IN THE HIGH COURT OF JUSTICE
KING'S BENCH DIVISION

Case No. QB-2017-003065

Courtroom No. 15

Royal Courts of Justice
Strand
London
WC2A 2LL

Monday, 12th December 2025

Before:
HIS HONOUR JUDGE GLEN
(Sitting as a Judge of the High Court)

B E T W E E N:

PAUL

and

THE ROYAL WOLVERHAMPTON NHS TRUST

MS A STAMP appeared on behalf of the Claimant
MS H WOLSTENHOLME appeared on behalf of the Defendant

JUDGMENT
(Approved)

HHJ GLEN:

Introduction

1. This is my judgment in a claim for damages arising out of the death of Parminder Singh Paul on 26 January 2014. The Claimant, Balbir Kaur Paul alleges that his death at that time was the result of negligence on the part of the treating clinicians at the New Cross Hospital in Wolverhampton employed by the Defendant in the period of 9-12 November 2012. The Claimant is represented by Ms Stamp of counsel and the Defendant by Ms Wolstenholme also of counsel.

2. For the purposes of determining this matter, I have heard evidence from the Claimant and from her sister-in-law, Mrs Avtar Paul. For the Defendant I have heard evidence from Dr Velu, Dr Khogali, Dr Arya and Dr Horton, the first three of those being the treating clinicians at the relevant time. That evidence took the form of witness statements and in addition they gave evidence and were cross-examined. I have also heard expert evidence, cardiology evidence from Dr McCanse and Dr Cripps and from nephrology from Dr Frankel and Dr Andrews. All of those experts produced reports and agreed to a greater or lesser extent the form of a joint statement of their evidence. In addition it is right to record that Dr Frankel produced a couple of supplementary letters. Again, those experts gave live evidence and were cross-examined. Finally, and in addition to all of that, I have been referred to a variety of documents including importantly the clinical notes made by the hospital at the time and certain papers and guidance emanating from a variety of sources to which I shall come.

3. I take all of that material into account in reaching my decision as I do, of course, the written and oral submissions made by counsel. Albeit on a deferred basis, I give this judgment orally and therefore to some extent *ex tempore.* I will focus on the issues that are relevant to the conclusions that I have reached but I wish to make it clear that I have considered all of the issues very carefully. If at the end of this judgment either party believe that there is an important issue which I have omitted to deal with, I will deal with that at the end. Likewise, if I make mistakes over dates, times, names, places things of that kind, I am happy to correct those at the end.

Background

4. Mr Paul, known to his wife as Gorge, was by all accounts a good man. He worked as a pharmacist but sacrificed a good part of his career to support his wife in her training to be a solicitor and indeed in that subsequent career. He helped to look after their three children, taking them to and collecting them from school. He was also very handy around the house.

5. As well as this immediate family, Mr Paul was part of a much wider one. He had four siblings as I understand it (the exact number varying in the evidence) including a brother known as Lakhbinder who was married to Mrs Avtar Paul. Sadly, in common genetically with most of his family including his late mother, Mr Paul also suffered from an unusual degree of poor health. In 1994 when still only 29, he was diagnosed with Type 2 diabetes and he struggled to control that diabetes, both in the medical sense and at times in his personal commitment to treatment and diet. It is right to record that there were other times when he clearly did make efforts to control his weight and to exercise. Diabetes brought with it numerous unpleasant complications, high blood pressure, retinopathy including the need for laser treatment, neuropathy and problems with his feet.

6. By the end of 2012 Mr Paul had developed stage 4 chronic kidney disease ('CKD'), the final stage before renal failure. In addition, as if that was not enough, in August 2010 he had a transient ischaemic attack. In paragraph eight of her witness statement the Claimant says this about her late husband:

> *"Gorge was a very proud and private man, and he did not like to talk to me in too much detail about his health. He would never want me to worry about things and would always tell me he was fine. I think this was his way of protecting me."*

7. On 9 November 2012, Mr Paul went to New Cross Hospital for a renal outpatient appointment where, as I understand it, he was going to have an iron infusion to treat his anaemia. Whilst there, he reported central dull chest pain when walking across the car park radiating into his left arm and settling on rest. That triggered, as it quite rightly should have done, a referral to the emergency department where a history was taken and that can be found at page 57 of the bundle. The record says; *"Complaint of central and left side of chest pain for the last two weeks, happens at night, waking, patient prior to this no history of chest pain."*

8. He was next assessed by a cardiac nurse, Nurse Shelly, at 15:45 on that day. Her assessment appears over a number of pages but in essence she records *"dull ache left side of chest and drawn to central chest, left arm, both jaws, precipitating factors walking and lying flat, no relief from Gaviscon/proton pump inhibitor."* She records the pain as being somewhere between one and two on a scale where zero is no pain and 10 is unbearable pain, together with associated shortness of breath and feeling tired.

