

Neutral Citation Number: [2006] EWCA Civ 1028

Case No: B3/2003/1176

IN THE SUPREME COURT OF JUDICATURE
COURT OF APPEAL (CIVIL DIVISION)
ON APPEAL FROM QUEEN'S BENCH DIVISION
OXFORD DISTRICT REGISTRY
His Honour Judge Charles Harris QC
1996 T 609

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 19/07/2006

Before:

THE PRESIDENT
LADY JUSTICE ARDEN DBE
and
LORD JUSTICE WALL

Between:

ARPAD TOTH
- and -
DAVID MICHAEL JARMAN

Appellant

Respondent

George Pulman QC and Clive Rawlings (instructed by Messrs Radcliffes Lebrasseur Solicitors) for the Appellant
Miss Mary O'Rourke (instructed by The Medical Defence Union) for the Respondent

Hearing dates: 7th and 8th March 2006

Judgment

Sir Mark Potter, P:

Introduction:

1. This is a judgment of the court, to which all members have contributed, upon the appeal by the claimant, Mr Toth from the judgment of His Honour Judge Charles Harris QC sitting as a Deputy High Court Judge in the Oxford District Registry dated 9 May 2003. The action before the judge was an unusual one in which Mr Toth sought damages for nervous shock and psychiatric injury which he alleged had been caused to him by the defendant, who was a medical practitioner, by his negligent treatment of Mr Toth's five year old son Wilfred in October 1993 almost ten years before.
2. The defendant was called to Mr Toth's house one morning in order to give emergency treatment to Wilfred who had suffered a hypoglycaemic attack and was unconscious. The defendant failed to administer an intravenous glucose injection and the judge held that he was negligent in that respect. Wilfred only received such an injection following his admission to hospital over an hour later. He never recovered consciousness and died the following week when the ventilator unit utilised to keep him alive was turned off on the grounds that he had suffered fatal, severe and irreversible brain damage. Having heard and considered the expert evidence and in particular that of two distinguished specialists, Professor Marks for the claimant and Professor Hull for the defendant, the judge held that he was not satisfied on the balance of probabilities that, had the defendant administered intravenous glucose at the time of his arrival as he should have done, it would have saved Wilfred's life in that, by that stage, Wilfred had already suffered irreversible brain damage. The claim thus failed on grounds of causation.
3. The judge went on to find that, had he been satisfied that causation was established, the claimant would have been entitled to damages in respect of his psychiatric injury on principles established in *Allcock v The Chief Constable of South Yorkshire* [1992] 1AC 310 at 399, as explained and applied in *North Glamorgan NHS Trust v Walters* CA, 6 December 2002 [2002] EWCA Civ 1792. He assessed the damages to which Mr Toth would have been entitled in the sum of £30,000 made up of £15,000 general damages and a further £15,000 for loss of earning capacity.
4. Mr Toth appeals from the judge's decision on the issue of causation and, in the event of success in that regard, he appeals also against the judge's assessment of damages.
5. The judge's finding on the question of causation was based principally on his preference for the evidence of Professor Hull. Mr Toth asserts that the judge was in error in that respect, and that he should have preferred the evidence of Professor Marks. He asserts that this is clearly demonstrable on close examination of their evidence in relation to which the judgment reveals a number of errors through misunderstanding or oversight on the part of the judge. However, he also relies strongly on a body of material not in evidence before the judge, and the subject of an application to adduce new evidence, as to the views and experience of Professor Hull and, in particular, as to his connections with the Medical Defence Union. He submits that, had that material been before the judge, it would have discredited or undermined the evidence of Professor Hull upon the central issue in the case to the extent that the judge would have preferred the evidence of Professor Marks and found for Mr Toth.

6. Mr George Pulman QC appeared on the appeal for Mr Toth and Miss O'Rourke appeared for the respondent instructed by the Medical Defence Union. Both counsel appeared below. We heard argument first upon the issue of causation, for which purpose we heard also the application of Mr Toth for admission of the new evidence which we considered in detail *de bene esse*, indicating that we would rule upon the application in the course of giving judgment. Having heard the arguments of counsel at length, each of us formed the clear conclusion that no material error was demonstrable in the judge's findings of fact or his application of the law upon the evidence before him. Nor was there reason to conclude that he would have come to any different conclusion had the additional material been adduced in evidence before him.
7. Nor was it necessary to hear argument upon the respondent's notice which supports the judgment on all issues save one, namely whether or not the circumstances surrounding the death of Wilfred witnessed by Mr Toth qualified as a "shocking event" of the type necessary to support a claim by Mr Toth as a secondary victim on the basis set out in *Allcock v The Chief Constable of South Yorkshire*. There is a cross-appeal in that respect.
8. Having informed the parties of the conclusion we had reached, because of the detailed arguments raised upon the appeal which it is necessary to deal with at some length, we informed the parties that we would give our judgment at a later date. Delivery of the judgment has taken longer than anticipated for reasons which appear below.

The Facts:

9. Wilfred suffered from glycogen storage disease (GSD), a rare but well recognised condition in which the liver is unable to release glucose into the circulation. The brain requires glucose to function. In a normal body glucose is absorbed from food (starch) and during this absorption phase the liver produces and stores glycogen. In the ordinary way it is able to release the glycogen as glucose when needed during periods without food and in particular at night. By reason of his condition, Wilfred's liver was unable to do so.
10. Wilfred would eat hourly during the day and it was the practice of his parents who had received and understood medical advice in this respect, to feed him a starchy meal late in the evening and to ensure that he slept with a nasal-gastric tube connected to a reservoir of glucose via a peristaltic pump. By this means he was drip-fed glucose during the night to ensure that his brain would be supplied with glucose after that available from his food had been used up. Without it, Wilfred would become hypoglycaemic. Hypoglycaemia literally means low blood glucose concentration. It is arbitrarily defined, and was treated by the experts in this case, as being an arterial blood glucose concentration of below 2.2 mmol/L. We shall turn to its features and consequences in more detail below. It is sufficient at this stage to note that, as hypoglycaemia advances, it can induce coma, fitting and brain damage which may be irreversible.
11. It was often the habit of Mr Toth and his partner Ms Gover (who was herself a qualified and experienced nurse) to put Wilfred to sleep between them in their double bed, with the pump and reservoir attached to a stand a couple of feet from the bed, the tube running behind and over the headboard to reach Wilfred as he slept.

12. That was what was done on the night of 8/9 October 1993. Wilfred was fed a large meal of nearly one pound of sago about 10:30pm and went to sleep in the double bed with Ms Gover. Although she stated in evidence that she had turned the pump on via its touch pad controls as usual, the judge held that unfortunately she had not. The pump did not work that night and there was no evidence that there was anything wrong with it. Ms Gover, who was seven months' pregnant at the time, woke to go to the lavatory about 2:30am when Mr Toth was coming to bed about the same time. Neither checked or noticed whether the pump was working. At 5:30am Ms Gover woke again and remained awake thereafter, noticing nothing wrong with Wilfred, who was apparently asleep beside her, until about 7:15am when he began to have a fit.
13. Ms Gover at once recognised that the pump was not working. She woke Mr Toth who went downstairs and made up a glucose and water solution which he administered via the nasal-gastric tube. It appeared to have no effect and at about 7:30am (according to Ms Gover) and 8:00am (according to the defendant) she telephoned the defendant, who lived about seven miles away and got to the house around 8:25am.
14. Both parents made clear to the defendant that Wilfred had suffered a hypoglycaemic fit and that he suffered from GSD. They explained that glucose had been given and asked the defendant to administer intravenous glucose by injection. Despite the fact that he carried this with him, the defendant did not inject glucose. Instead, he administered two suppositories which had the effect of somewhat, though not very substantially, reducing the fit. He stated that Wilfred should go to hospital and wrote a referral letter in which he stated that Wilfred had been fitting since 7:15am.
15. The defendant left at 8:50am. The claimant did not use the ambulance service but set off for the hospital some 45 minutes later at about 9:35am. He arrived with the child at around 10:00am, at which stage Wilfred's blood sugar level was 0.5mm.
16. Wilfred was immediately taken into the care of the Paediatric Metabolic Team and given glucose intravenously. However, he had further seizures and by 3:30pm it was concluded that he had probably suffered severe and irreversible brain damage. In the event, he never recovered consciousness and died the following week.

Causation:

17. It was not the claimant's case that he would have suffered psychiatric injury had his son not died. It was thus necessary for him to demonstrate that, by the time of the defendant's arrival between 8:00am and 8:30am, had he administered intravenous glucose, it would probably have saved Wilfred's life.
18. At this point it is necessary to elaborate on the nature and consequences of the state of hypoglycaemia. It is to be distinguished from signs and symptoms of damage which it produces in the brain (neuroglycopenia), which are usually temporary but may, if hypoglycaemia persists long enough, become permanent. Hypoglycaemia comes about when glucose leaves the blood circulation faster than it enters it either from the intestine, so long as food remains there, or from the liver when absorption from the intestine has ceased. The rate at which glucose leaves the circulation depends upon a large number of factors, the most important being the concentration of glucose and of

insulin in the blood and the sensitivity of the bodies' tissues to the action of insulin. The concentration of insulin in the blood is itself largely determined by the blood glucose level. However the brain can extract glucose from the blood even in the absence of insulin. Ordinarily, therefore, once all the glucose derived from the starch in a meal has been absorbed and disposed of into the tissues, the only organ removing glucose from the circulation is the brain. This absorption is met by an equal and opposite release of glucose from glycogen produced and stored in the liver during the absorptive phase so that the blood glucose concentration remains at what is referred to as the "overnight fasting level". This ability to maintain the blood glucose level is less well developed in infants, but improves with age.

19. In the case of a child suffering from Wilfred's condition, namely glycogen storage disease type 1, the liver is unable to break down glycogen properly and release glucose into the circulation. As a result, during fasting, once all the glucose and the food have been disposed of, the blood glucose concentration falls gradually as the brain continues to extract glucose without its being replenished by the liver. As the blood glucose concentration falls below 2.2mmol/L, brain function begins to deteriorate. The more severe consequences of hypoglycaemia produce objective signs of brain malfunction of which the most overt are convulsions, especially in children, and impairment or loss of consciousness.
20. There is a further uncertainty in the case of children such as Wilfred who had for some years had continuous nocturnal naso-gastric treatment of his GSD, in that such a process carries an increased risk of death from hypoglycaemia. It leads to increased dependence on such infusion ("habituation") so that interruption of the infusion may lead to more rapid (and therefore more severe) brain damage than in untreated children who have adapted to hypoglycaemia but suffer physically and mentally in other ways.
21. On the present state of medical knowledge and research, it is not known with any degree of certainty how long the human brain can tolerate hypoglycaemia of sufficient severity to cause coma without becoming permanently damaged. The evidence available is unsatisfactory since it would be quite unethical to observe a person known to be in a hypoglycaemic coma without immediately attempting to restore their blood glucose level to normal with intravenous glucose.
22. In a report to the court dated 6th November 2000, Professor Marks the expert appearing for Mr Toth said this:
 - “29. I have been asked to attempt timing onset of severe hypoglycaemia potentially capable of producing irreversible brain damage. I believe that this is an impossible task but one that can be approached from either of two directions. The first is to try and assess how long it would have taken Wilfred's blood glucose levels to fall to below 1.1mmol/L-the level experimental evidence suggests it is necessary to produce potentially permanent brain damage.

30. I understand that Wilfred had a meal containing 55gms of carbohydrates (mainly in the form of cooked starch) and 7.7gms of fat at about 10.30-11pm. It is impossible to say, with any certainty, how long this would have taken to be absorbed. Work from my own laboratory (ARENDRT et al 1982) suggests that absorption, by young adults of a mixed meal of a similar composition to that taken by Wilfred is not complete until about 5-6 hours have elapsed although the blood glucose level returns to fasting levels much sooner than this.
31. Assuming that this applies also to children of five years of age, it would suggest that absorption of the meal Wilfred took at 10.30-11pm was continuing up to about 4.30-5.30am, or possibly later. From then on as the blood glucose level fell and insulin secretion stopped, glucose would leave the blood only by entering the brain. It would not, however, be replenished, in Wilfred's case, at the same rate by glucose released from the liver as this does not occur in glycogen storage disease type 1. Nevertheless it is probable that his liver would have liberated some glucose-albeit at a rate insufficient to meet the brain's needs through the action of the de-brancher enzyme system which remains intact. It is possible, therefore, that Wilfred did not become hypoglycaemic (i.e. blood glucose \leftarrow 2.2mmol/L) much before about 5am on the morning of the 9th and not seriously so (i.e. blood glucose less than 1.1mmol/L) much before 6-7am. This is less than three hours prior to the doctor arriving and before irreversible brain damage would have occurred.
32. The other-and I believe more likely-scenario is that Wilfred became seriously hypoglycaemic at or shortly before he began convulsing at 7.15am...Such a scenario would imply that by 8.30am, when the doctor arrived, Wilfred had been seriously hypoglycaemic for 80 minutes or so. He would, therefore, on the evidence available, be unlikely to have already suffered irrecoverable brain damage."
23. In his initial report Professor Hull, the defendant's expert, gave his opinion in response to a series of questions as follows:
- "9.2 *When did the hypoglycaemia start?* In my view it probably began in the early hours of the morning and certainly before 7.15am.

