



Neutral Citation Number: [2026] EWHC 704 (KB)

Case No: KB-2023-MAN-000306

**IN THE HIGH COURT OF JUSTICE**  
**KING'S BENCH DIVISION**  
**MANCHESTER DISTRICT REGISTRY**

Manchester Civil Justice Centre  
1 Bridge Street West, Manchester M60 9DJ

Date: 31<sup>st</sup> March 2026

**Before :**

**HER HONOUR JUDGE CLAIRE EVANS**  
**(sitting as a Judge of the High Court)**

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**Between :**

**MR CALLUM JOE GUEST**

**Claimant**

**- and -**

**NOTTINGHAM UNIVERSITY HOSPITALS**  
**NHS TRUST**

**Defendant**

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**Jason Wells** (instructed by **Fletchers Solicitors**) for the **Claimant**  
**John Coughlan KC** (instructed by **Browne Jacobson LLP**) for the **Defendant**

Hearing dates: 12<sup>th</sup> to 18<sup>th</sup> March 2026

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**Approved Judgment**

This judgment was handed down remotely at 10.30am on Tuesday 31<sup>st</sup> March 2026 by circulation to the parties or their representatives by e-mail and by release to the National Archives.

**Her Honour Judge Claire Evans :**

1. This is the trial of breach of duty and causation in a clinical negligence claim following spinal surgery which the Claimant underwent at the Defendant's hospital on 29<sup>th</sup> May 2019. The surgery was long and complex. During its course the Claimant's condition deteriorated and a decision was made to stop the operation. It was after that decision was made, and whilst the Claimant was being turned from the prone position he had been in throughout the operation to supine that he suffered a cardiac arrest. He was successfully resuscitated but it subsequently transpired that he had at some point suffered posterior ischaemic optic neuropathy (PION), causing substantial bilateral loss of vision.
2. PION is a recognised albeit rare complication of the Claimant's surgery. In this case, however, he claims that it arose as a result of the cardiac arrest rather than as a complication of the surgery itself. The cardiac arrest, he claims, was due to hyperkalaemia (high serum potassium) which the treating anaesthetist negligently failed to treat.
3. Over the five days of the trial I read evidence from the Claimant and his parents and heard evidence from the treating consultant anaesthetist Dr Leong. The expert evidence consisted of anaesthetic evidence on breach of duty and causation from Dr Power for the Claimant and Dr McCrirrick for the Defendant, cardiology evidence on causation from Dr Clifford for the Claimant and Dr Norell for the Defendant, and ophthalmology evidence on causation from Dr Eke and Dr Elston, all of whom gave oral evidence.

**The relevant facts**

4. The Claimant's surgery was intended to address his scoliosis and associated chest wall deformity. It was a two stage process. The operation with which I am concerned was the second stage of the process. The anaesthetic was started at approximately 09:30 on 29<sup>th</sup> March 2019. The Claimant was turned prone at 11:00. The surgical procedure began at 11:45.
5. Bloods were taken regularly throughout the surgery. The blood samples were taken from a radial arterial line. Dr Leong's evidence was that she or her specialist registrar would have taken the bloods (save for the one taken at 19:34 taken during the cardiac arrest where her evidence was that she did not know who had taken that one because there would have been any number of staff in the room at that point).
6. Once taken, the blood sample would be taken by the Operating Department Practitioner (ODP) to the arterial blood gas (ABG) machine which gives a rapid result for various parameters, to the thromboelastograph (TEG) machine, which in layperson's terms shows a real time formation of a blood clot to give information about various matters relating to blood clotting, and sometimes also to the laboratory

for a full blood count (FBC) and other clotting results. The ABG results are typically returned within 5 minutes, the TEG in some 15 to 20 minutes. The lab results can take in the region of an hour or more.

7. The arterial blood gas (ABG) results for the duration of the Claimant's operation appear in the table below, which is a table prepared by Dr Leong which I have amended to show the exact times that the ABG results were printed from the relevant machine.