9. At page 60 of the bundle she notes the various risk factors for coronary heart disease. All of them are ticked, other than smoking. That includes the death of his mother, from as I understand it, a myocardial infarction. Troponin levels were taken and revealed a level of 107. Her impression was this;

> *"Multiple risk factors for heart disease. Two-week history of occasional chest pains usually at night. Worst episode 7 November, two days before today. Walking into hospital appointment this morning precipitated cardiac sounding chest pain relieved by rest. Raised troponin although in the presence of renal failure treat as ACS."*

ACS (Acute Coronary Syndrome) is a spectrum of conditions including amongst others an NSTEMI (a non-ST segment elevation of myocardial infarction). To use a more prosaic term, this is a minor heart attack.

10. Further discussion in her notes starting at page 1677 of the bundle again includes the two-week history of chest pains usually occurring at night and described as a mild, giving the location sometimes radiating to both jaws. This description is given,

> *"Walked across the car park this morning to attend hospital appointment when developed pain as above, pain radiating to teeth, pain settled once he slowed down and rested. Currently pain free. Impression/Plan – cardiac sounding chest pains occurring on minimal exertion and rest in a patient with multiple risk factors, troponin is significantly raised although renal function may have exacerbated it.*

The clinical context suggests ACS – transfer to cardiology ward for consideration of in-patient angiography."

11. A second troponin test taken about 4½ hours later demonstrated a troponin level of 111. At 13:15 on that day he had a further episode of chest pain documented at pages 60 and 64 of the bundle. The complaint was of central chest pain non-radiating and an ECG was repeated. This was the first, as I understand it, in a series of seven ECGs performed during the course of Mr Paul's stay in the hospital. They have been variously described in the evidence as normal and as borderline abnormal (which means with mild or less than 1mm ST depressions) but in any event they were regarded at the time as unremarkable. It is common ground to all of the experts that the ECGs demonstrated no change.

12. At about 16:40 on 9 November, Mr Paul began to be treated with appropriate medication for someone with ACS. At 01:00 on 10 November Mr Paul was transferred to the cardiology ward where he was given a diagnosis of ACS with a line put through it and NSTEMI written next to it. Prior to this he was noted to have had no further chest pain and had none on admission but at about midday on 10 November, there was a further complaint of pain. A further ECG was undertaken and no changes were shown. He was seen by Mr Velu and his notes are at pages 60, 61 and 62. He was the cardiology registrar at that time and is now a consultant.

13. He noted the presence of a number of factors including CKD, anaemia, previous TIA and so on. He then writes this,

> *"One episode of rest chest pain, awaken from sleep on Wednesday, pain in the chest, jaw burning, says burning pain since being on ferrous sulphate. No clear exertional symptoms and the plan is to mobilise and repeat the ECG. If symptomatic and/or new ECG changes, then for CATH. If not, then stress assessment will be helpful..."* [That is a reference to a dobutamine stress echo cardiogram or a DSE] *"...this patient not keen on CATH if it will affect kidney function unless it is necessary."*

The existence of any further clinical signs in the form of pain is somewhat controversial. We have a note at page 1680 of the bundle dated 11 November and timed at 05:20: *"Patient complained of pain on the left side of the chest upon rest. Observations recorded and were stable."* It is apparent that this can only have been a reference to chest pain on the previous evening, the Saturday.

14. There are various other notes made, medication is given as prescribed and then there is another note which appears to be in a different pen and potentially in a different hand*: "Patient complaining of chest pain on rest, but pain explained as indigestion type pain. Patient stated pain since settled without any treatment."* That is not dated or timed. There is a question mark over whether that is effectively an explanation or expansion on the previous note or whether that is a report of fresh pain. Certainly, there is nothing to tie into that suggests the latter.

15. Mr Paul was next seen by Dr Sandhu, the only treating clinician who has not given evidence before me. The note timed at midday is: *"No complaint of any pain or discomfort reported."* Further down it says this: *"Seen by Dr Sandhu on ward round, chest tightness at night, a bit*

of shortness of breath on exertion, plan: needs to have a cardiac investigation DSE. Will discuss with Dr Khogali, query angio." There are also notes at page 1665 which record essentially the same thing but do add this: *"Last chest pain at 8pm 10 November at 20:15..."* [That appears to be the reference to the previous evening's chest pain] *"...Troponin 100, ECG. Plan - needs further investigation including DSE that will confirm with Dr Khogali. Patient concerned about contrast angiography."*

16. The next treating clinician was Dr Khogali. His notes are on page 1666 of the bundle. Having noted various points, he writes this*: "Plan - echo today. If echo* [echocardiograph] *shows impaired left ventricle function, then to angio* [angiography]. *If echo is normal, then DSE as inpatient or otherwise outpatient."* The results of the echo were satisfactory; there were no signs and no further instances of chest pain. At 14:20 on 12 November he was seen by Dr Arya and later discharged that same day. On 16 November Mr Paul did a treadmill test and in the course of that reported some jaw pain, but no chest pain and there were no ECG changes. A DSE test was normal.