9.3 *When might it had been reversed without permanent brain damage?* If he only became profoundly hypoglycaemic for the first time at 7.15am then it is surprising that he did not respond to oral glucose within 30 minutes. The failure to respond to the oral feed (which appeared to have been effective on a previous occasion) suggests that hypoglycaemia had been present for some hours before the fitting started when his brain was irreversibly damaged.

9.4 The claimant's position is that whilst the hypoglycaemia could not be reversed by oral feeds it would have been reversed by intravenous glucose up until the time he would have been seen by Dr Jarman. That is a possibility. If that were so it might also have been reversed at the hospital 30 minutes later. I do not know how it can be established that intravenous glucose would have stopped the fits either when Dr Jarman saw him or in the hospital 30 minutes later. We do know that IV glucose did not stop the fits when he did arrive in hospital just after 10am, and it did not save his life indicating that the damage to the brain cells were severe.

9.6 *Why did the oral glucose not work? ...*

... When Wilfred arrived in hospital he was hypoglycaemic (glucose level 0.5mmol/L). If his father had given Wilfred a glucose solution before 7.30am and Wilfred was profoundly hypoglycaemic at the time, then it is likely that it would have been used up within 2 to 3 hours. When Wilfred was admitted, intravenous glucose did not stop the fitting. So the possibility remains that neither oral or intravenous glucose would have reversed his fitting at 7.15am. It would be helpful to know how much glucose his father gave Wilfred down the tube, and whether he also gave the polycal, which Wilfred should have received overnight. If generous amounts of both had been given, I would conclude on the balance that Wilfred would not have responded to intravenous glucose at 8.30am.

9.10 *If Dr Jarman had given intravenous glucose would it have made any difference?* Oral glucose given at

7.25am did not stop the fits over the following hour. By 8.45am Wilfred had been fitting for 90 minutes. It is possible that a large bolus of intravenous glucose might have worked where a smaller amount orally did not. On balance I think it unlikely. The same might have been the position if he had been taken to hospital by 9.15am. We do know that intravenous glucose given at 10.15am did not stop the fits.”

24. Thus, in respect of Wilfred, it was the defendant’s case advanced by Professor Hull that, on the probabilities, Wilfred had by the time of the defendant’s arrival already suffered gross deficiency in his blood sugar level and consequently irreversible and potentially fatal brain damage, too serious to be revived by an IV dose, based principally on the fact that Wilfred’s fitting had failed in any way to respond to the parents’ administration of glucose by nasal-gastric drip at about 7:15am. Professor Marks, for Mr Toth, disagreed. A summary expression of the experts’ rival views was set out in their written response to questions posed by the lawyers acting for the parties prior to the hearing.

25. In response to the question: “**Is it likely that permanent brain damage had occurred before the onset of fitting at 7:15 h?**” The experts replies were as follows

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Professor Hull: If the Sago was [Wilfred’s] last source of oral glucose then in my view, by 7:15h profound hypoglycaemia is likely to have been present for some hours. If oral administration of glucose in adequate amounts at that time did not reverse the effects of the hypoglycaemia then it is likely in this instance that permanent brain damage had already occurred.

Professor Marks: By 7:15h the hypoglycaemia was unlikely to have been severe enough or have lasted long enough to cause irreversible brain damage. I cannot accept David Hull’s statement which is inconsistent with most of the experimental and clinical evidence on the effects of hypoglycaemia in the brain.

26. In response to the questions: “**Would the administration of intravenous glucose during the period following the arrival of Dr Jarman about 8:30h and his departure at about 8:50h probably [have] avoided (a) death and (b) permanent brain damage? Would the administration of i.v glucose between 9:15h and 10:15h have avoided (a) death and (b) permanent brain damage?**” the replies were:

Professor Hull: If adequate amounts of glucose had been given orally at 07:15 hours and did not lead to recovery – as they had done so in the past – then intravenous glucose was unlikely to be effective. I am not aware of any disorder that results in a child becoming unable to absorb glucose delivered down a nasal-gastric tube which is rapidly absorbed.

Professor Marks: “If” and “adequate” are big words here in David Hull’s reply. Glucose delivered down a nasal-gastric tube is ordinarily rapidly absorbed – indeed

hypoglycaemia accelerated gastric emptying under experimental conditions. Nevertheless William Whittlek, with unrivalled experience of treating patients with what, up to that time, have been described as “irreversible hypo-glycaemia coma” during Sakel treatment of schizophrenia, treated many patients that had failed to respond to glucose given by gastric intubation by giving them glucose intravenously (treatment of prolonged insulin coma: J Ment Sei 1961: 107, 194-238). They responded in many cases, by returning to consciousness immediately or soon after administration of glucose intravenously. A minority took up to a few days. Kay attributed the failure of oral glucose and effectiveness of intravenous glucose to gastric paresis induced by prolonged and serious hypoglycaemia. In a few cases permanent brain damage did occur but was far from inevitable. Indeed only four of sixty-two patients died and this was before the days of modern therapy for cerebral oedema though he did have the benefit of ACTH.

27. In response to the question: “**What is the latest time at which the administration of i.v glucose would probably [have] avoided the fatal outcome?**” the replies were:

Professor Hull: My comments above relate to permanent brain damage. The information available is insufficient for me to give an opinion on when IV glucose might have avoided death. Wilfred failed to respond to IV glucose when it was given once he was admitted to hospital.

Professor Marks: There is no time before death when the process of hypoglycaemic brain damage becomes predictably irreversible. Patients certified by “conventional” methods as dead and taken to the morgue have been revived on being given appropriate treatment. Others known to have been comatose for up to twelve hours have recovered. Partial recovery of cognitive brain function of up to twenty days of hypoglycaemic induced coma is well recorded and survival for over twenty years in a vegetative state (but not requiring artificial ventilation) is not unknown.

28. In response to the question: “**What is the probable reason for Wilfred’s failure to respond to an oral feed of glucose given by his parents soon after the commencement of fitting? Is the history of the incident on 6.9.90 relevant to your answer?**” the replies were:

Professor Marks: The probably (sic) reason for Wilfred’s failure to respond was that his blood glucose – for whatever reason – was not in the normal range when the doctor arrived. It is impossible to be certain since the simple precaution for measuring it was not taken. He would in any event not have come to any harm by given intravenous glucose and it might have prevented a fatal outcome. The previous fitting incident on 06.09.90 is probably not relevant except in so far as it shows that Wilfred was not damaged mentally by 45 minutes of convulsions (status epilepticus) and that, once started, restoration to normoglycaemia did not bring immediate cessation to them. This is not unusual.

Professor Hull: If he was given an adequate amount of glucose by nasal-gastric tube, then his failure to respond was because his fits at that stage were due to another cause of fitting or to brain damage caused during the current episode of hypoglycaemia.

[In the event the case proceeded on the basis that there was no evidence of some other cause of fitting than brain damage.]

29. The oral expert evidence of each expert developed and elaborated upon the views expressed in his reports and the questions and answers I have quoted. Certain concessions were made by Professor Marks, but neither shifted his view in any significant respect. The evidence was concerned with a difficult and speculative area in which published research results (which largely related to animals and adults) were of very limited assistance. The area of specialism of each expert was thus highly relevant to the level of his expertise.
30. Having set out the issues on causation at para 19 of his judgment, the judge said this:

“20. I heard evidence on this topic from two distinguished specialists: Professor Marks, for the claimant, and Professor Hull for the defendant. Counsel on both sides sought to detract somewhat (not unnaturally) from the eminence or suitability of their rival experts. Professor Marks (now largely retired) was a former consultant and lecturer in clinical pathology and Professor of Clinical Biochemistry at Surrey University. He long had an interest in hypoglycaemia and has published on it. He has had no recent experience in treating children with the condition. Indeed, he has not treated children at all for over thirty years. Based on his experience with adults, he felt that the glucose from the 10:30 feed would have been used up in five or six hours, that is to say, by 3:30 to 4:30, and he feels that it is unlikely that there would have been irreversible (fatal) brain damage before the fits began at 7:15. He felt that, had Wilfred been given intravenous glucose within an hour of the onset of the convulsions, he would have survived with minimal brain damage. Professor Hull is a retired paediatric specialist. He has had experience of children with GSD and has researched and extensively published on energy metabolism. For twenty-five years he was academic Head of Child Health at University of Nottingham and a practising paediatrician. Professor Hull felt that the sago would have maintained the blood glucose for at least three to four hours. This would have taken Wilfred to 1:30 to 2:30, say some five hours before the fit was noticed. He thought that he was likely to have been “in trouble” with hypoglycaemia by about 3.am.

21. Professor Hull wrote:

“If he only became hypoglycaemic for the first time at 7:15, then it is surprising that he did not respond to oral glucose. The failure to respond to the oral feed ... suggest that hypoglycaemia had been present for some hours, and that fitting started when his brain was irreversibly damaged.”

31. It has not been suggested that there is any error in that summary. It is important to note in parenthesis that the references to “oral” glucose were a loose shorthand for the administration by nasal-gastric drip by the parents at about 7.15am.
32. Having referred to the medical evidence and considered the various arguments advanced, the judge concluded at para 32 of his judgment:

“32. The conclusion that I reach is that, on the balance of probabilities, enough oral glucose would have been administered by Mr Toth, and / or perhaps by his wife, to have been expected to produce relief, at least transiently, from Wilfred’s fit if his brain was not already past redemption. Or, to put the matter another way, I am not satisfied, on the balance of probabilities, that the administration of intravenous glucose at 8 or 8:30 would have saved Wilfred. I prefer Professor Hull’s evidence about the nature of hypoglycaemic children’s food absorption to that of Professor Marks, *inter alia*, since he has much more recent experience of children, and the focuses of his career have been much upon children. I find his evidence the more logical. I think it probable that, as Professor Hull says, the effect of the last meal had worn off perhaps four hours or so before the fit, that Wilfred was especially vulnerable to a shortage of glucose since he was not habituated to it, that he had suffered irreversible brain damage before the parental administration of oral glucose and, thus, the glucose which the father and mother administered failed to have the desired effect.

33. So, had Dr Jarman administered intravenous glucose, as would have been prudent, soon after his arrival, either at 8 or 8:30, it cannot be concluded, on the balance of probabilities, that it would have saved Wilfred’s life.

34. It is perhaps to be noted that Dr McShane, the treating Consultant in Paediatric Neurology, told Mr Toth on 22 November 1995 that he doubted that intravenous glucose would have changed things, had it been given on arrival by the GP. He accepted that it might have, and it may of course be that what he said was merely intended as a sedative remark to the claimant.

35. It follows that the claimant’s claim will fail...”

33. In finding as he did in relation to causation, namely that it had not been shown that administration of intravenous glucose on the defendant’s arrival would have saved Wilfred’s life, the stages of the judge’s reasoning and his findings on the evidence may best be summarised as follows.
34. It was common ground that by 7.15am when Wilfred began fitting, he must have been profoundly hypoglycaemic, and that, after one hour of fitting, the damage to his brain would be potentially, but not necessarily, fatal. It was also common ground that intra-

gastric administration of glucose (“IG”) delivered by naso-gastric tube was ordinarily quickly absorbed but that, if available, the most effective remedial action, in the sense of that action which would have most immediate effect, was intravenous injection (“IV”). Given that the parents administered IG glucose to Wilfred by his tube shortly after 7.15am; and given that (as Miss Glover stated in evidence) it usually took effect in about 10 minutes but on this occasion failed to have any effect; and given that thereafter the defendant did not arrive until some time between 8:00am and 8:30am, the causative hurdle to be overcome by the claimant was whether, if IV had been administered on the defendant’s arrival, it would have been effective to revive Wilfred (as suggested by Professor Marks) or whether by then (as suggested by Professor Hull) Wilfred had been hypoglycaemic for so long, and brain damage had occurred to such extent, that he was already beyond revival. In this respect a number of subsidiary issues required consideration in order to establish on the probabilities for how long Wilfred had been in a state of “profound” or “severe” hypoglycaemia to the extent that his brain began to suffer damage.