<b>Time</b>	<b>10:00</b>	<b>13:20</b>	<b>14:14</b>	<b>16:07</b>	<b>17:38</b>	<b>18:28</b>	<b>19:34</b>	<b>19:51</b>
<b>FiO<sub>2</sub></b>	60	40	40	40	40	100	100	100
<b>pH</b>	7.462	7.359	7.388	7.353	7.282	7.257	7.173	7.296
<b>pO<sub>2</sub></b>	35.3	24	21.6	13.5	9.33	11.6	9.81	38.2
<b>pCO<sub>2</sub></b>	5.5	5.98	5.62	5.99	6.87	5.8	5.9	10.4
<b>Hb</b>	125	114	98	96	110	107	119	92
<b>Hct</b>	38.4	34.9	29.9	29.3	33.7	32.8	36.5	28.1
<b>lactate</b>	0.9	2.2	2.8	2.6	2.0	3.0	6.8	8.3
<b>BE</b>	+5.6	-0.2	+0.4	-0.6	-2.4	-7.7	-12.2	-11.7
<b>HCO<sub>3</sub><sup>-</sup></b>	29.1	24.1	24.7	24.9	24.3	19.4	16.2	38.1
<b>Na<sup>+</sup></b>	137	137	136	135	134	136	130	150
<b>Ca<sup>++</sup></b>	1.17	1.17	1.25	1.26	1.20	0.59	0.97	1.32
<b>K<sup>+</sup></b>	4.1	4.4	4.3	4.7	5.2	6.2	9.5	5.2

Key: FiO<sub>2</sub> – inspired oxygen concentration, pCO<sub>2</sub> – carbon dioxide partial pressure, pO<sub>2</sub> – oxygen partial pressure, Hb – haemoglobin, Hct -haematocrit, BE – base excess, HCO<sub>3</sub><sup>-</sup> – bicarbonate, Na<sup>+</sup> - sodium, Ca<sup>++</sup>- calcium, K<sup>+</sup> - potassium

8. The surgery was, as I said, long and complex and involved significant blood loss. It was challenging to manage the Claimant's blood pressure which dropped on a number of occasions. By 18:00 the Claimant had been given some 11 litres of fluids (9 litres of Hartmann's solution which is a crystalloid solution containing potassium, 500 ml of a colloidal solution, 652 ml of cell-saved blood and 2 units of allogenic blood).
9. In terms of the Claimant's potassium level, it can be seen from the blood results table that it was rising slowly during the course of the operation. At 17:38 it had risen to 5.2 mmol/l from 4.7. Dr Leong described it as borderline high at this point but not at a level where she would routinely treat it: she would reassess with another ABG in an hour's time. At that stage her main concern was the bleeding and his increasing instability.

10. At 18:10 Dr Leong called for a surgical pause. The Claimant was on very substantial doses of metaraminol (a drug used to raise blood pressure) but notwithstanding that and the fluids (now saline fluid rather than a potassium containing fluid) he continued to be unstable and she was not content to continue. The decision was made to stop the surgery and to close the surgical wound which would take some 40 minutes.
11. The next ABG was done at 18:28. It showed a serum potassium level of 6.2. That is a level which Dr Leong and the expert anaesthetists agree warrants treatment. Had she seen that result she would have treated the Claimant with calcium gluconate, which has the effect of stabilising the cardiac muscle membrane and thus protecting against cardiac arrest. According to the records, the result did not in fact arrive in the theatre until 19:00, there subsequently being a suggestion that the result had been delayed whilst the ODP attempted to mend the broken TEG machine. Dr Leong's evidence in cross-examination was that she did not actually recall whether she ever saw that result. Whether she did or not, she did not act upon it.
12. From 19:00 preparations were being made to turn the Claimant from prone to supine. Dr Leong described that as her pressing priority, given the increasing likelihood of needing to perform CPR and the difficulty of doing that on a patient in the prone position.
13. Some ECG changes are noted (but not particularised) on the records at 19:15. Dr Leong told me that they were not typical of hyperkalaemia which she said she understood to give rise to huge peaked T waves.
14. Between 19:20 and 19:25 all the monitors and infusions were disconnected in order to turn the Claimant supine. Once the turning had been done and the monitors were reconnected, very quickly there was a drop in end-tidal CO<sub>2</sub> and the Claimant went into cardiac arrest due to Pulseless Electrical Activity (PEA). CPR was started and he was given calcium gluconate, sodium bicarbonate and adrenaline. On the third round of CPR the PEA rhythm converted to Ventricular Fibrillation (VF) and the defibrillator was used successfully. The total "downtime" was some 10 minutes. End-tidal CO<sub>2</sub> was present throughout CPR.
15. Very shortly after the Claimant went into cardiac arrest a further blood sample was taken, the ABG results for which were printed at 19:34. They show a rise in serum potassium to 9.5 mmol/l. The next ABG results were printed at 19:51 and show a fall to 5.2 mmol/l.
16. At 20:00 on the same day the Claimant underwent a bedside echocardiogram in the ICU. It was noted as "LV [left ventricle] hyperkinetic/ RV [right ventricle] mildly moderate dilated, N [normal] function TAPSE 2.2 no pericardial fluid."

