain. There may be an obvious gross abnormality of the brain structure (which would be visible on a magnetic resonance brain scan, for instance), or the abnormality may be limited to the microscopic structure and function of the brain. This form of brain injury is now thought to be the commonest cause of cerebral palsy.

➤ Infection

The foetus may become infected within the uterus, leading to permanent brain damage. This is particularly likely with congenital infections such as toxoplasmosis, rubella and cytomegalovirus. The infection spreads throughout the whole foetus and usually leads to a combination of clinical signs such as eye abnormalities, reduced fetal growth of head and body, and liver enlargement. The diagnosis is confirmed by blood and urine tests after birth. Acute infection of the brain may also occur in the newborn period (neonatal meningitis and/or encephalitis) leading to permanent brain injury.

➤ Mechanical birth trauma

As the head passes through the birth canal it is exposed to large mechanical forces which may cause tearing and rupture of delicate structures within the cranial cavity. This form of brain injury is now very rare in developed countries due to improved obstetric care. It is diagnosed by characteristic patterns of intracranial haemorrhage, often occurring over the surface of the brain, which may be detected by brain scanning after birth.

➤ Intraventricular/periventricular haemorrhage

This refers to haemorrhage within and around the cerebral ventricles, which are fluid-filled cavities in the centre of the brain. This form of haemorrhage is very rare in babies born at full-term, but is common in the very pre-term infant and may lead to permanent brain injury. Although the haemorrhage may occur before birth, it usually occurs in the first week after birth in very pre-term babies who are undergoing intensive care.

➤ Hypoxic-ischaemia

Birth asphyxia has two elements:

- (a) hypoxia, which means a reduction in oxygen available to the tissues of the brain;
- (b) ischaemia, which means a reduction in blood going to the tissues of the brain.

Asphyxia can be either acute (severe but short lasting) or chronic (a lesser reduction in supply of oxygenated blood over a longer period). Causes of acute asphyxia would include cord prolapse or placental abruption. Examples of chronic asphyxia would include cases of placental deficiency.

The brain depends upon a constant supply of oxygen and nutrients, principally glucose, in the blood stream. It also requires the constant removal of waste products, principally carbon dioxide. In practice, shortage of oxygen and blood supply are inextricably intertwined and the pathological process is usually described as hypoxic-ischaemia. Total interruption of the oxygen and blood supply for a few minutes, or partial interruption of several hours frequently leads to permanent brain injury or death. Hypoxic-ischaemia may occur before labour (for example due to compression of the umbilical cord) or after delivery in the newborn period, (for example due to the obstruction of an airway). Acute hypoxic-ischaemia of sufficient severity to cause permanent injury is usually followed by a period of profound neurological abnormalities (hypoxic ischaemic encephalopathy) which commence within 24 hours and last for several days.

There are four conditions that have to be met before cerebral palsy can reasonably be attributed to perinatal hypoxia:

- (1) There must be evidence of prolonged or profound foetal distress during labour.
- (2) The baby must be in a poor condition at birth - having poor Apgar scores, preferably with evidence of acidosis and requiring prolonged resuscitation.
- (3) There must be signs of hypoxic ischaemic encephalopathy in the first days of life.
- (4) Other possible causes should have been ruled out as far as possible.

- (1) Evidence of prolonged or profound foetal distress can be found by:
- (a) recording the foetal heart rate (primarily using a CTG monitor);
 - (b) examination of the liquor for the presence of meconium;
 - (c) testing a sample of foetal blood for acidity (pH);
- (2) Evidence of poor condition:
- (a) Apgar scores

| | | | | TOTAL |
|--------------------|---------|----------------------------|-----------------|-------|
| SIGN | 0 | 1 | 2 | 0-2 |
| HEART RATE | Absent | Slow <100 | >100 | |
| RESPIRATORY EFFORT | Absent | Slow irregular | Good cries | |
| MUSCLE TONE | Flaccid | Some flexing extremities | Active movement | |
| REFLEXES | Nil | Grimace | Cry | |
| COLOUR | Blue | Pink body blue extremities | All pink | |
| | | | TOTAL | 0-10 |