35. First, was the issue how long it was before the effect of the glucose from the sago feed would have ceased. In this respect, the judge accepted the assessment of Professor Hull, based on his long experience of children rather than adults, that it would have been 3 to 4 hours, rather than Professor Marks’ estimate of 5 to 6 hours: see paras 20, 21 and 32 of the judgment.

36. Second, and related, was the issue whether, and for how long, although fitting did not start till 7.15am, Wilfred had in fact been in a state of coma, i.e. profoundly unconscious, rather than simply sleeping as the mother believed. In this respect, the judge found that, awake though the mother was as she lay in bed with Wilfred,

“since she was reading the paper and watching television, she might well not have noticed any sign of peculiarity such as profound unconsciousness” (paragraph 23 of judgment).

37. Third, and crucial, was the issue how long a brain, and in particular the brain of a child such as Wilfred, would be likely to tolerate profound hypoglycaemia before being irreversibly damaged. In this respect, again the judge preferred the view of Professor Hull, based on long clinical experience with children in the field of paediatrics, over that of Professor Marks, an expert in the chemical pathology of hypoglycaemia who sought to underpin his opinion with the findings contained in various research papers. The judge set out his reasons at paragraphs 24 and 25 of his judgment.

38. The judge went on to state:

“26. Professor Marks regarded it as “an impossible task to time the onset of severe hypoglycaemia capable of producing irreversible brain damage”, but then proceeded to do so, suggesting Wilfred might either have been seriously hypoglycaemic before 6:00 or 7:00 but more likely shortly before 7:15. In cross-examination he said that he could not say that there was no irreversible brain damage by 8:30 or, indeed, 7:15, though he thought it unlikely. He conceded that

it was possible that Wilfred already suffered irreversible brain damage by 7:15. He said to that “anything over an hour of fit is potentially fatal”, and so this would have been the case by 8:15. The child may have been profoundly unconscious before he began to have a fit.

27. Dr Hull disputes Professor Marks’ belief that children’s food absorption rates are to be regarded as the same as adults’. He believes that they can absorb quicker. He would have expected the oral administration of glucose by the claimant to have worked if there had not been grave brain damage by the time it was given. As he put it “I think Wilfred had irretrievable brain damage by 7:15, because the oral glucose did not reverse it.” He did not think that intravenous glucose would have worked if the oral had failed. He felt that Wilfred was “in trouble and ill” some hours before he was noted to be fitting. In his report of 27 September 2002 he reports upon the case of a child at 3½ with GSD, whose nocturnal pump became disconnected for a maximum of 2½ hours. That child died, despite oral and intravenous glucose.”

39. Fourth, in relation to the question whether or not the failure of Wilfred to respond to the naso-gastric administration of glucose by his parents at about 7:15am was indicative of a state of hypoglycaemia so profound that it would not have been relieved by an IV administration from the defendant, an issue was explored as to the adequacy of the amount of glucose administered or whether, as Mr Pulman put it for the claimant, it was “too little or too late”. In this respect, the evidence of Mr Toth and Ms Gover had been imprecise and that of Ms Gover appeared inconsistent with an earlier statement. Mr Toth had mixed and administered the main infusion and, when it was not effective, Ms Gover gave an amount shortly before the defendant’s arrival, the amount of which was in dispute (see further below)

40. So far as IG administration was concerned, Ms Gover gave evidence that Wilfred had “had lots and lots of fits”, most of them “minor” but a few “grand mal” like the one in question and that they kept a store of glucose to be given by IG for just this event. She said that in the past, when they had given oral glucose, it had taken effect in 5 or 10 minutes. As between the experts, there was no issue that, unless there was some obstruction to its absorption by the gut, IG was an effective form of administering glucose subject only to its delayed effect as against IV infusion. Professor Marks offered as an explanation for the possible ineffectiveness of the parents’ IG infusion that Wilfred might have been suffering from “gastric stasis” which would have obstructed absorption through the gut. Gastric stasis would have been circumvented by IV administration. However, that evidence was at best speculative and unsupported by any specific evidence.

41. Professor Marks relied on a paper published in 1961 by a Dr Kay relating to 62 cases of insulin-induced coma in which the usual treatments of IG and / or IV glucose had failed to bring the patient out of coma. Dr Kay considered gastric stasis as a possible reason why IV glucose might not have worked in some patients, but provided no evidence that it did in fact occur in any of them and came to no clear conclusion as to why it might have occurred in some patients rather than others. Although Professor Marks claimed that the majority of the patients in the Kay study had not revived on IG glucose but had revived when given IV glucose, the numbers did not appear in the article nor did the article give any figures for cases believed to have involved a patient suffering from gastric stasis. When asked “Is there any research or evidence that indicates that [gastric stasis] has ever happened as far as a child is concerned?”, Professor Marks simply answered “No”.
42. The judges findings on this issue were as follows:
 - “29. It was argued on the claimant’s behalf that the reason the parental glucose did not work must have been because there was not enough of it. Professor Hull thought that 25 grams might have been the right amount. It would be very surprising, in my judgment, if that particular combination of parents – anxious, intelligent, knowledgeable and with relevant past experience – failed to administer a respectably generous dose, one likely to be efficacious. I would certainly have expected Mr Toth, who had, as he himself put it, “read voraciously around the subject of Wilfred’s illness” and had a good deal of contact with the treating specialists, to have been keen to administer a suitable quantity, as presumably he had in the past. Furthermore, Mr Toth told an interviewing psychiatrist (his own psychiatric witness) that he administered “a substantial bolus” of solution. Ms Gover too, as an experienced nurse, concerned that her child was not getting what she felt he needed, or reacting properly to what he had had, would have been unlikely to have contented herself with “a few remaining drips”. Bearing in mind that this is the evidence of a nurse whose “few drips” are, in my judgment, hardly consistent with “I had given Wilfred more glucose via his tube” as she said in her 1994 statement.
 30. Furthermore, even if the quantity given orally had been small, the evidence suggests that some response would have been expected, albeit not a long-lasting one, but there was, on the evidence, no response at all.
 31. To deal with this difficulty, Professor Marks suggested that perhaps the fit had the effect of preventing the child’s digestive system from working properly. I did not find this possibility (gastric stasis, as it was called) very likely or convincing. When the brain is desperate

for a substance put into the body, it seems likely to obtain it – Professor Hull spoke of the surprisingly beneficial effect of merely putting glucose inside of a cheek. He, with his specific experience of children, had never come across a child unable to absorb glucose.”

The judge then went on to state his conclusions in paragraphs 32 and 33 of his judgment, already quoted at paragraph 31 above.

43. Having set out the issues and the judge’s findings at some length, we are bound to say that we consider that, on the face of it at least, the structure and content of the judgment on causation is unimpeachable. Certainly no inconsistencies or misunderstanding are apparent. Furthermore, having thoroughly examined the material before the judge, it is clear to us that (save in one small, and in our view immaterial, respect) there is no finding or observation of the judge in respect of which he lacked evidence which it was open to him to accept, albeit such evidence was subject to challenge. Indeed, the contrary has not been the principal thrust of Mr Pulman’s argument on this appeal.
44. Mr Pulman raises a number of points on the evidence which he submits should have led the judge to prefer the view of Professor Marks, which points are set out in the Amended Grounds of Appeal. However, the main emphasis of Mr Pulman’s argument has depended upon the new evidence sought to be adduced, by way of an attack on Professor Hull for material which he is alleged to have suppressed rather than advanced in court, and which, if brought to the judge’s attention, would have led him to reject rather than accept the views expressed by Professor Hull.
45. However, we shall first deal with the points raised by Mr Pulman which are based upon the evidence before the judge and are not dependent upon the new evidence sought to be adduced, taking them in the order, and under the headings, set out in the Amended Grounds of Appeal.
46. By way of preliminary, at paragraph 8 of the grounds it is stated that the judge failed to understand that, even if irreversible brain damage had occurred by the time Dr Jarman arrived, that damage would not necessarily have resulted in death, nor would it necessarily have precluded Wilfred from living a full, fruitful and independent life. Indeed (it is submitted) such damage, had it occurred, may have been reversible.
47. Examination of the evidence reveals a short answer to this assertion. The judge was plainly alive to the fact that the progress of brain damage, whether in the course of a coma or as a result of fitting, was progressive and he took up with the experts the meaning and significance of their references to irreversible brain damage at that time. This is manifest from a series of questions which he put to Professor Marks at the end of his evidence as follows:

“Judge Harris: I just want to clarify this irreversible brain damage business. We may have been guilty of some loose language. You said at one stage in your evidence that this child had suffered irreversible brain damage by

10:15. I think that you were really meaning that at that stage fatal brain damage, were you not?

A. Well, he failed to recover. I mean, I assume that he died from brain damage.

...

Q. When you said that, am I to understand the word “irreversible” was being used as a synonym for fatal?

A. It was genuinely irreversible and – yes –

Q. Yes, Mr Pulman’s ...very sensible question was that you could have all sorts of levels of irreversible brain damage?

A. Yes.

...

Q. Right. Then when you were asked whether you could say there was irreversible brain damage by 8:30 – we are to take that in the same way?

A. I think one has to say that I am not in any position to say that it could not have occurred. As I say, I did not –

Q. So you could not say that there has been no fatal brain damage by 8:30, but you think it unlikely?

A. Yes.

Q. That is what you were saying?

A. Yes.

Q. So irreversible in the sense that we have been using it in your evidence up until the time Mr Pulman asked his question, we were using -

A. As fatal.

Q. As a synonym for fatal brain damage, were we?

- A. Yes. Yes I had been...
- Q. You see, I asked you a number of questions about can you have irreversible brain damage without a fit and then you are saying things like: “mostly fits do not result in irreversible brain damage”. Is that use of the word “irreversible” – were you understanding that to mean fatal?
- A. I was understanding it to mean fatal and –I mean, you can be left with neuronal fallout and some people do often suffer from partial damage to their brain, but what I was trying to distinguish was between, complete irreversible brain damage, which is for example, being in deep coma which is –
- Q. Yes but we are not talking in this case about somebody left with some sort of mental deficit... [which] could be referred to accurately as irreversible brain damage, but would not affect their vitality or ability to live?
- A. Yes. One never knows whether serious brain damage is going to lead to death until, as it were, it has happened.”

48. So far as Professor Hull was concerned, when engaged upon this issue in cross-examination, he put it this way:

“... There clearly comes a point when in fact it [hypoglycaemia] becomes irremediable by giving any sort of treatment. So if you are talking about when is the point reached when the brain damage is irreversible and lethal, that is the brain is damaged beyond recovery, then that is bound to be a very narrow window of time. But the period before that, it is likely that the brain has been damaged and it is unlikely to make a full recovery. But the debate about what sort of timing happens in all sorts of insults to the brain from asphyxia through to hypoglycaemia, is a big debate, and sometimes it is to do with the magnitude of the initial insult and sometimes it is to do with the increasing insult as the brain gets into increasing trouble. The length and the time it has to deal without glucose or whatever alternative it is using as its energy source.... There is obviously some time where it is no longer possible for any treatment to have lasting benefit.

Judge Harris: So it is deteriorating all the time?

- A. Yes.
- Q. But at what point it becomes –
- A. Irreversible –
- Q. At the stage that it is irreversible or fatal –
- A. Depends on the circumstances.”

