

# MINUTE BY MINUTE

## SUZANNE FARG ON APPORTIONING INJURY WHERE A BABY'S OXYGEN IS RESTRICTED DURING BIRTH

**A High Court decision by Mr Justice Ritchie in January 2023 considered the scientific basis for causation of brain damage in birth injury claims; and whether damages could be apportioned when the cause of the injury was a combination of negligent and non-negligent factors.**

The events in *CNZ v (1) Royal Bath Hospitals NHS Foundation Trust, (2) The Secretary of State for Health and Social Care* [2023] EWHC 19 (KB) dated back to 1996.

The claimant was one of twins born at Royal Bath Hospital. Her twin sister was born first by vaginal delivery. After this, labour slowed and there was a negligent delay in performing a caesarean section to deliver the claimant. Prior to birth, the umbilical cord became compressed, causing the claimant to experience a period of lack of oxygen, described as acute profound hypoxic ischaemia (PHI), which injured her brain. She developed cerebral palsy affecting all four limbs.

The negligent delay in delivery related, in part, to failures in the process of advising and obtaining the mother's consent on appropriate methods for expediting birth. The judgment provides interesting commentary on the factors affecting the standard of informed consent in historic cases.

The judge found that, in 1996, the modern test (as per the Supreme Court decision in *Montgomery v Lanarkshire Health Board* [2015] UKSC 11) applied; and that without an indication from the Supreme Court, he could not substitute a watered down version to take into account changing levels of medical paternalism over time.

Having established that there was a negligent delay in delivery, causation was considered.

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## Mechanism of injury

The claimant was born one hour after her twin sister. She had no heartbeat and was not breathing. Her cord blood gas tests showed that she had metabolic acidosis, associated with lack of oxygen. She was resuscitated and by approximately 3.5 minutes after delivery, she had a heartrate over 100bpm. Her subsequent MRI brain scan showed injuries consistent with having suffered a period of acute PHI lasting between 15-20 minutes.

It was accepted that a baby's body can usually compensate for the first 10 minutes of acute PHI. After that, brain damage starts to occur, beginning with the areas of the brain with the highest metabolic demands. The deep grey matter including the basal ganglia is damaged first, followed by the cortex, and then spreading to the rest of the brain.

The damage is not wholly sequential and overlap can occur; however, the overall picture is of incremental damage occurring over time. The longer the duration of acute PHI the more damage will be sustained. Precisely how this translates into a relationship between the duration of damaging acute PHI and the extent of the claimant's functional outcome was a contested issue.

## 'But for' causation

It was held that the claimant should have been delivered between 00.55 and 00.58, and her resuscitation would have been 1-2 minutes shorter. She had experienced a period of 14-18 minutes of acute PHI, of which 4-8 (midpoint 6) minutes were damaging.

On the balance of probabilities, had the claimant been born between 00.55 and 00.58, she would have avoided all of the damaging acute PHI. Therefore, causation of the whole of the claimant's brain injury and her consequent disabilities was established on the ordinary 'but for' test.

## Material contribution and apportionment

The judge acknowledged, however, that if the extremes of his ranges for time of delivery and duration of acute PHI were applied, then only part of the damaging acute PHI would be due to negligence. In this scenario, it would be necessary to consider whether the claimant's injury could be apportioned, such that damages were awarded only to the elements of her injury caused by negligence.

The claimant's paediatric neurology expert considered that every additional minute of damaging acute PHI caused increasing disadvantage to the claimant's ability to perform activities of daily life. But it was not possible to define the functional impairment caused by each minute of acute PHI.

The defendant's expert opined that it was possible to identify a difference in functional outcome by reference to duration of acute PHI, in some circumstances. He described 5 minute 'aliquots' of acute PHI causing different levels of functional disability in connection with mobility, speech and cognition. In summary:

- 10-15 minutes: Mild to moderate disabilities arise affecting motor control but independent mobility, ability to eat and speak are retained and cognition is preserved.
- 15-20 minutes: Moderate to severe disabilities arise including severe movement limitation, significantly impaired hand function, severe dysarthria, and some cognitive impairment.
- 20-25 minutes: Severe profound disabilities arise including immobility, wholly dependent for care, significant cognitive impairment, and often requires gastrostomy feeding.

On hearing the evidence, it was found that the claimant's brain damage was a divisible injury. The damage was dose dependent: the longer the duration of acute PHI, the greater the damage that occurs and the worse the functional outcome. However, the test of material contribution could be applied if the claimant's functional disabilities arising from that damage were indivisible based on current scientific knowledge.

The judge considered whether the aliquot theory could be used to apportion the claimant's disabilities where she had experienced eight minutes of acute PHI, of which five minutes were caused by negligence, and up to three minutes would have occurred in any event.

He found that 'on logical analysis the Aliquot theory falls apart'. It could lead to cases involving longer periods of negligent delay having damages apportioned (where the negligent and non-negligent periods of acute PHI fell in different aliquots) and receiving less compensation, as compared with cases involving shorter delay periods that could not be apportioned.

Further, there would be difficulty in apportioning the plethora of possible disabilities because these are not comprehensively described in the aliquots (for example, the aliquots do not include reference to impairments of vision, hearing, continence or behaviour).

Most significantly, the judge found that the scientific basis of the evidence was insufficient to support apportionment. He noted that a paper published in 2011 had called for a database of cases of cerebral palsy, correlating the duration of acute PHI experienced with the extent of functional disabilities; but the database had not been created and scientific knowledge, as it currently stands, did not allow the Court to identify the functional effect of each minute of acute damaging PHI.

Ritchie J found that: '... the Aliquot theory, honestly and helpfully put forwards, as it was, by an impressive and experienced expert, is not an acceptable, fair or practicable way to apportion quantum in this Cerebral Palsy case caused by acute PHI'. Even if only part of the period of damaging acute PHI in the claimant's case had been due to negligence, her functional disabilities were indivisible and so she would recover damages for the whole of her injury, under the principles of material contribution.

## Comment

This case highlights the importance of defining the claimant's injury when analysing causation. Although a relationship between duration of acute PHI and damage to the brain could be established, the Court looked beyond that to whether the scientific literature described a sufficiently precise relationship with the resulting functional disabilities.

The judgment does not rule out the possibility of apportionment in future cerebral palsy cases, but this is likely to be challenging without further scientific data.

*Suzanne Farg is a senior associate at Fieldfisher*

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