(b) Evidence of acidosis

The human body burns glucose to produce energy for its metabolic activity. Waste products from the energy producing metabolism produce carbon dioxide gas and carbonic acid chemical particles. The carbon dioxide gas is expelled through the lungs during respiration, while the carbonic acid particles are 'buffered' with chemical alkaline substances and carried by the body's internal blood circulation to be excreted by the kidneys. It is possible to check the blood of a newborn baby to determine the amounts of carbon dioxide or buffering capacity present at the time of birth (by an umbilical cord blood analysis) or later (by an arterial blood analysis). If the baby has been exposed to perinatal asphyxia, blood gas test result will reflect the metabolic results of the oxygen deprivation. There are three basic components of the blood gas analysis, namely:

- (1) the acid-based balance (pH)
- (2) carbon dioxide content (PCO_2)
- (3) bicarbonate (HCO_3)

It is the relationship between the carbon dioxide concentration and the bicarbonate buffer concentration which determines the acid-based balance of the blood, the pH. A normal pH measurement for a newborn who has not been exposed to perinatal asphyxia is in the region of 7.32 to 7.42. Readings below the normal range indicate that the foetus has been deprived of oxygen.

(3) Hypoxic ischaemic encephalopathy

An encephalopathy refers to a range of abnormal neurological signs. An encephalopathy can be graded from:

- Mild - some abnormal signs of the newborn recovering within 48 hours
- Moderate - some convulsions and irritableness - 1 in 4 such babies will eventually suffer from cerebral palsy
- Severe - the newborn is in a coma, suffering from seizures and, if survives, will almost certainly develop cerebral palsy

In addition to neurological signs, one can expect evidence of other organ involvement and particularly the kidneys. A warning sign to note is a newborn's failure to pass urine.

EPIDEMIOLOGY OF CEREBRAL PALSY

The epidemiology of cerebral palsy has been reviewed extensively by *Paneth and Stark (1983)* and *Stanley and Alberman (1984)*. Its frequency rates in industrial countries have remained relatively constant and average approximately 2 to 2.5 cases per 1,000 school-aged children. The proportion of cerebral palsy that is related to birth asphyxia is estimated to range between 10% and 25% in several large independent studies. Importantly, the majority of asphyxiated infants do not develop cerebral palsy; however, risks increase with decreasing birth weight and gestational age (*Ellenberg and Nelson, 1979; Paneth and Stark, 1983; Pharoah and colleagues, 1990*). By way of example, for infants who are born weighing less than 1,500g, the risk for cerebral palsy is approximately 10%, or 50 times the risk for term infants (*Stanley and Alberman 1984*).

INVESTIGATION

Images of the brain taken by one of a variety of methods:

- Ultrasound
- CT scan
- MRI scan

can provide evidence of the timing and nature of the hypoxic ischaemic insult.

Damage in the watershed region of the brain will indicate a prolonged period of chronic partial hypoxia.

Damage to areas of the brain such as the thalamus or basal ganglia will evidence damage caused by a shorter period of acute hypoxia.

A series of ultrasound scans can show development and resolution of swelling caused by an insult and help with the timing of that insult.

LITIGATING CEREBRAL PALSY CLAIMS

- 1 Obtain preliminary instructions:
 - in the office
 - at family homeand prepare first draft liability statement

- 2 Sort out funding
 - at present, Legal Aid on child's means

- 3 Obtain medical notes
 - obstetric
 - paediatric

- 4 Pagination/indexing/preliminary assessment of medical notes

- 5 Review liability statements for parents (and other *key* witnesses)

- 6 Instruct initial experts
 - obstetrician
 - paediatricianto consider breach of duty

- 7 Review expert reports and discuss with family. If favourable, review funding and increase cover.

- 8 Instruct further experts on causation
 - paediatric neurologist
 - paediatric neuroradiologist

- 9 First Instructions to Counsel - initial conference with key experts and family
 - Review evidence and consider strengths and weaknesses
 - Identify litigation strategy
 - Split trial?