49. It is thus apparent that when the judge stated his conclusion on causation at paragraph 32 of the judgment and found in terms that Wilfred “had suffered irreversible brain damage before the parental administration of oral glucose and, thus, the glucose which his father and mother administered failed to have the desired effect” he was using the word irreversible as a synonym for fatal, just as the experts had done. Further, in his questioning of Professor Hull, it is apparent that Mr Pulman was proceeding on the basis of the same convention and that the question he posed whether the IG administration of glucose by the parents was “too little or too late” was posed on a similar basis.
50. The first ground of appeal (ground (a)) asserts that the judge made a major error in determining that the die was already cast by 7:15am. It states that such a conclusion was not open to him on the basis that (i) the evidence of both experts amounted to an agreement that fitting was an early warning sign of hypoglycaemia; and (ii) he had found that the fitting itself only began at 7:15am as described by Ms Gover.
51. The assertion in (i), is simply a misstatement of the position. In seeking to make it good, Mr Pulman isolates a single answer given by Professor Hull in the course of his oral evidence to the effect that “it would be exceptional for a child to go straight into a deep coma without fitting”. Relying on that answer, Mr Pulman argues that Professor Hull and Professor Marks were essentially at one in the view that the fitting was an early warning sign of hypoglycaemia and not a sign that irreversible brain damage might already have been occasioned. Examination of the transcript shows that the answer is a misquotation and that, in any event, the overall nature of Professor Hull’s answers was to the opposite effect. The relevant passage of transcript reads as follows:

- “Q. But is it possible for it to sustain serious brain damage, potentially fatal brain damage without having a fit?
- A. Yes.
- Q. It is and why would that be?
- A. Well, the damaged brain – if the child was deeply unconscious, the brain is being damaged by hypoglycaemia. It usually results in the child showing some fit expressions but it does not necessarily do so.

Q. It generally would but occasionally it does not you say?

A. Yes. It would be exceptional for the child to go straight into a deep coma and not recover.

...

Miss O'Rourke: Professor Marks said that he was surprised in this case that the child had died. Do you share that surprise?

A. Yes. I think that we would all have hoped that a child who first started to fit at 7:15 would have responded to treatment, and it was the expectation when he got to hospital that he might respond to treatment. So it is a surprise.

Q. And you have said in your report and in your answers in the joint statement that your view is that on the balance of probabilities he had irreversible brain damage by 7:15 or not long after that. Why do you say that?

A. My reason for saying that rests on the fact that I think that glucose given orally would have been absorbed. If the glucose had been given orally and had been absorbed, it would have reversed the fit. If it did not reverse the fit, then that fit was irreversible by oral glucose and it would have been irreversible an hour later by intravenous glucose."

52. As to (ii), so far as the evidence of Ms Gover was concerned, whilst the judge accepted her evidence that the fitting only began at 7:15am, he did not accept, as Mr Pulman suggests, that the fact that she had noted no problems with Wilfred prior to that time meant that Wilfred was not in a coma, rather than simply asleep: see paragraph 23 of the judgment quoted at paragraph 35 above.

53. It is also argued that, in relation to this issue, the judge wrongly failed to pay attention to the evidence given in the written statement of the defendant, who did not appear as a witness, that, at 8:30am, he had not noticed any evidence of coma such as sweatiness or pallor, that Wilfred appeared to be sleeping soundly and in a stable condition and "not profoundly hypoglycaemic". That point is also without merit. As Miss O'Rourke has made clear to us, it was Mr Toth's case at trial that the defendant's statement should be discounted in its entirety as self-serving and unreliable. The case as then advanced was that the defendant was *wrong* when he

said he did not think that Wilfred was profoundly hypoglycaemic. The whole thrust of the case was that, by that time, Wilfred *was* profoundly hypoglycaemic, having become so at or shortly before the time he began convulsing at 7:15am. Neither counsel asked either expert to consider what the defendant had said on that topic; nor was any reference to it made in the final submissions of Mr Pulman below.

54. The second ground of appeal (ground (b)) complains that there was no finding on the question whether the defendant was late in arriving at Mr Toth's house. It was the evidence of Mr Toth and Ms Gover that they made a call by telephone to the defendant at 7:30 or 7:35am and that he unduly and negligently delayed in that he did not arrive until about 8:30am from his home about seven miles away. It was the defendant's case that he was only called at about 8:00am and that he came with all due dispatch. The judge declined to make a finding as to the time of the defendant's arrival and / or whether the time which he took involved any negligence on his behalf because, as the judge stated at paragraph 12 of his judgment:

“It does not seem to me that the allegation of late arrival takes matters very far, since, even if the doctor had been there half an hour earlier, presumably he would still not have administered intravenous glucose, and there is no evidence that, had the child arrived in hospital half an hour earlier than it did, it would have made any difference”

55. Given the nature of the dispute on causation, the inevitable approximation in timings, and the view taken by the judge on Professor Hull's evidence that by 8:00am it was likely that the die was cast in any event, we do not consider that there is any substance in this ground of appeal.
56. In the third ground of appeal (ground (c)), criticism is made of the judge's finding as to the sufficiency of the glucose administered by Mr Toth as an indicator of whether or not an IV administration of glucose by the defendant would have been likely to succeed. It was Professor Hull's evidence for the defence that an adequate amount of glucose would have been about 25 grams, although a smaller amount might have been expected to work. The grounds of appeal also state that it was Mr Toth's evidence that he mixed up two teaspoons-full of glucose, injecting it into the nasal-gastric tube as a large bolus, Ms Gover stating that whilst waiting for Dr Jarman she administered a residue of the solution still left.
57. Mr Toth's evidence was in fact that, in mixing the glucose solution, he had used “about two heaped tablespoons – not tablespoons, teaspoons which, as you know, glucose is very, very sticky so you can have a very, very tall heap when you spoon glucose... it might be, what, an inch and a half tall.” There was debate in court about the effect of this evidence. It was put by Mr Pulman to Professor Hull, (who did not know, but accepted for the purposes of argument) that the ordinary measure of a teaspoon is 5 grams. On that basis, the description of Mr Toth (of 2 teaspoons piled as much as an inch and a half high) could well have amounted to 25 grams (the dose which Professor Hull regarded as the recommended amount, a view which was not challenged). However, Professor Hull also suggested that a smaller amount would have been effective, though he did not know how small. There was also an issue whether the further dose which Ms Gover stated she gave to Wilfred shortly before the arrival of the defendant was merely the remnants of the mixture earlier

administered by Mr. Toth or the product of a larger dose further mixed by her. It was plainly open to the judge to doubt the accuracy of her evidence without casting any aspersions on her honesty and, in our view, upon the evidence before him, he was entitled to come to the conclusion that, on the balance of probabilities, enough IG glucose would have been administered by Mr. Toth and/or Ms Gover to have been expected to produce relief: see paragraphs. 29 and 32 of the judgment, as quoted above at paragraphs 40 and 31 respectively.

58. The fourth ground of appeal (ground (d)) is that the judge without good or proper reason, rejected Professor Marks' 'gastric stasis' theory advanced by him as a possible explanation for the failure of the oral glucose to take effect. It is asserted that the judge failed to understand the scientific evidence concerning the difficulties in absorption of glucose administered orally. Mr. Pulman has wholly failed to demonstrate that the judge misunderstood the evidence. The judge was fully entitled to reject Professor Marks' 'gastric stasis' theory in the light of the state of the evidence before him. While Professor Marks asserted that it was possible that the reason for Wilfred's non-absorption of his IG administration of glucose was that he experienced 'gastric stasis', there was no evidence to this effect. We have already referred to the nature of the paper published in 1961 by Dr. Kay upon which Professor Marks founded this possibility (see paragraph 41 above). That study apparently related to adults, who had all been given insulin as part of psychotherapy. When Professor Marks was asked by Miss O'Rourke in cross-examination whether there was any research or evidence to indicate that 'gastric stasis' had ever happened so far as a child was concerned, he answered simply "No".
59. The fifth ground of appeal (ground (e)) is that the judge unreasonably relied too heavily on Professor Hull's opinion as against that of Professor Marks because:
- (i) Professor Hull provided no research literature in support of his opinion, relying on his experience (characterised as "anecdote" in the grounds of appeal) whereas Professor Marks provided research material and references. It is said that Professor Marks' opinion was supported by a number of other eminent clinicians whereas Professor Hull had no such support; (ii) Professor Hull, though a retired paediatrician, specialised in thoracic medicine, whereas Professor Marks specialised in metabolic medicine which was the subject matter of the causation issue. (iii) Professor Hull stated that parents are the experts on their child's condition, therefore any unusual behaviour or signs (if present) would have been recognised by them or by the defendant, who was a GP with declared substantial paediatric experience. (iv) The judge unreasonably marginalised Professor Marks in favour of Professor Hull, particularly in relation to his comments on the time Professor Marks had been in retirement when compared with Professor Hull. (v) Professor Hull, during his time as a clinician, relied upon, inter alia Professor Marks' work on hypoglycaemia; therefore the judge had no good reason to discount such research in favour of "anecdotal" evidence. In any event (it is stated), Professor Hull's anecdotal evidence was contradicted by medical and scientific evidence in Wilfred's medical records.
60. In our view the judge was plainly entitled to prefer the evidence of Professor Hull over that of Professor Marks, based on his experience and the substance of his evidence, as well as the manner in which he gave it. Despite his eminence as a clinician and an expert on hypoglycaemia, Professor Marks had limited experience and, as he accepted, little expertise in treating children and in particular any with

glycogen storage disease, in contrast with the considerable experience of Professor Hull in treating children generally and some experience in caring for children with glycogen storage disease. Professor Marks conceded that he was rarely concerned with day to day management of patients but, when he was, he had treated adults rather than children and had in any event retired from clinical practice in 1995. His involvement in treating children had ceased 35 years previously and he had never had any day to day responsibility for the management of children such as Wilfred with GSD. He had only ever seen 3 or 4 cases of GSD (and then not as the treating doctor) and had no personal experience of the death of a child with GSD from hypoglycaemia. He conceded that he would not be competent to address the question of irreversible brain damage occurring in a 5 year old child in the absence of fitting. Professor Hull on the other hand, was an experienced paediatrician who had had consultants' responsibility for children with hypoglycaemia and with GSD. He did not accept that Professor Marks was an expert on treating children with hypoglycaemia, describing him as a distinguished chemical pathologist. Given that the experts were not of the same discipline, and given their differences in experiences and expertise, the judge was entitled to reach the conclusions he did in assessing their evidence.

61. In relation to the points enumerated in paragraph 59 above, the position is as follows.
62. Also to point (i), it is correct that Professor Hull did not rely upon any research literature in support of his opinion which he stated to be based upon his own experience, whereas Professor Marks relied upon material to which we will shortly turn. Such material was found by the judge to be of little assistance in relation to the assertions and opinions of the experts for reasons which he expressed.
63. The scientific and medical literature produced by Professor Marks in his opinion was as follows. There were 3 papers which it is clear the judge considered specifically and referred to in his judgment. First, the 1961 paper by Kay "The treatment of long insulin coma"; second, a paper by Khan and Myers "Insulin induced hypoglycaemia in the Non-human Primate"; and third, extracts from a work by Sakel "The pharmacological shock treatment of schizophrenia". All were the subject of oral evidence and found to be of little assistance in Wilfred's case for the reasons expressed by the judge at paragraphs 24-25 and 27 of his judgment, in which he indicated that he accepted and preferred the comments and evidence of Professor Hull as to the weight to be attached to such papers.
64. The remainder of the literature supplied by Professor Marks was as follows. A 1966 paper by Marks "The treatment of hypoglycaemia with diazoxide"; a 1982 paper by Marks and others "24 hour profiles on melatonin, cortisol, insulin, c-peptide and gip following a meal and subsequent fasting"; a 1967 paper by Anthony and others "Studies in hypoglycaemia of infancy and childhood" concerning 13 cases of children with "idiopathic hypoglycaemia" (i.e. *excluding* those with a known aetiology such as GSD); a 1960 paper by Boley and others "Functioning pancreatic adenomas in infants and children"; a 1999 paper by Marks and others "Hypoglycaemia: factitious and felonious", dealing with cases of hypoglycaemia which was self-induced or wrongfully induced by insulin for sulfonylurea; a 1989 paper by Maytel and others "Low morbidity and mortality of status epilepticus in children", concerning children suffering seizures from a wide range of causes and concluding that death from seizure per se is rare; finally, a paper by Nuoffer and others "Treatment with low-dose

diazoxide in two growth retarded prepubertal girls with glycogen storage disease type 1a resulted in catch up growth”.