17. At the same attendance his face was noted as “++ swollen” which accords with the evidence of his parents who both describe seeing him on the evening of the surgery and his face being so swollen that he was unrecognisable.
18. At 20:25 the Claimant underwent a pulmonary angiogram (CTPA). It was reported as “filling defects seen within the left upper lobe lingula segmental and subsegmental pulmonary arteries as well as the lobar and segmental pulmonary arteries of the right lower lobe”. The cardiology experts agree this indicates bilateral pulmonary emboli within the left upper lobe, left lingula and right lower lobe. It did not show a central embolism.
19. At 23:35 the Claimant underwent a transthoracic echocardiogram. It was recorded as having been a technically difficult study. It relevantly reported the right ventricle as “non dilated with normal systolic function. Tricuspid annular plane systolic excursion (TAPSE) 1.6 cms.”
20. It became apparent in the days and weeks following the surgery that the Claimant had suffered bilateral PION. He has almost no vision at all in the right eye and severe impairment of vision in his left eye.

### **The issues in the case**

21. By the end of the trial the anaesthetic and cardiology experts were agreed that there were two possible causes of the cardiac arrest, namely hyperkalaemia or pulmonary embolism. They were also agreed that if the 19:34 serum potassium level of 9.5 mmol/l was a true result, accurately reporting the amount of serum potassium in the Claimant’s blood at the time of the sample, then on the balance of probabilities the arrest was due to hyperkalaemia, whereas if it was a spurious result then, on the balance of probabilities, the cause was pulmonary embolism.
22. The first issue, then, is the cause of the cardiac arrest.
23. Pulmonary embolism is a non-negligent cause of cardiac arrest. If the arrest was due to hyperkalaemia then the Claimant’s case is that prompt treatment of his raised serum potassium shown in the 18:28 ABG result would have had the effect of averting the cardiac arrest. In fact, as can be seen from the summary of the facts above, no such treatment was given until after he had gone into cardiac arrest.
24. The Claimant alleges that the Defendant was in breach of duty in not acting on the 18:28 ABG result by, at the latest, 19:00. The Defendant admits breach in not acting upon the result once it was available at 19:00 and pleads that treatment should have commenced by 19:20. Whilst not admitted in the Defence, the relevant experts agreed in evidence that treatment by way of calcium gluconate at or before 1900 would on the balance of probabilities have averted the cardiac arrest. It is denied in the Defence

that treatment commenced at 19:20 would have averted the arrest, and the experts disagreed on that point of causation.

25. The second and third issues, then, which only arise if the cause of the cardiac arrest was hyperkalaemia, are the extent to which the Defendant was in breach of duty in relation to treatment of the hyperkalaemia, and whether that breach of duty caused the cardiac arrest.
26. The fourth issue, which only arises if breach of duty and causation of a hyperkalaemic cardiac arrest are proved, is whether the PION was caused (on a “but for” basis) by the cardiac arrest. The fifth issue is whether if such causation cannot be proved, the Claimant can prove that it was contributed to by the cardiac arrest, and/or that its severity was exacerbated by the cardiac arrest.
27. I turn then to the first issue, whether the cardiac arrest was caused by hyperkalaemia or pulmonary embolism.

### **The cause of the cardiac arrest**

#### **Hyperkalaemia – the expert evidence**

28. If the result of 9.5 at 19:34 is a true result then the Claimant’s serum potassium level rose by 3.3 mmol/l in just over an hour, and (after the administration of sodium bicarbonate) fell by 4.3 mmol/l in less than 20 minutes. Dr Power for the Claimant accepted that these figures were unusual and exceptional.
29. In terms of the rise in potassium, Dr Power’s evidence was that, given the result of 9.5 at 19:34, the likely potassium level immediately before the arrest was above 8 mmol/l, accepting that the cardiac arrest will itself have caused some rise in the levels. He ascribed the rise in potassium from 5.2 to somewhere above 8 mmol/l to transcellular shift: the movement of potassium from the intracellular space to the extracellular space, which he said had occurred as a result of cell damage, particularly damage to muscle, and evolving acidosis.
30. He accepted that he had never in practice seen a rise of this magnitude during planned surgery with an anaesthetist present, nor was there any such reported case in the literature. He accepted there was no literature describing rises even as high as 8 due to this theory of transcellular shift, but maintained it was a plausible physiological explanation.
31. He disagreed that the cardiac arrest itself was capable of causing such a rise in potassium levels (a point which was subsequently accepted by Dr McCrerrick). Similarly he disagreed with Dr McCrerrick’s theory of haemolysis of the 19:34 sample. He accepted that haemolysis was an extremely common problem. He said it generally occurred if the sample is not fully flowing, if it is difficult to obtain from the