- 10 Proceedings issued in High Court

- 11 Initial quantum statements of fact

- 12 If no split trial, instruct quantum experts (eg: care, aids and appliances, accommodation, employment, etc)

- 13 Prepare Schedule of Special Damages of past (identifiable) loss and estimate of future expenses

- 14 Receive Defence
 - Fix timetable for litigation

- 15 Exchange Witness Statements and experts reports on
 - liability and causation
 - quantum

- 16 Prepare for trial
 - negotiations
 - post-trial/settlement

- 17 Attend trial with leading and junior Counsel

DAMAGES

Traditionally been a lump sum. In cerebral palsy cases, this can be in the region of £2m to £4m.

Sometimes we are able to agree that, in return for some of the capital, the NHS will pay an inflation-linked annuity to meet future costs. That has been the basis on which I have settled several of these cases.

The Rules have recently changed so that, for the first time, the Courts have been given the power to order periodical payments. The parties are obliged to consider the appropriateness of periodical payments at the earliest opportunity. There is no longer any minimum figure for future loss claims for this issue to be on the Agenda.

MAKING AMENDS/NHS REDRESS

This was the report issued in June 2003 by the Government's Chief Medical Officer in which proposals were set out for possible reform of clinical negligence in the NHS.

- There was a specific recommendation in relation to cerebral palsy claims. In fact, in the NHS Redress Bill, which was published a few weeks ago, this proposed was dropped.

GLOSSARY

| | |
|------------------------------------|--|
| CTG | <i>Cardiotocograph</i> : Trace; ie: a monitor used to record uterine contractions and fetal heart rate |
| Afebrile | Normal temperature |
| APGAR score | A subjective assessment of the neonate's condition arrived at by scoring 0-2 on five different tests: <ul style="list-style-type: none">• colour• tone• heart rate• respiration• reflex response |
| Auscultation | Listening (to fetal heart rate) |
| Baseline | The average fetal heart rate |
| Bradycardia | Very low heart rate |
| Cerebral palsy | A lesion of the brain sustained <2 years of age leading to a non-progressive motor disorder. |
| Decelerations (early) | Nadir of heart rate deceleration coincides with peak of contraction. |
| Decelerations (late) | Nadir of heart rate deceleration follows after peak of contraction. |
| Encephalopathy | A group of neurological signs |
| Febrile | High temperature |
| Fetal scalp electrode (FSE) | Used to get better recording of fetal heart rate if abdominal transducer not working well (still records on CTG trace). |
| Fetal scalp pH estimation | A sample of blood from baby's head to assess degree of acidosis (lack of oxygen). |
| FHR | Fetal heart rate |
| First stage labour | Onset of contractions until full dilatation of cervix (9-10cms) |
| Hypoxia | Lack of oxygen |

| | |
|---|--|
| Hypoxic Ischaemic Encephalopathy (HIE) | A key term used to describe an infant who is suffering from a neurological disorder as a result of reduced oxygen carried in the blood supply |
| Intrapartum | During labour. |
| Ischemia | Reduced blood supply |
| MRI scan | (Nuclear) Magnetic Resonance Imaging |
| Multi parous | A mother who has had more than one pregnancy. |
| Partogram | A3 record of labour. |
| Persistent fetal circulation | When in the uterus the bby's blood flows from the heart out of the descending aorta and around the body. The blood does not get its oxygen from fetal lungs so that initial 'circuit' from heart-lungs-heart and then around the body is not required. For some babies – particularly premature babies – their system does not change and hence PFC leading to unoxygenated blood circulating. |
| pH level | Acid/base balance. In birth hypoxia cases a figure of >7 will suggest acidosis of the blood. |
| Pinard's stethoscope | A type of listening trumpet used when CTG not available/required |
| Placental abruption | Placenta comes away from uterine wall – a potentially fatal complication in the absence of swift action. |
| Prima gravida | A mother giving birth to her first child. |
| Second stage labour | Full dilatation of cervix until delivery. |
| Silent pattern | Variability in baseline of <5 bpm |
| Sinusoidal pattern | Very rare slow wave. Very serious. Baby anaemic/shocked. |
| Tachycardia | Very high heart rate |
| Third stage of labour | Delivery of infant until delivery of placenta. |