65. It is not apparent that the judge was referred at trial to any specific passages in this additional literature. Nor were we. Our own study leaves us unpersuaded that there is information to be derived from them which assists Mr Toth’s case.
66. The reference to supporting opinion from other eminent clinicians is to evidence which was specifically excluded by the judge, namely letters or reports from several physicians to whom Mr. Toth had written concerning his case. He had not sought or obtained any permission for such documents to be admitted in evidence at any pre-trial application. There was an attempt to introduce them in evidence at trial. However, the reports did not meet any of the requirements of CPR Part 35 or the accompanying Practice Direction and were the subject of objection upon that ground by Miss O’Rourke when Mr. Pulman sought to introduce them in cross examination of Professor Hull on the ground that they were documents referred to by Professor Marks in his report. In the face of that objection, they were withdrawn and there is no appeal against that ruling by the judge.
67. As to point (ii), The assertion that Professor Hull was a paediatrician who specialised in thoracic medicine is based upon the statement of Professor Hull in his original report that between 1965 and 1972 he was a paediatrician at Great Ormond Street with a general service and “an interest in infectious and respiratory diseases”. However, Professor Hull went on to state that from 1972 to 1997 he was head of the academic Department of Child Health at the University of Nottingham and a paediatrician to the hospitals in Nottingham.
68. He had also been President of the British Paediatric Association. It is a matter of which complaint is made (see further below) that Professor Hull did not go into more detail. Mr Pulman has told us that it was only in the course of examination in chief by Miss O’Rourke that Mr. Pulman learned from Professor Hull that he had a good deal of relevant experience. In the light of what is said in respect of that experience we propose to set out *in extenso* the relevant passages in the transcript from the point when Miss O’Rourke asked Professor Hull about his experience with hypoglycaemia, to which he said he had experience with both new born and older children with various forms of hypoglycaemia.

Q. Right. And in what way have they had hypoglycaemia, or what have been [their].... underlying conditions, and what are they?

A. The area in which I have done quite a lot of research is hypoglycaemia in the new born period, to new born babies and in the first weeks of life. But when I was at the hospital for sick children, Great Ormond Street, I had patients referred to me with metabolic disorders, a number of which had got hypoglycaemia and glycogen storage disease, but many had other metabolic disturbances which led to hypoglycaemia.

- Q. Right. And what about first line management of children with hypoglycaemia or with glycogen storage disease? Do you have experience of that?
- A. Yes, because I was the Consultant responsible for their care, and when I see them, I see them for the rest of their life until they grow up.
- Judge Harris: What sort of number of children have you dealt with, with this glycogen storage disease?
- A. With glycogen storage disease, this particular form is relatively rare, and I can remember nearly the individual names of the children, but must be about 3 or 4 in Great Ormond Street and about 3 or 4 when I have been in Nottingham.
- Q. So 6 to 8?
- A. Yes.
- Miss O'Rourke: And have you been involved in their management over a number of years?
- A. Yes. Yes, I keep in contact with some of them.
- Q. Right. And in terms of your work on metabolic diseases on hypoglycaemia and glucose, have you published on it, have you performed research?
- A. My research has been on energy metabolism, which is glucose and fatty acids. I started that when I was in the Nuffield Institute for Medical Research here in Oxford. I continued then for many years doing that research and have published over 100 papers on all aspects of glucose and fatty acid metabolism.
- Q. Right. What about textbooks? Do you edit any textbooks in respect of the management of hypoglycaemia in children?
- A. Yes. I edit the Essential Paediatrics which is for undergraduate students, which is now in its fourth edition. It is widely used. I edit Hospital Paediatrics, which is for Registrars and Senior Registrars, which also identifies how they recognise and management of the various forms which cause hypoglycaemia. I was the initiator and Chairman of the Editorial Board of Current Paediatrics, which produces expert statements regardless of consultant paediatricians on the management on a range of diseases, including hypoglycaemia. And since I have finished my clinical appointment, I have been Chairman of the Royal

College of Paediatrics and Child Health Medicines Committee which produced a book for the Medicines for Children, which gives clear guidance on the management of hypoglycaemia in different shapes and forms in children”.

69. We will refer further to the question of Professor Hull’s qualifications and expertise below. However, suffice it to say at this stage that there is no substance in the point that Professor Hull’s speciality was simply in thoracic medicine.
70. As to point (iii) it is not in dispute that Professor Hull stated that parents in general, and these parents in particular, were experts on their own child’s condition and would be likely to notice unusual behaviour or signs, if present. There is no suggestion that the judge ignored this evidence. Indeed, it was part of the argument as to whether Ms Gover might or might not have assumed that Wilfred was merely asleep when, according to Professor Hull, it was likely that he was profoundly hypoglycaemic and effectively in a coma.
71. As to point (iv), we are not persuaded that the judge’s assessment of the reliability and relevant expertise of the experts amounted to “unreasonable marginalisation” of Professor Marks’ evidence in favour of Professor Hull.
72. As to point (v) neither Professor Hull nor the judge decried Professor Marks’ expertise upon the causes and aetiology of hypoglycaemia. However, it was the case that Professor Marks accepted that his relevant work was done with adults and that the issue for the judge related to the likely response of a child to therapeutic attempts at his revival following the onset of profound hypoglycaemia and prolonged fitting.
73. We pause at this point to return to the reservation which we expressed in parenthesis at paragraph 43 above in respect of the evidential basis for one of the judge’s observations. At paragraph 31 of his judgment, when dealing with Professor Marks’ suggestion that Wilfred might have suffered from gastric stasis, the judge said this:
- “I did not find the possibility (gastric stasis, as it was called) very likely or convincing. *When the brain is desperate for a substance put into the body, it seems likely to obtain it* – Professor Hull spoke of the surprising beneficial effect of merely putting glucose against the inside of a cheek. He with his specific experience of children, had never come across a child unable to absorb glucose”. (emphasis added)
74. Mr. Pulman has criticised the sentence which we have italicised, as an intuitive observation by the judge unfounded in the expert evidence, and which Mr Pulman asserts submits was a misconception going to the heart of the judge’s assessment of that evidence. We accept the first part of the submission, but not the second. In our view the judge’s use of words was essentially the metaphor of a layman which was not unreasonable given the evidence before the judge that even glucose gel rubbed inside the child’s mouth was likely to be rapidly absorbed and to stop hypoglycaemic symptoms. In any event it was not an observation on which the judge based his causation decision. The reasoning for that decision was logically developed, as we

have summarised it, between paragraphs 19 and 34 of the judgment with no evident misunderstanding of the medical or scientific facts.

75. Finally, we refer to an additional point which does not figure in the grounds of appeal but was nonetheless argued by Mr Pulman. He submitted that the judge failed to take into account the fact that the evidence showed that, prior to his last sago meal at 10:30pm, Wilfred had had two meals at 5:30pm and 8:30pm. That is indeed the case. However, since neither Professor Marks nor Professor Hull raised or alluded to that point in evidence, it seems plain that the experts regarded the last and largest meal as the relevant source of glucose to be considered.
76. We turn now to the application to adduce new evidence.

The application to adduce new evidence

77. The new evidence sought to be adduced by Mr Toth on this appeal falls into two distinct categories. (1) Evidence relating to the extent and nature of Professor Hull's experience and expertise, including his areas of research and authorship, which evidence is said to have been unavailable to Mr Toth and his advisers at trial. It is said that, if known to the judge, it would have adversely affected his view of Professor Hull's authority as an expert in relation to the issues in the action as compared with that of Professor Marks. (2) Evidence of Professor Hull's connections with, and involvement in, the affairs of the Medical Defence Union (MDU) who instructed Miss O'Rourke on behalf of the defendant. That evidence is said to raise an undisclosed conflict of interest between Professor Hull's duty of objectivity as an expert and his interest in assisting in the defence of a member of the MDU. It is said that this would at least have caused the judge to regard his evidence with circumspection. Mr Pulman submits that, taken separately or in combination, both categories of evidence would or might have caused the judge to have rejected Professor Hull's evidence in favour of that of Professor Marks.

Should the new evidence relating to Professor Hull's expertise be admitted?

78. In relation to category (1), we have already noted the position at trial whereby, as Mr Pulman has informed us, he did not become aware of any suggestion that Professor Hull had an expertise in the treatment of hypoglycaemia, or that he had been concerned with a child or children suffering from GSD, until the beginning of Professor Hull's evidence, when Miss O'Rourke directed certain questions to his experience. Mr Pulman at once objected, but that objection was not sustained by the judge.
79. Miss O'Rourke asked Professor Hull about his experience with hypoglycaemic children, followed by various questions and answers which we have already summarised at paragraph 66 above. Having laid that foundation, she proceeded to ask further questions directed to the particular issues which had crystallised by the close of Professor Marks evidence in which he had, (without objection from Miss O'Rourke), elaborated very considerably upon the relatively spare terms of his reports and the summary expression of the experts' rival views which we have set out at paragraphs 25 – 28 above. Despite intermittent objection from Mr Pulman, Miss O'Rourke was permitted to amplify Professor Hull's views on the basis that Mr Pulman had himself been afforded a good deal of latitude (albeit without objection

raised) and that a combination of the short adjournment and the continued presence of Professor Marks, would enable Mr Pulman to cope with what were not new points, but answers to matters already considered and raised by Professor Marks.

80. When cross-examining Professor Hull, as he did at length, Mr Pulman did not challenge him to any substantial extent upon the relevance or accuracy of the experience to which he spoke. However, as Mr Pulman has made clear to us on this appeal, he had no opportunity to go to the various papers and other publications to which Professor Hull made general (but un-particularised) reference: see paragraph 68 above. That opportunity has now been taken by Mr Toth. Various papers and materials are exhibited to his statement dated 7 November 2005 in support of his application to adduce new evidence. In response to a request for information, Professor Hull has also supplied his CV with a long list of published articles attached, and, by his witness statement in reply dated 5 January 2006, Mr Saffron, Mr Toth's solicitor, raises a number of argumentative points of comment and interpretation in relation to that CV. In Mr Toth's statement, he purports to explain the significance and effect of the evidence which he asserts establishes that Professor Hull:

- “(a) Does not have the experience of the condition which my son had which he, in his oral testimony, claimed to have;
- (b) Has not conducted any research relevant to my son's condition as he claimed to have done;
- (c) Has not published a number of research papers as he claimed;
- (d) Could not reasonably have believed that the evidence that he gave during the trial was correct.”

81. Although it does not form a separate ground of appeal, it is Mr Pulman's submission in support of his application to adduce the material produced by Mr Toth that the judge was in error in permitting Professor Hull to elaborate at trial upon his CV and experience, in order to fortify his own expressions of view and his criticisms of Professor Marks' opinion. In particular, because Mr Pulman had no real opportunity to investigate the position for the purposes of his cross-examination he submits that a situation of “ambush” was thereby created. Mr Pulman submits that, now there has been opportunity to investigate the extent of Professor Hull's experience, as well as the publications to which he referred, the result is erosive, rather than supportive, of Professor Hull's claims to expertise in relation to treatment of hypoglycaemia.

82. In the face of that submission, it is appropriate to approach the case first by asking whether the judge was in error to rule as he did, in the circumstances and upon the state of play which existed at the time his ruling was made; and second, by applying to the new evidence relied on, the well known principles in *Ladd v Marshall* [1954] 1 WLR 1489, as set out at paragraph 52.11.2 on page 1534 of *Civil Procedure (2006)*.

The judge's ruling as to the first question

83. In our view the judge acted well within his discretion in permitting Professor Hull to elaborate as he did upon his knowledge and experience.
84. Mr Pulman relies upon the “Practice Direction – Experts and Assessors” which supplements CPR 35 and is set out at p.964 et seq of *Civil Procedure* (2006) Vol 1. Para 2.2 (Form of content of Expert’s Reports) provides that an expert’s report must (1) give details of the experts qualifications; (2) give details of any literature or other material which the expert has relied on in making the report. Mr Pulman also cites the Code of Guidance on Expert Evidence which was current at the time of trial and which appears set out at p.938 et seq *Civil Procedure*(2005) Vol 1. Part 1 of that code provides that an expert witness is under an overriding duty to help the court to deal with the case “justly”, that being the overriding obligation of the court under Part 1.1(1) of the CPR as further defined in Part 1.1(2) which requires that the case should be dealt with justly and so as to ensure that the parties are on an equal footing. Paragraph (15) of the Code of Guidance requires that:
- “All experts’ reports should contain the following information:
- (a) The experts’ academic and professional qualifications;
-
- (e) Details of the documents or any other evidence upon which any aspects of the advice or report is based;
- (f) Relevant extracts of literature or any other material which might assist the court in deciding the case;”
85. Mr Pulman submits that the way in which the matter was conducted in relation to the evidence of Professor Hull placed the defendant in breach of both the letter and the spirit of those provisions. That question is by no means clear-cut. Professor Hull set out briefly his academic and professional qualifications and, so far as his reports and proposed evidence were concerned, at the start of the trial he appears to have had no intention of making detailed reference to research or the work of others, apart from various limited references identified in his reports and in respect of which no complaint is made. Nor were there relevant extracts of literature or other material upon which he relied for the purposes of his opinion. He simply relied on his general experience as a treating paediatrician. But, even if it be accepted that Professor Hull should more fully have set out his qualifications or professional publications in the reports which constituted his witness statements, the judge plainly had power to permit him to elaborate upon them in his evidence-in-chief if he considered that there was good reason not to confine that evidence to the contents of the reports: see CPR 32.5(2) – (4). This power was not fettered by reason of the fact (if it was the case) that the omissions amounted to a breach of some other rule or Practice Direction, although plainly, if they did, it went to the issue of overall fairness as between the parties in relation to the judge’s ruling on Mr Pulman’s objection.
86. Having studied the transcripts of the relevant exchanges, it is our view that the judges’ decision to permit in evidence the further questions and answers of Professor Hull in relation to his experience was both rational and fair for the reasons he gave (see

paragraph 79 above). It is a separate question whether the evidence now sought to be adduced is admissible on *Ladd v Marshall* principles, to which question we now turn.