artery or vein, or if using a tourniquet: he said one tends to see less haemolysis if taking a clean sample from an arterial line and that haemolysis in those circumstances would be “pretty unlikely”.

32. Dr Power accepted that the post-arrest drop in potassium levels was a very significant drop and that calcium gluconate does not have any effect on potassium levels. He ascribed it to the administration of sodium bicarbonate, although he accepted that the 2020 Renal Association Clinical Practice Guidelines - Treatment of Acute Hyperkalaemia in Adults (“Renal Association Guidelines”) said that there was little evidence that sodium bicarbonate lowered serum potassium and that the rationale for its use in hyperkalaemic cardiac arrest was to mitigate the effects of metabolic acidosis which exacerbates hyperkalaemia.
33. Turning to the Defendant’s evidence, Dr McCrirrick’s evidence was that once the administration of blood and potassium containing fluid was stopped (that is, before the sample reported at 18:28 was taken) one would not expect a further increase in serum potassium, in fact one would expect to see a gradual decrease. He rejected Dr Power’s theory of transcellular shift, on the basis that if it was able to cause such a significant rise then marked intra-operative rises of serum potassium would be common, which they are not. He noted that Dr Leong, in taking ABGs every hour or so, was very conscientious; he would usually measure every 2 to 3 hours in major operations. If such rises could occur during major surgery, so that there could be a rise from a normal result of 5.5 to a dangerously high result of 8.5 in an hour, then anaesthetists would need to be measuring serum potassium every 15 to 20 minutes to avoid this type of catastrophe. He said this simply does not happen.
34. Similarly he rejected the suggestion that there was an evolving acidosis, noting that the pH at 17:38 and 18:28 was unremarkable and showed only slight deterioration.
35. He accepted that muscle injury and acidosis could cause rises in serum potassium level but he said one would expect small increases, of say 0.2 or 0.3 mmol/l per hour, not rises of this magnitude.
36. His view was that the result was almost certainly spurious. He described such a rise as “almost inconceivable”. His view was that the true potassium level immediately before the cardiac arrest was around 6 given that no potassium containing solutions had been administered in the previous hour.
37. In his initial report he posited that the cardiac arrest itself led to the significant rise. In the joint report he gave an alternative explanation of haemolysis of the 19:34 sample. Haemolysis resulting in falsely high potassium readings is, he said, an extremely common problem.

38. In cross-examination he abandoned the suggestion that cardiac arrest itself could have caused the rise in the serum potassium level to 9.5, which he said he had accepted in the course of the joint meeting. He said that one could not identify a haemolysed sample by appearance, one identified it by the fact that it gave a result which was an outlier. He said that anything which allows the red cells to leak potassium into the sample can cause haemolysis leading to falsely high potassium levels, giving the examples of shaking the sample or dropping it. He said there are multiple papers reporting falsely high potassium levels as a result of haemolysis, although he accepted that the paper he produced in support [Saleh-Anaraki K, et al. Pseudohyperkalaemia: Three Cases and a Review of Literature. The American Journal of Medicine Vol 135 July 2022, p 150-154] dealt with only three cases, each relating to phlebotomy methods in venepuncture, whereas here the sample was drawn from an arterial line.
39. He accepted that all of the (large number of) other samples drawn from the line gave normal results. He noted that blood gases in ICU are always taken from arterial lines but a small proportion are nonetheless haemolysed. He noted that the circumstances in which this sample was taken were different from those of the other samples, in that this one was taken in the course of the cardiac arrest. Usually the effect of the arterial blood pressure is that the syringe fills up without needing to use any force in taking the sample, but he posited that during a cardiac arrest the pressure would be much lower and so one could envisage having to use force to take the sample. In any event the person taking the sample would be working under stress in an emergency situation. Those might be good reasons why this sample was different from the others. It was suggested to him that there was no documented difficulty of getting the sample, but he said it would not necessarily be documented, he was not sure that he himself would document that during a cardiac arrest.
40. He thought that overwhelmingly on the balance of probabilities the cause of the 9.5 result was haemolysis rather than an unexplained “bizarre” increase where no potassium was being introduced.
41. As to the drop in potassium levels at 19:51, he said that the administration of sodium bicarbonate could reduce the level by anything up to 0.4 mmol/l but to go down by that amount was verging on the impossible.
42. Both anaesthetic experts agreed that hyperkalaemic cardiac arrests generally present as a VF arrest rather than PEA arrest (Dr Power said one “generally expects” a VF arrest, Dr McCrerrick said the “vast majority” present with VF) and that cardiac arrests due to pulmonary emboli typically present as PEA arrest. Both cardiology experts said that hyperkalaemic cardiac arrest could present as either PEA or VF.