Could the evidence have been obtained with reasonable diligence for use at the trial?

87. In our view there can only be one answer to the question whether the evidence could with reasonable diligence be obtained before trial. It is plainly yes. From the long history of the case, during which Mr Toth has generally left no stone unturned to pursue his claim, and given the nature of the differences between the experts so clearly set out in their reports and the summary expression of their rival views (see paragraphs 25 – 28 above), the need for and/ or likelihood of cross-examination on the experts rival areas of expertise must have been apparent well before trial, if not to Mr Toth, then at least to the solicitors who represented him and who were thoroughly experienced in medical negligence cases. It would of course have been open to Mr Toth's solicitors, upon receipt of Professor Hull's report to request a full CV, including a list of papers and other publications standing to his name. It has not been made clear to us why they did not do so. Nor is it clear from Mr Toth's affidavit whether, and if not why not, he or his solicitors instituted other enquiries by way of an internet or other search into the extent of Professor Hull's qualifications and expertise as illustrated by such papers or publications.
88. The only explanation advanced by Mr Pulman for his ignorance on these matters is that, taking the content of Professor Hull's original report at face value, he was simply a retired paediatrician who had specialised in thoracic medicine, whereas Professor Marks was a retired specialist in metabolic medicine, so that there was no need for further investigation, the case for Mr Toth being best enhanced by preserving and emphasising that difference.
89. In our view that was an approach based on frail and unverified foundations. We have already set out what Professor Hull said as to his experience and qualifications at paragraph 68 above. He referred to his special interest in thoracic medicine only up to 1972, whereafter he became the head of an academic department and a distinguished paediatrician, in relation to which he gave no details of his specialisms or publications, a feature which invited exploration. Thus, if it be the case that, not only Mr Pulman, but Mr Toth and his solicitor, arrived at trial in ignorance of those matters, it is a position which could have readily been avoided by inquiry.
90. That being so, the application to call new evidence on this aspect fails to satisfy the first of the *Ladd v Marshall* criteria. However, in the interests of exploring the overall justice of the case, to which strict application of the criteria in *Ladd v Marshall* does not invariably provide an answer, we proceed summarily to consider the second of those criteria.

If available to the judge would the additional material have had an important influence on the result of the case?

91. There are two levels at which Mr Pulman has argued that the additional evidence would have had an important influence on the views of the judge. First, he submits that it would have demonstrated that Professor Hull in fact lacked relevant experience which the judge believed him to have and, second, that the evidence shows that he

was less than frank in what he said. Without exploring every detail of the criticisms made, the principal points are as follows.

92. In the passage of the transcript quoted above at paragraph 68 Professor Hull dealt with four aspects of his experience. First, hypoglycaemia in new born babies, into which he stated he had carried out research, but did not claim to have published it; second, GSD, which he said was rare, but that he had nonetheless treated 3 or 4 cases at Great Ormond Street (1966-1972) and a similar number at Nottingham (1972-1976); third, his research publications in relation to energy metabolism i.e. aspects of glucose and fatty acid metabolism; fourth, his editing of textbooks on the identification and treatment on the range of diseases, including hypoglycaemia. None of the new evidence demonstrates any inaccuracy in those respects.
93. First, there is nothing to show that Professor Hull did not have extensive experience of children with hypoglycaemia, in the light of his long experience as a paediatrician and of his particular interest in hypoglycaemia in children in the first weeks of life of which he made mention. Nor does the fact that he did not publish it gainsay his assertion that he had carried out research in that respect.
94. Second, there is nothing to show that he was not truthful or accurate in referring to his contact in the course of his career with 6-8 children suffering from GSD. In his statement, Mr Toth attempts to use statistics from the Department of Health to prove that Professor Hull could not have been telling the truth when he said that, in thirty years as a general paediatrician from 1960 to 1996, he had seen that number of children with GSD. The statistical evidence relied upon by Mr Toth involves extrapolation and assumption in relation to the thirty years prior to 1996, based on statistics limited to the period 1996-2002. Further, in response to Mr Toth's attack, Professor Hull states that, on further reflection, he has no reason to change his answer as to the number of children with GSD he had seen during his career. In this respect, he explains that Great Ormond Street, where he was from 1966 to 1972, was a centre for referral of rare metabolic disorders; and that, when he moved to Nottingham, he was frequently asked to see such children because he had had experience of seeing them at Great Ormond Street.
95. Third, Mr Pulman submits that, later, in responding to the question whether he had published any research on hypoglycaemia, Professor Hull essentially side-stepped the question by saying that his research had been on energy metabolism, which was glucose and fatty acids, thereby giving the judge the impression that he had conducted a body of relevant research, which he had not. We have considered the submission carefully, together with the statement of Mr Toth, and the additional material now available. We can not find any justification for the submission made. As it seems to us, Professor Hull was making the point that his research was general research relating to the alternative energy sources of the body, namely glucose and fatty acids, as opposed to specific research into hypoglycaemia which he had been asked about earlier and had explained as being related to his work with new born babies at Great Ormond Street. On this basis, his evidence was not misleading and, had there been any ambiguity about it, it would have been open to Mr Pulman, with Professor Marks present and available to advise him, to have challenged Professor Hull on that point. There is no reason to suppose that the judge did not understand the distinction made, but, even if he did not, there is equally no reason to suppose that, having been apprised of it, he would have changed his view of the merits of the experts in relation

to the problem before him. As already made clear, the judge's preference for the expertise of Professor Hull was based less upon his research background than his practical experience in the treatment of hypoglycaemic children and their response to IV and /or IG administration of glucose.

96. Finally, so far as Professor Hull's editorship of textbooks is concerned, Mr Toth takes the point that Professor Hull's evidence conflicted with views expressed on those textbooks to the extent that, in relation to the management of hypoglycaemic attack, all recommend immediate treatment by IV injection. Mr Toth observes that this is contradictory of Professor Hull's evidence that he would have expected oral administration of glucose to be effective. In our view, that simply does not follow. The recommendations for immediate IV administration are all based on the need for urgency in treatment and are not related to the question whether, in the absence of IV administration, IG infusion could be expected to be effective. No doubt the reason for this is that, when a child becomes hypoglycaemic, not only should the quickest available remedy be applied, but he will rarely have a naso-gastric tube in place whereby IG administration is practicable; further if he is in coma, he will be unable to swallow and the best that could be achieved short of IV injection would be to rub gel into his mouth. It is therefore not surprising that none of the medical protocols put forward by the claimant mention naso-gastric administration and, when they refer to oral administration, they refer to it in the strict sense rather than as a shorthand for IG administration.
97. The distinctions above referred to readily appear from the "Emergency Regimens" protocol for hypoglycaemia in children produced by the London Centre for Paediatric Endocrinology dated January 2004, which is among the material produced by Mr Toth:
- “1. If the child shows clinical signs of hypoglycaemia, i.e. sweatiness and drowsiness, a glucose drink should be given.
 2. If the child does not tolerate oral glucose or remains persistently hypoglycaemic then the following procedure should be adopted:
 - (a) Give glucose intravenously...
 - (f) If intravenous access is lost ...glucose may be administered by naso-gastric tube or glucose gel...”

There is nothing to suggest that infusion by naso-gastric tube is not generally effective, as was common ground between Professor Marks and Professor Hull, and that it would be expected to produce results, within 10 minutes or so. In those circumstances therefore, we do not consider that the literature disclosed carries the matter beyond the position considered by the judge on the evidence of the experts.

98. We therefore conclude that the second of the criteria in *Ladd v Marshall* is not made out in relation to category (i) of the new evidence relied on.

Should the judgment be set aside on the ground that Professor Hull failed to disclose a conflict of interest?

99. As to category (ii) we now move to a different subject, namely the appellant's attack on the judge's reliance on the evidence of Professor Hull having regard to the non-disclosure by him of a conflict of interest. As has been seen, the expert evidence played a crucial role in the judge's decision on the decisive issue of causation, such that the appeal has necessarily involved an attack the judge's findings based on Professor Hull's evidence. To this end, Mr Pulman placed before us a proposed new ground of appeal based on the non-disclosure by Professor Hull of a conflict of interest in the form of his membership of the Cases Committee of the Medical Defence Union ("the MDU"). That conflict, Mr Pulman submitted, was sufficient to require this court to set aside the judge's order, although he did not submit that anything in Professor Hull's evidence was actually influenced by his interest. We are not persuaded by this challenge for reasons which we explain below. However, we consider that it involves important points of principle and practice, and we therefore think it appropriate to make some observations on the general issues raised.

100. We start with the point of principle. Does the presence of a conflict of interest automatically disqualify an expert? In our judgment, the answer to that question is no: the key question is whether the expert's opinion is independent. It is now well-established that the expert's expression of opinion must be independent of the parties and the pressures of the litigation. Authority for this can be found in paragraphs 1 and 2 of the guidance which Cresswell J gave in *National Justice Compania Naviera SA Prudential Assurance Co Ltd* ("the Ikarian Reefer") [1993] 2 Lloyd's Rep.68 as summarised on pages 938-9 of *Civil Procedure* (2006):

"1. Expert evidence presented to the court should be, and should be seen to be, the independent product of the expert uninfluenced as to the form or content by the exigencies of litigation (*Whitehouse v Jordan* [1981] 1 W.L.R. 246, HL, at 256, per Lord Wilberforce).

2. An expert witness should provide independent assistance to the court by way of objective unbiased opinion in relation to matters within his expertise (see *Pollivitte Ltd v Commercial Union Assurance Company Plc* (1987) 1 Lloyds Rep. 379 at 386, per Garland J., and *Re J* (1990) F.C.R. 193, per Cazalet J. An expert witness in the High Court should never assume the role of an advocate. ..."

101. Moreover, CPR 35.3 sets out the overriding duty of an expert witness. His duty is to assist the court in relation to matters which fall within his expertise. The need for the expert to give an independent opinion flows also from this duty, which is stated to override any duty which the expert may owe to his client:

"(1) It is the duty of an expert to help the court on the matters within his expertise.

(2) This duty overrides any obligation to the person from whom he has received instructions or by whom he is paid."

102. However, while the expression of an independent opinion is a necessary quality of expert evidence, it does not always follow that it is sufficient condition in itself. Where an expert has a material or significant conflict of interest, the court is likely to decline to act on his evidence, or indeed to give permission for his evidence to be adduced. This means it is important that a party who wishes to call an expert with a potential conflict of interest should disclose details of that conflict at as early a stage in the proceedings as possible.
103. We first explain Mr Pulman's challenge in more detail before giving our reasons for rejecting it. The thrust of Mr Pulman's challenge is that the decision of the judge should be set aside because the judge preferred the evidence of Professor Hull who had a conflict of interest which was not disclosed to him. The MDU is the body which is defending this claim on behalf of Dr Jarman. The organ of the MDU which made decisions in relation to Dr Jarman's defence was the Cases Committee, of which Professor Hull was a member at the time he wrote his reports. Although Mr Pulman does not suggest Professor Hull was actually biased, he submits the judge should have been told that he was a member of the Cases Committee because his interest and obligations as a member of that Committee might have had a subconscious effect on his evidence. Consequently in default of disclosure, the judge's judgment should be set aside.
104. The MDU is effectively a mutual insurer for medical practitioners who are members of it in relation to clinical negligence claims against them. Mr Pulman draws an analogy between the position of Professor Hull and that of a member of the bar who was also a director of the Bar Mutual Indemnity Fund ("BMIF"). He submits that that member of the bar would have a conflict of interest if he was asked to give expert evidence in professional negligence proceedings brought against a barrister who was insured with BMIF.
105. Mr Pulman also submits that non-disclosure of Professor Hull's conflict of interest means that Mr Toth's right under article 6 of the European Convention on Human Rights ("the Convention") was necessarily violated. We can deal with this point forthwith. The requirement in that article for an "independent and impartial tribunal" relates to the integrity of the tribunal. It does not mean that an expert witness called by the parties must satisfy the same test of independence as a judge is required to satisfy. Mr Pulman complains that Mr Toth's Convention rights to a fair trial were violated in a further respect. He submits that Mr Toth was entitled to be put on an equal footing with Dr Jarman. We deal with this submission below.
106. Miss O'Rourke submits that there was no question of information as to Professor Hull's decision being deliberately or consciously withheld. The appellant made no request for a curriculum vitae ("CV") for Professor Hull. If it had been requested at or about the time of delivery of Professor Hull's report, it would have disclosed Professor Hull's membership of the Cases Committee from 1984 onwards. The CV then in use by Professor Hull and produced in evidence in this court shows just that. However, it is also the position that Professor Hull ceased to be a member of that Committee on 19 November 2002, that is some six months before the trial of the action in 2003. Professor Hull was also a member of the Council of the MDU during the same period; however, at the relevant time, that organ had no responsibility for individual cases. Moreover, following a reorganisation taking effect in about 2000, the role of the Cases Committee was not to take decisions, but simply to give advice

in relation to individual cases. This appears from the witness statement of Mr Nicholas Bowman, secretary of the MDU, adduced on this appeal to counter the additional evidence relied on by Mr Toth.