### Pulmonary Embolism – the expert evidence

43. Taking Dr McCrerrick's evidence first, his opinion was that the most likely cause for the arrest was pulmonary embolism for which he said the evidence was strong. Pulmonary embolism is the cause of 5 to 13% of all cardiac arrests. The development of deep vein thrombosis (the precursor to pulmonary embolism) is very common in patients such as the Claimant who had been immobile and bedbound for some days prior to the operation.
44. His view was that the most likely scenario was that there had been a massive saddle embolus (a central clot) which had been heralded, as was often the case and is a well-recognised presentation, by smaller pulmonary emboli at the times of hypotension earlier in the operation. The reason that the saddle embolus had been dislodged and so caused the arrest was the turning of the Claimant from prone to supine, which movement was sufficient to dislodge the clot. In his view cardiac arrest in these circumstances is almost pathognomonic for pulmonary embolisation. The rapid decrease in end-tidal CO<sub>2</sub> which was noted by Dr Leong immediately before the cardiac arrest is, he says, a common presentation of massive pulmonary embolism as the blood flow to the lungs is significantly compromised. The presenting rhythm was PEA, which is the typical rhythm for an arrest caused by pulmonary embolism.
45. Most importantly, he said, the CTPA scan demonstrated evidence of very significant pulmonary embolisation throughout much of the lung, sufficient in magnitude to cause cardiac arrest. Whilst there was not a single massive central clot shown on the CTPA he said that external cardiac massage often breaks up the clot and dissipates it throughout the lungs producing CTPA findings such as these. He maintained those views in cross-examination.
46. In cross-examination he accepted that it was relatively rare to survive a cardiac arrest caused by pulmonary embolism absent thrombolytic therapy (which the Claimant did not have) but said that some people do survive, he believes because the effect of cardiac massage is to disperse enough of the clot to allow survival. The Claimant was extremely fortunate to be in hospital, already ventilated, able to receive expert resuscitation immediately.
47. It was suggested to him that there being end-tidal CO<sub>2</sub> measurable throughout the arrest was inconsistent with a massive pulmonary embolism, and he said that for the first 10 to 20 seconds one might not expect it to be present but that thereafter the clot would be broken up quite quickly with cardiac massage.
48. Turning to Dr Power, his opinion, of course, is that hyperkalaemia caused the cardiac arrest. In his original report he pointed to the fact that post-arrest echocardiography did not demonstrate a right heart under significant strain and said that CTPA refuted a large central embolism obstructing the outflow of the right heart.