107. As in the case of the category (1) evidence, Miss O'Rourke's submission is that the new evidence from Mr Toth about Professor Hull's membership of the Cases Committee does not comply with the criteria laid down in *Ladd v Marshall* for the admission of new evidence on appeal, because the new evidence could easily have been obtained before the trial began. However, we do not consider that point should be decisive. While it may very well be that the appellant could have discovered this information prior to trial, we do not consider that the principles in *Ladd v Marshall* provide an answer to the point which Mr Pulman seeks to raise because, if that point is right, the court ought not to have acted on the evidence of Professor Hull, and in those circumstances it would not be in the interests of justice to decline to admit the new evidence.
108. Miss O'Rourke makes a number of submissions about the existence of the conflict of interest and the need to disclose it which we do not accept. First, she contends that it is a sufficient answer to say to Mr Pulman's challenge that, as there was no request for this information, there was no obligation to disclose it. We do not accept this. If there was a conflict of interest which was not obviously immaterial, it should have been disclosed by Professor Hull to the defendant's solicitors and by them to the appellant's solicitors. Nor do we consider it is any answer to say that Professor Hull had no financial interest in the outcome of this case, or that in practice he would not have been asked or been able to sit on any item of business before that Committee arising out of this case, or that the role of that Committee is advisory only. The position is that, so long as he was a member of the Cases Committee, and that Committee had any responsibility for this case, he was in principle subject to a conflicting duty as a member of that committee. It is, moreover, insufficient to say that in fact, under the practice or constitution of the MDU, Professor Hull would have been precluded from attending the proceedings of any meeting of the Committee which considered this case because of his involvement as an expert. The relevant information should have been made available to the other party and the court. The likelihood is that the relevant information would not have been known to Mr Toth or to the court without disclosure and explanation, and it plainly raised a question as to a conflict of interest. In such a situation, the expert should disclose such information to enable the court and the other party properly to assess the conflict of interest.
109. However, that is not the end of the matter. In our judgment, in view of the practice of the Cases Committee to exclude any member of the Committee who was an expert in a particular case from deliberations of the Committee in relation to that case, we do not consider that membership of that Committee would automatically disqualify a person from acting as an expert witness. Moreover, despite the indication in Professor Hull's CV (produced to this court) that Professor Hull was a member of the Cases Committee of the MDU, it is clear from Mr Bowman's statement that he ceased to be a member of that Committee in November 2002, that is, before the trial in this case. In the circumstances, in our judgment, even if Professor Hull's conflict of interest arising out of his membership of the Cases Committee had earlier been a disqualifying interest, it then became immaterial. The same is true of his appointment as a Council member. In fact, at the material time, the function of the Council

appears to have been advisory only. It did not consider individual cases and therefore, in our judgment, appointment as a Council member also did not automatically give rise to a disqualifying conflict of interest. It follows that the non-disclosure of which Mr Toth wishes to complain did not place him on an unequal footing with Dr Jarman at the trial of this action. Mr Toth could, and did, call his own eminent expert witness, and his counsel cross-examined Professor Hull without difficulty. In those circumstances we refuse leave to Mr Toth to raise this new ground of appeal and to adduce the evidence as to Professor Hull's membership of this Committee.

110. We find support for our conclusion in the decision of this court in *Field v Leeds City Council* (2000) 17 EG 165. That was a housing disrepair claim in which the question arose whether the court should give permission for the evidence of a surveyor employed by the City Council to be adduced as expert evidence. It was argued that the surveyor could not bring the necessary objectivity to bear on the issues in the case. This court did not hold that the surveyor could not be an expert witness in the proceedings. The court held that there was no assumption that an employee could not be an expert witness. Lord Woolf held that the objection could in appropriate circumstances have some force, but that the judge in that case could not decide whether the surveyor was qualified to give expert evidence without more information to satisfy himself that he was aware of the duties of an expert witness, in particular the need for objectivity.
111. Nonetheless, we propose to go on to consider whether Professor Hull should have disclosed his membership of the Cases Committee at or about the time of delivering his report, because that question has implications beyond this case. We also consider below the form of declaration annexed to the expert's report. We proceed on the basis that the time for disclosing the existence of a possible conflict of interest is when the report of the expert is first served on the other parties. It may be earlier, if the permission of the Court is sought to adduce a particular named expert witness. If, however, the conflict of interest only arises after that time, the appropriate time for disclosure will be the first practicable date thereafter.
112. Miss O'Rourke suggests that, when the report of Professor Hull was served on the appellant, the view was likely to have been taken that his membership of the Cases Committee was an immaterial conflict of interest, that that was a reasonable view to take and that, therefore, any non-disclosure should be excused. We can understand that (in the absence of guidance from the court) a party who calls an expert witness at trial, or serves an expert's report in advance of trial, may be aware of a potential conflict of interest but consider that it is not material and that it therefore need not be disclosed. However, for the future, we do not consider that a party should take the course of non-disclosure. We say this because it is for the court and not the parties to decide whether a conflict of interest is material or not. The court may take a different view from that of the parties as to whether an expert has a conflict of interest which might lead the court to reject the independence of his opinion: see, for example, *Liverpool Roman Catholic Archdeacon Trustees Inc v Goldberg* (No 2) [2001] 1 WLR 2337. Similarly, in the interests of transparency and of deflecting suspicion, the other party ought to have the information as soon as possible. We do not consider that the parties can properly agree that a conflict of interest which is otherwise disclosable need not be drawn to the attention of the court. A party who is in the position of wanting to call an expert with a potential conflict of interest (other than of

an obviously immaterial kind) should draw the attention of the court to the existence of the conflict of interest or possible conflict of interest at the earliest possible opportunity. By the same token, it is obviously desirable for the other party to make any objection that it may have to the admission of expert evidence at as an early a stage in the proceedings as practicable. It follows that, in this case, we consider that Professor Hull's position as a member of the Cases Committee and Council of the MDU should have been disclosed in, or at the time of, his report.

113. The obligation to disclose the existence of a conflict of interest in our judgment stems from the overriding duty of an expert, to which we have already referred and which is clearly laid down in CPR 35.3, and also from the duty of the parties to help the court to further the overriding objective of dealing with cases justly (CPR 1.3). The court needs to be assisted by information as to any potential conflict of interest so that it can decide for itself whether it should act in reliance on the evidence of that expert.
114. We note that the practice of the court as set out in Civil Procedure Rules and practice directions, the valuable new Protocol for the Instruction of Experts to give evidence in civil claims (set out at p966-977 *Civil Procedure* (2005)), and the commentaries in *Civil Procedure* and *Civil Court Practice*, do not refer in terms to the need for disclosure by an expert of a conflict or potential conflict of interest. We also note that the standard form of order used by Master Ungley and Master Yoxall, to whom clinical negligence matters in the Queen's Bench Division in London are assigned, requires the production of an expert's CV but this is only where the court has directed a single expert, and the parties cannot agree on who it should be. As already stated, in our judgment, an expert should produce his CV when he provides his report, and that CV should give details of any employment or activity which raises a possible conflict of interest. This may indeed already be best practice.
115. The practice direction supplementing CPR 35 provides that an expert must verify his expert's report by a statement of truth in the following form:

“I confirm that insofar as the facts stated in my report are within my own knowledge I have made clear which they are and I believe them to be true, and that the opinions I have expressed represent my true and complete professional opinion.”
116. In addition, the practice direction also requires the expert's report to state any qualification, if the expert is not able to give his opinion without qualification, and that the expert understands his duty to the court and has complied and will continue to comply with that duty.
117. An expert will often make a declaration at the end of his report which not only contains the statement of truth required by the practice direction but also contains other matters designed to meet other requirements of the CPR or practice directions, or other issues which have arisen in practice. No doubt, in principle these declarations serve the valuable purpose of focusing the mind of the expert on all these matters.

118. In this case there was an extended declaration by Professor Hull. There was some discussion in argument before us as to whether the declaration he signed in fact covered the question of a conflict of interest. He signed his report on 13 June 2001. The declaration at the end of the report stated that he had “drawn to the attention of the Court all matters, of which I am aware, which might adversely affect my opinion”. That statement is, on the face of it, directed at ensuring that the expert has fairly disclosed matters which might cause him to give an opinion *less* favourable to his client, rather than a conflict of interest which might have the opposite effect.
119. In our judgment, the Civil Procedure Rules Committee should consider extending the requirement for an expert’s declaration at the end of his report. Its present form is directed to ensuring that the contents of the report represent the independent and unvarnished opinion of the expert making the report. But, as we have explained above, there is another side to independence. The expert should not leave undisclosed any conflict of interest which might bring into question the suitability of his evidence as the basis for the court’s decision. The conflict of interest could be of any kind, including a financial interest, a personal connection, or an obligation, for example, as a member or officer of some other body. But ultimately, the question of what conflicts of interest fall within this description is a question for the court, taking into account all the circumstances of the case.
120. Without wishing to be over-prescriptive or to limit consideration by the Civil Procedure Rules Committee, we are of the view that consideration should be given to requiring an expert to make a statement at the end of his report on the following lines:
- i) that he has no conflict of interest of any kind, other than any which he has disclosed in his report;
 - ii) that he does not consider that any interest which he has disclosed affects his suitability as an expert witness on any issue on which he has given evidence;
 - iii) that he will advise the party by whom he is instructed if, between the date of his report and the trial, there is any change in circumstances which affects his answers to (a) or (b) above.
121. As we see it, a form of declaration to this effect should assist in reminding both the expert and the party calling him of the need to inform the other parties and the court of any possible conflict of interest. Thus pending any further consideration by the Civil Procedure Rules Committee, we give the guidance set out above.

The application to restore the appeal for further hearing

122. Having reserved judgment in this case on 9 March 2006, the court received a letter dated 22 March 2006 from Mr Toth’s solicitors requesting that the court “re-open these proceedings, notwithstanding that the ordinary appeal process has been concluded and direct that there be a re-hearing of the appeal”. The letter cites the decision of this court in *Taylor v Lawrence* [2003] QB 528 as authority for the jurisdiction of the court to take such a course. The ground on which such course is said to be justified is that

“... Counsel representing the Appellant in the appeal, Mr George Pulman QC, (a) had not prepared adequately (b) did not comply with the instructions given to him both by the firm and the appellant (c) did not present to the court all the relevant arguments in relation to the appeal and (d) presented his arguments on the appeal frequently in a way that was neither coherent nor accurate.”

The letter then sets out at length criticisms concerning the degree and extent of leading counsel’s preparation and presentation of the case, in a situation where the decision whether or not legal aid would be granted for the appeal had been delayed until a week before the hearing. The letter contains a lengthy and sustained attack on the manner in which Mr Pulman conducted the appeal, setting out a number of instructions given and allegedly overlooked, or not followed, suggesting that Mr Pulman misunderstood the case and failed to take various points available to be taken.