49. In the joint report he said little more save that he tended to the view that a pulmonary embolism sufficient to cause an on-table cardiac arrest would be likely to lead to a more extended period of haemodynamic instability and problematic gas exchange. He again said that the echocardiography was suggestive of normal right ventricular function. [I interject here to note that he did not deal either in his original report nor in the joint report with the dilation of the right ventricle noted on the bedside echocardiogram, nor with the TAPSE measurement on the transthoracic echocardiogram of 1.6 cm: in his original report he said that a TAPSE of 1.6 cm or less suggests impairment of the right ventricle, although neither he nor any of the other experts were cross-examined about this save for Dr Clifford, who said he did not know what a TAPSE was.]
50. In cross-examination Dr Power accepted most of Dr McCrerrick's points in favour of pulmonary embolism as being correct, including the supposition that there had been smaller pulmonary emboli at the times of hypotension earlier in the operation; that one could reasonably link the cardiac arrest to the turning of the Claimant; that a rapid decrease in end-tidal CO<sub>2</sub> was commonly seen with massive pulmonary embolism; and that it typically presented as a PEA arrest.
51. He disagreed with Dr McCrerrick's theory that a massive embolism could be broken down by external CPR. He described it as an attractive hypothesis but without a good evidence base. He accepted it was a plausible medical possibility but thought it "vanishingly unusual".
52. Turning to the cardiology evidence, Dr Norell like Dr McCrerrick thought that on the balance of probabilities the cardiac arrest was caused by pulmonary embolism. The postoperative CTPA showed bilateral pulmonary emboli. His opinion in the joint report was that there was a slight right ventricle abnormality on the echocardiogram and that CPR was sufficient to disperse the pulmonary embolism into both lungs, thereby relieving any strain on the right ventricle.
53. In cross-examination he said for the first time that he was struck by the marked deterioration in the Claimant's condition at or around 18:00 when there was a marked drop in blood pressure, he required 100% oxygen and his expired CO<sub>2</sub> was falling which suggested falling cardiac output. That was compatible with significant pulmonary vascular obstruction, whether an acute large pulmonary embolism or the culmination of previous multiple events. The Claimant was able to cope with that obstruction thanks to the treatment he was receiving, particularly in the form of fluids, but the turning of him from prone to supine was, he thought, the final straw. The cardiac arrest may have been due to the dislodging of a large saddle pulmonary embolus, or it may have been that the effect on movement on venous return to the right side of the heart was, in the Claimant's precarious position, the final straw which tipped him into cardiac arrest.

54. Either of those explanations was in his view plausible and both fitted with the CTPA which in his view showed sufficient evidence of clot affecting supply to multiple areas of the lung to equate to a massive pulmonary embolism. He accepted that there was no evidence of central pulmonary embolism on the CTPA but thought that the cardiac massage received by the Claimant was very effective and had dispersed the clot. He accepted that survival after central pulmonary embolism was unusual but did not think it affected the possibility of the Claimant in this case surviving.
55. Dr Norell accepted that the theory of a deterioration beginning at 18:00 was new and not contained in his report, but said that he had now in the course of the trial been struck by the anaesthetic records and the issue at 18:00.
56. Moving to Dr Clifford, he said in his original report that there were three potential causes of the cardiac arrest, being hyperkalaemia, hypovolaemia or pulmonary embolism. He said that it was “difficult to accurately assign the contribution of each of the potential causes of his cardiac arrest but it is my opinion that the relative contributions were hyperkalaemia (60%), hypovolaemia due to blood loss (25%) and segmental pulmonary embolism (15%).” In cross-examination he said that what he meant there was that hyperkalaemia was the predominant cause but the other factors contributed.
57. Rather like Dr Norell, he did not give any detailed reasons for his opinion in his original report. In the joint report he said that whilst the CTPA showed bilateral pulmonary emboli, there was no right heart strain on echo which would be expected with a pulmonary embolism sufficient to cause a cardiac arrest.
58. In cross-examination he accepted that he had nowhere in his reports referred to the bedside echocardiogram which referred to a dilated right ventricle, and said that was because his view was that ICU bedside echocardiograms are not as reliable as ones done on a proper machine by a proper sonographer. Measuring right ventricle dimension is hard even for a trained sonographer. The fact that there was normal function, he said, militated against there being a defect.
59. He said more than once in cross-examination that his understanding was that the Defendant had admitted that hyperkalaemia was likely to have caused the cardiac arrest, but then said that he had misspoken and that it was his own opinion that hyperkalaemia was the cause.
60. He accepted that there was evidence of pulmonary emboli on the CTPA scan in three areas of the lung. He accepted that in principle external massage could break up a clot but said he thought it would be very unlikely as it is difficult to break up a clot even when using an instrument internally to mechanically break it down.

61. He agreed that turning from prone to supine may have been a factor in relation to pulmonary embolism: anything that disrupts the veins, including the movement in the veins when turning, makes it more likely that there would be movement of a clot.

Analysis: the 19:34 potassium result and the cause of the cardiac arrest

62. Whilst various criticisms have been made of the experts on both sides in terms of their general expertise, their approach to giving their opinions, errors and inconsistencies in their reports, and so on, none of that assists me in this particular case in deciding whether the 19:34 result was spurious, which is what the whole issue of hyperkalaemia (and so the cause of the cardiac arrest) turns upon. This is an issue, in my judgment, which can and should properly be decided on an assessment of the medical literature and the agreed shared clinical experiences of the experts and what logically flows from that.
63. None of the experts, who have between them many many years of clinical experience, have seen anything approaching such a precipitous rise and then fall in serum potassium levels in such short periods of time in any type of planned surgery, including major surgery. Similarly nowhere in the literature are there any reports of changes of this magnitude in such periods, or anything approaching them.
64. No criticism was made of Dr Leong for waiting an hour to measure serum potassium levels after the result of 5.2. Dr McCrerrick was not challenged on his evidence that she was very conscientious in doing hourly ABGs, whereas he would usually take them two or three hourly in major surgery. If it were the case that in major surgery such as this, which involves significant bleeding and muscle damage, serum potassium levels could rise by this amount in an hour, so that they could rise from normal levels of around 5 to dangerous levels of 8 or higher in that time, then logically (as Dr McCrerrick said) it would surely be the case that anaesthetists in such operations would routinely be measuring those levels every 15 or 20 minutes.
65. It is, then, highly improbable that the 19:34 result was a true result. A rise of that magnitude in that time is not supported by expert clinical experience, by ordinary clinical practice, nor by any literature whatsoever.
66. Not only that, but there is a plausible explanation for the spurious result, in the form of haemolysis of the sample. Unlike such precipitous rises and falls in serum potassium, haemolysis giving rise to falsely high potassium readings is well known to the literature and accepted by the experts as something which can and does occur. The circumstances in which the 19:34 sample was taken were significantly different to the circumstances in which all of the other samples were taken, and so the fact that no other sample was haemolysed does not appear to be me to be a basis for discounting haemolysis or finding it unlikely. Although the sample was taken from an arterial line, Dr McCrerrick's suggestions that the drop in arterial pressure caused by the cardiac

arrest could have a potential effect upon the ease of obtaining the sample, and that the busy and stressful circumstances in which it was taken may also have had an effect, seem to me to be entirely plausible and to fit with the various mechanisms by which haemolysis can occur.

67. All of that evidence, in my judgment, leads inexorably to the conclusion that the Defendant has proved on the balance of probabilities that the 19:34 result was spurious.
68. If that is right then the experts agree that the cardiac arrest was caused by pulmonary embolism rather than hyperkalaemia.
69. As a cross check against the finding that the result was spurious I turn to consider the evidence in relation to the pulmonary embolism. If the evidence for pulmonary embolism as the cause of the cardiac arrest is very weak, or the evidence against it very strong, it might suggest that the finding of a spurious result is wrong and should be revisited. But in my view the evidence in relation to the pulmonary embolism is not such that it can possibly undermine what seems to me to be a very clear, indeed almost inevitable, finding as to the 19:34 result.
70. The Claimant was at risk of pulmonary embolism. The cardiac arrest coincided with the turning of him from prone to supine, which suggests a strong temporal connection between the arrest and the movement (which in turn fits with a pulmonary embolism being dislodged as the turning took place, or with the disruption in venous return being the tipping point after which the pulmonary obstruction could no longer be compensated for). The CTPA scan showed evidence of multiple pulmonary emboli in multiple areas of the lung. The experts all agreed that, at least in principle, external cardiac massage could break up a large clot, notwithstanding that they disagreed significantly as to the likelihood of that actually happening. Survival of cardiac arrest caused by pulmonary embolism without thrombolytic therapy is relatively rare but not unknown to medicine, and the Claimant's cardiac arrest occurred in theatre whilst he was already intubated and ventilated with an expert team around him. None of the experts suggested that normal right ventricular function (if normal it was, taking the Claimant's case at its highest and ignoring the bedside echocardiogram and the 1.6 TAPSE result on the later echocardiogram) could not possibly be present after a pulmonary embolism cardiac arrest where effective immediate CPR had been given, particularly if it was indeed the case that the cardiac massage dispersed the clot fairly quickly as the Defendant's experts posit.
71. It is, then, entirely plausible that the Claimant's cardiac arrest was due to pulmonary embolism which was subsequently dispersed by way of external cardiac massage, unlike the theory that his serum potassium rose and fell by such large amounts in the short periods as the 19:34 result would indicate, which is wholly implausible.

72. On the balance of probabilities, then, the 19:34 result was spurious and the cardiac arrest caused by pulmonary embolism rather than hyperkalaemia.