123. The particulars of the matters complained of set out, under paragraphs labelled (a)-(g), various matters (stated to be “non-exhaustive”) in support of “the core of the Appellant’s case ... that he did not receive a fair trial”, to which we will shortly turn. It is stated that the matter in (a) was not dealt with fully, adequately or accurately and that the matters in (b)-(g) were not put at all by Mr Pulman on the appeal. The letter complains of failure by Mr Pulman to articulate Mr Toth’s case on causation, which is stated to be quite simple, and then lists a number of further respects (a)-(f) in respect of which his presentation of the appeal was said to be deficient.
124. Having carefully considered the contents of the letter, the court replied on 12 April 2006 by letter from the Deputy Master of Civil Appeals, stating that it was not prepared to grant the request or to grant various Directions set out at the end of the solicitors’ letter. The letter stated:

“...It is the view of the court that there is nothing of substance in the contents of points (a)-(g) of your letter, set out as illustrative of the core of the appellant’s case that he did not receive a fair trial, which was not made clear to the court as a matter of complaint in the course of the argument. Nor does the court consider that the further series of points (a)-(f) set out as the respect in which it is asserted that leading counsel’s presentation of the appeal was deficient are matters which, even if established, would alter the view formed by this court as to the appeal on liability.

Consequently, the members of the court propose to proceed with the preparation of their judgment, which will not now be ready until next term...”

125. This letter elicited a further argumentative response from Mr Toth’s solicitor dated 10 May 2006 requesting the court to treat the letter of 22 March, as supplemented by the letter of 10 May 2006 as a formal application to the Court of Appeal. By letter dated 12 June 2006 from the Deputy Master of Civil Appeals the court informed Mr Toth’s solicitors that the application was refused for reasons which would be stated in this judgment. Our reasons now follow.

126. The application is highly unusual. It is not one which seems to us to fall strictly within the jurisdiction of the Court of Appeal considered in *Taylor v Lawrence* which, as the opening lines of the judgment in that case make clear, relates to the power of the Court of Appeal to revisit an appeal after it has given final judgment and that judgment has been drawn up. That is, of course, not the case here, albeit the court indicated what its decision would be at the time of reserving judgment.
127. We do not doubt, that, in exceptional circumstances, the court has power in such a case to recall the matter for further argument on the basis of some unforeseen development, or some serious error or omission brought about by oversight of counsel. However, that is plainly not the basis of the application in this case, which is based on a general allegation of incompetence against counsel conducting the appeal. In this respect, we can say at once that, while it was evident to us that, from time to time in the course of the argument, Mr Pulman was having difficulty in making good the grounds of appeal, that was, not for reasons of incompetence, but because the judgment under challenge was a careful and considered one with no apparent errors of reasoning, having been resolved by preference for the evidence of one eminent expert over another. In our view, the new evidence sought to be relied on in truth added little, if anything, further to illuminate the issues before the court. Finally, to the extent that any of Mr Pulman's submissions might otherwise have been left unclear following their deployment, they were supplemented in writing as the case proceeded.
128. It seems to us, that the allegations of incompetence are based less upon objective considerations, than upon the subjective perceptions of Mr Toth and his solicitor, Mr Saffron. The latter's evident sympathy for, and identity with the cause of Mr Toth in the sad circumstances of this case have led him to a regrettable intemperance of expression at the expense of Mr Pulman, whose appearance before the judge at trial had plainly been of sufficient quality to lead to his continued retention as leading counsel in the appeal. That fact alone makes it unlikely that Mr Pulman did not understand the issues on the appeal.
129. In Mr Saffron's letter to the court dated 10 May 2006, when urging the court to re-open the matter, he stated:

“The entire thrust of our letter to the court is that for reasons not known (some, of course, attributable to the LSC) Leading Counsel failed through oversight or lack of time or for some other reason to present the Appellant's case in the manner instructed or authorised.”

This passage, and indeed the grounds of application set out in the earlier letter, appear to us to be based upon the assumption that counsel is no more than the creature or conduit pipe of his client for the purpose of addressing the court. That is not, of course, the case. The advocate's duty to represent his client to the best of his ability and to carry out his client's instructions does not require him to do so in such a way as to preclude his own judgment as to the points which are better emphasised on the one hand, or to be argued but lightly or even omitted on the other. Accordingly, there is a heavy burden upon any appellant such as Mr Toth in seeking a re-hearing of his appeal on the grounds of errors made by counsel in the course of argument. In his letter of 10 May 2006, Mr Saffron has referred at length to a number of cases decided by this court in its criminal jurisdiction when dealing with the question whether the

failings of counsel may directly or indirectly lead to a conclusion that a conviction is unsafe. We do not find those decisions helpful as a guide to the position in civil appeals in which it will usually be wholly inappropriate to embark on any examination of the state of the instructions as between counsel and those sitting behind him.

130. However, we do not consider it necessary further to consider that question because we are content, having carefully considered the points taken in Mr Saffron's letters, to deal with the matter on the merits of the points taken. They can be dealt with summarily in the light of the examination of the issues which we have already conducted. We turn to the letter of 22 March 2006 for that purpose.
131. As to the submission that the core of Mr Toth's case was that he did not receive a fair trial and the complaints set out in that respect, the position is as follows.
132. As to point (a), contrary to the assertions on behalf of Mr Toth, Mr Pulman made us well aware of the respects in which it was stated that Professor Hull had failed to comply with the obligations imposed on him as an expert under the CPR in the respects. We do not accept the assertion that, by the third day of the appeal, Mr Toth's case had been reduced to reliance solely on the failure of Professor Hull to disclose a potential conflict of interest. It is certainly right to say that, by that stage, the weaknesses of the appeal had been revealed for the reasons we have already set out. It is asserted that Mr Pulman had no instructions to limit the presentation of Mr Toth's case to such a significant and detrimental extent. He did not do so.
133. As to points (b)-(g), the assertion that these points were not put at all by leading counsel is a misrepresentation of the position. Sub-paragraph (b) deals with the issues of causation. It asserts that the judge made findings without any evidence to support them as to the sufficiency of the oral glucose administered and as to the ability of the brain to find the way of obtaining a substance which it needs. Those matters have already been dealt with by us at paragraphs 73 to 74 above on the basis that the points were indeed taken by Mr Pulman.
134. Sub-paragraph (c) is the same point in a different guise i.e. the intuitive observation of the judge, which we have criticised but found to be immaterial.
135. Sub-paragraph (d) complains that the judge was not entitled to make a finding that the appellant was not a reliable witness, given that, in relation to the causation issue, everything he said was supported by the evidence of Ms Gover. While that may be an observation which caused concern to Mr Toth, nothing hinged upon it in relation to the causation issue as decided by the judge.
136. Sub-paragraph (e) is irrelevant, relating as it does to the issue of quantum.
137. Sub-paragraph (f) asserts that the trial judge acted unreasonably / irrationally in preferring the "unsupported and anecdotal evidence" of Professor Hull in the face of the "substantial scientific and medical literature, and the written opinions of other eminent paediatricians". This point was taken by Mr Pulman and we have dealt with it at length in this judgment.

138. Sub-paragraph (g) asserts that the trial judge should have treated the submissions of the MDU at trial with caution having regard to the fact that the Court of Appeal had found in December 2000 that the MDU had accepted in 2001 that the MDU had misled the court on its strikeout application and had continued to defend the negligence allegation despite being in possession of a GP's report which confirmed the negligence of the defendant. Those paragraphs are a tendentious and self-serving version of events earlier in the proceedings, which in any event went to the question of negligence, (on which the judge found for Mr Toth,) and not to the issue of causation.

139. So far as the criticisms of Mr Pulman are concerned, the principal criticism is that he

“...failed in an incoherent way to explain the relevance of the guidance in the textbooks and hospital protocol to the issue of causation, as distinct from the issue of negligence

Leading counsel's submission was essentially that the guidance in these documents is there for a reason and that that reason is because the recommended treatment is expected to work. Such a submission wholly fails to articulate the appellant's case, which is in fact quite simple. *The point is that the consistent recommendation is that there should be repeated intravenous injections or infusions of glucose following the oral administration of glucose precisely because (a) intravenous administration of glucose is effective and (b) there is an expectation that the glucose administered orally will not be effective....* Professor Hull's categorical view, however, was that intravenous glucose would not have worked in Wilfred's case because the oral glucose administered by the Appellant would have worked and the fact that it did not showed that it was already too late. Such a firm conclusion is unsupportable having regard to the literature, for which Professor Hull was either responsible or of which he would have been aware, making it quite plain that oral glucose does not always work. This forcible point was entirely lost in the incoherence of the presentation. What was also lost was the supplemental point was that Professor Hull failed to disclose relevant material adverse to his opinion.” (emphasis added)

140. We do not accept any of this criticism as correct. All the points made were fully appreciated by us as a result of Mr Pulman's submissions when we indicated our decision. Mr Pulman addressed us on causation and not upon negligence. He made the point of principle, supported by the literature, that IV administration of glucose is expected to be effective. However, he did not submit, and was correct not to submit, that there was an expectation that IG administration of glucose would *not* be effective. He was correct not to do so because, as already indicated, Professor Marks had himself recognised, as previous experience with Wilfred confirmed, that IG glucose was ordinarily expected to work. Indeed, the textbooks and protocols put forward by the claimant, in so far as they refer to the topic, also indicate the same expectation.

141. It is misleading to refer to Professor Hull's "categorical view", in order to criticise his firm conclusion as "unsupportable having regard to literature". His evidence was at almost every stage given in terms of possibility, probability or likelihood, premised on his view that he would have expected IG glucose to work if Wilfred's brain had not been irreversibly damaged by 7.15am. In that respect he said "oral administration of glucose can be expected to work and work well and work very quickly" in "a vast majority" of cases.
142. So far as the other respects in which Mr Pulman's presentation of appeal is said to be deficient, our view is as follows.
143. As to sub-paragraph (a), we do not accept that leading counsel did not understand (or at any rate make clear to us) the distinction between hypoglycaemia and profound hypoglycaemia and the effect of each on the brain. He was circumspect on occasion in answering a question directly by the court concerning the stage at, or extent to which, the use of such expressions was appropriate. However, such hesitation is often a wise course for the avoidance of unconscious error and we do not consider that Mr Toth's case was in any way prejudiced by such hesitation.
144. As to sub-paragraph (b), we did not find that Mr Pulman was inaccurate or contradictory when dealing with the issue of the time by which Dr Jarman ought reasonably to have been expected to have arrived at the appellant's house. The judge made no finding on the question, on the basis that the decision of timing in that regard was not conclusive on the issue of causation. We have dealt with the appeal on that basis. Neither Mr Pulman nor Mr Saffron have persuaded us of error in this respect.
145. It is complained in sub-paragraph (c) that Mr Pulman, despite instructions, did not ask that the issues be remitted to the trial judge. We do not recall whether or not that is so. However it is in our view not a matter of concern in the light of the conclusion we have reached.
146. At sub-paragraph (d), it is complained that Mr Pulman did not pursue an application in relation to requests under CPR Part 18 notwithstanding instructions to do so. We are not clear what these requests were and therefore can say no more about them. It is also complained that he did not ask for time to consider late material produced by the defendant's advisers on the last day of the appeal relating to Professor Hull's membership of the MDU Committees. This appears to be a reference to the statement of Mr Nicholas Bowman to which we have referred. The position in relation to this evidence had in fact been flagged up by Miss O'Rourke earlier in the hearing. No doubt Mr Pulman regarded it as truthful and straightforward and considered that it did not require substantial time to consider.
147. Complaint is made at sub-paragraph (e) that Mr Pulman did not object to the late submission to the court by the defendant of a letter from Professor Hull, despite his instructions that he should so object. This was no doubt a matter which Mr Pulman considered in the exercise of his discretion as counsel in charge of the case, deciding to do that which he thought most appropriate in the conduct of the appeal. The letter referred to contained Professor Hull's brief comments upon Mr Saffron's witness statement in support of the application to call new evidence. It was not itself concerned to assert new evidence, but to comment on observations made by Mr Saffron as a layman on the content of Professor Hull's research.

148. At sub-paragraph (f) the complaint is made that Mr Pulman frequently referred to Ms Gover as Miss Gove in the course of appeal. No doubt the error was triggered because a similar error appears throughout the judge's judgment below. We attach no importance to it.
149. The above are the main points taken in support of an application based, as we have said, upon the alleged failure of Mr Pulman to place Mr Toth's arguments properly before the court. In the light of our observations, it will be apparent that we find no substance in the points raised. Indeed, we are satisfied that all relevant matters were properly before us. In those circumstances we have no hesitation in rejecting the application contained in the letters of 22 March and 10 May 2006.

Conclusion:

150. The appellant's application to adduce new evidence upon the appeal and his substantive appeal against the judgment below will both be dismissed.