### **The remaining issues**

73. It follows from the finding that I have made as to the cause of the cardiac arrest that the rest of the issues between the parties fall away. I do not propose to address them in any detail but give a brief summary below of the findings I would have gone on to make had I found that the cardiac arrest was due to hyperkalaemia.
74. First, I would have found no breach of duty other than that which was admitted by the Defendant. The allegations of breach of duty were diffuse and essentially unparticularised. I would have rejected the suggestion, which was made for the first time in cross-examination, that Dr Leong should, at a time when the Claimant's condition was increasingly unstable, have sent her specialist registrar off on a hunt to attempt to track down the ODP, and I would have accepted her evidence that there was no one spare in theatre who could have been sent to do that.
75. Secondly, I would have found that had Dr Leong acted on the ABG results at 19:00 she would have administered calcium gluconate by 19:20 and on the balance of probabilities the arrest would have been avoided. That was the majority view of the experts (Drs Power, Clifford and Norell; Dr McCrerrick accepted it as "possible").
76. Thirdly, I would have found that on the balance of probabilities the PION had already occurred at the time of the cardiac arrest. PION is a recognised, albeit rare, complication of this type of surgery. Its usual incidence is 1 in 10,000 cases. There are a number of recognised risk factors for the development of PION, all of which applied to the Claimant so that his risk was agreed to be 1 in 1,000. Cardiac arrest is not a recognised risk factor for PION. PION as a complication of cardiac arrest is not recognised anywhere in the literature, nor in the experience of any of the experts in this case. I can see, and the various experts agree, that a cardiac arrest could theoretically cause this type of injury, but whilst there is ample evidence of cardiac arrest leading to hypoxic injuries affecting all sorts of neurological function, there is no evidence of it causing this type of injury to the optic nerve. That in itself in my judgment makes it more likely than not that the PION occurred as a result of the surgery rather than the cardiac arrest. The other important piece of evidence on this issue is the significant facial swelling experienced by the Claimant after the surgery, which is indicative of raised introrbital pressure. Raised intraorbital pressure is not caused by cardiac arrest. It is a risk factor for PION. The fact that it would appear that the Claimant suffered from a prolonged period of raised intraorbital pressure such as to give rise to this extreme swelling adds to the likelihood that it was the surgery which caused the PION, and that it was caused at some point during this lengthy surgery, but prior to the cardiac arrest which took place at the very end.

77. As to the last issue, whether the PION which had already been caused was exacerbated by the cardiac arrest, the difficulty for the Claimant is that the evidence on this was speculative. It amounted to an acceptance that a period of “downtime” due to cardiac arrest could have resulted in a period of reduced perfusion of the optic nerve which could have contributed to the severity of the PION. The ophthalmologists defer to the anaesthetists as to the extent to which the tissues of the optic nerve would have been oxygenated, but the anaesthetists did not give any meaningful evidence on the point. Whilst there was some evidence in particular from Dr Elston that the fact that vision is worse in one eye than the other might suggest an additional factor at a late stage of the progression of the injury, the evidence was not such as would have enabled me to make a finding that but for the cardiac arrest the PION would on the balance of probabilities have been less severe nor to find that it had made a material contribution to the severity of the PION.

### **Miscellaneous matters**

78. Allegations of bias were made against Dr McCrerrick in the course of cross-examination. As put to him by Mr Wells they were allegations of deliberately reading the CTPA scan in a way intended to be advantageous to the Defendant, and then in changing his opinion as to the manner in which the spurious potassium result could have come about. Mr Wells somewhat rowed back from that in his closing submissions but still suggested unconscious bias.

79. For the avoidance of doubt, I reject any suggestion that Dr McCrerrick demonstrated bias, whether deliberate or unconscious. His reading of the CPTA scan in fact accorded with Dr Clifford’s reading of the scan so could hardly be properly criticised. His change of opinion as to the potassium levels was as a result of him engaging in the proper process of discussion and consideration of Dr Power’s opinion, and then reflecting upon what then could give rise to the results which in the event I have found to be spurious.

### **Conclusion**

80. It follows from my finding as to the cause of the cardiac arrest that the claim must be dismissed.

81. It is inevitable that the trial and the judgment focus on the expert evidence rather than on the Claimant personally, so it is important for me to say that at the heart of this case, of course, is the Claimant, who has suffered significant life-changing consequences of this surgery. He was present throughout the trial. No one could fail to be impressed by his dignity and by the efforts he has made to make the best of his situation, including the great achievement of graduating from university in 2023. I wish him and his family all the best for the future.