17. After these events, Mr Paul's renal function continued to decline. In June 2013 after some missed appointments, he commenced on peritoneal dialysis but that was not successful. In August 2013, he commenced on haemodialysis three times per week, which with travelling took a full day and left him very tired afterwards. In September 2013, the possibility of a transplant began to be discussed, albeit with no reference to any putative donor. It is however the Claimant's case that the possibility of the Claimant or Lakhbinder being a live donor was discussed and Mr Paul was keen on that possibility.

18. The view taken was that Mr Paul ought to have an angiography before being considered or confirmed for a transplant and that was why he saw Dr Horton. Arrangements were made for him to have an angiograph but sadly before it could be done, he died whilst out shopping with his daughters, a day before (according to the Claimant) he was due to return to work. This must have been truly shocking and upsetting for his daughters. A post-mortem in January 2014 showed that Mr Paul was at the time suffering from severe three vessel coronary artery disease.

The Issues

19. The claim form in this case was issued on 25 October 2017 and originally the Claimant was joined as a claimant with her two daughters who had witnessed Mr Paul's death. That element of the claim was struck out and an appeal all the way to the Supreme Court was unsuccessful, hence the significant delay in this matter coming on for trial. The core of the Claimant's allegations are set out in the particulars of claim and I propose to identify them because they are important. They are; (a) failing to heed that the deceased had multiple risk factors for the development of coronary artery disease; (b) failing to heed that the deceased's presentation was typical for coronary artery disease, including typical presentation on exertion of chest pain, abnormal troponins, borderline ECG and highly adverse risk factors; (c) placed undue weight on the fact that troponin levels can be unreliable in the context of CKD and insufficient weight on the totality of the factors set out in (a) and (b); and (d) failing in the light of the above to arrange coronary angiography.

20. The Claimant says that if an angiogram had been undertaken, Mr Paul would have been referred for coronary artery bypass surgery or CABG as it has been referred in evidence. That

would have resulted in an extension to his life expectancy. In addition, he would have been assessed as being fit for a transplant and he would have had one. It is acknowledged that he probably would not have survived until transplant had it been on the basis of a cadaverous donor, not least because Mr Paul belonged to a very rare blood group. It is however the Claimant's case that his wife or Lakhbinder would have offered their kidneys and would have been judged suitable, albeit that that would only actually have happened through what is known as the 'altruistic donor swap scheme'. This is because his wife also has a relatively rare blood group.

21. If he had had a transplant, it is contended that he would have had an extended life expectancy. He would have returned to full time work. He would have resumed all of his childcare and DIY activities. The claim reflects the losses flowing from his having never done any of those things as well as of course the usual statutory damages and damages for pain, suffering and loss of amenity. There is also a claim for aspects for funeral expenses and one aspect of those, the cost of flying Mr Paul's ashes out to India for disposal in a sacred river, is controversial.

22. The Defendant denies there was any breach of the duty of care. It denies that Mr Paul would have been revascularised, denies that he would have achieved a transplant of his kidney, denies that he would ever have returned to work or returned to providing his family service in the way that he had. There are some relatively inconsequential differences on the measure of general damages for pain, suffering and loss of amenity and as I have said there is a small, discrete but likely dispute on the question of funeral expenses.

The Evidence

23. I now summarise the evidence that I heard. Inevitably it is only a summary. I do not propose to summarise the witness statements in any depth; they are there to be read. I will summarise the evidence in its logical rather than the chronological order that I received it.

24. The Claimant in her witness statement gives her perception of the history of the matter. In paragraph 19 she describes how Mr Paul was keen on the idea of her donating a kidney for transplant, that she would have been willing to do so and that the possibility of Lakhbinder providing a kidney had also been discussed. She acknowledged that Lakhbinder was diagnosed with diabetes a couple of years later. I have no evidence from Lakhbinder. She also in her statement gives evidence about Mr Paul's employment history and the fact that at the time of his death he had a full-time contract with Boots the Chemist, including of course a pension plan.

25. In cross-examination she acknowledged that she was not fully aware of Mr Paul's medical history at the time. She said that after the TIA he had been a bit unsteady and wobbly, but it did not affect his ability to do his activities, although they had diminished towards the end. She described him as having been very unwell before dialysis had commenced, but then he had improved. She was unwilling to acknowledge that Mr Paul may have failed to attend medical appointments and/or failed to take, or been resistant to taking, medical treatment. She did however in the end acknowledge that his engagement with statins may have been 'on and off'. She told me that Mr Paul always made informed decisions regarding his own care and gave an example of him having been unhappy with his eye treatment at the hospital. She was adamant that despite his various medical issues, he was preparing to return to work the day after his death, albeit at a different location because of his mobility issues. She accepted that

she had not attended any of his renal outpatient appointments and that she had not taken any steps to see if she was suitable as a donor. She was waiting to see if it was a viable proposition at all. There had been a family discussion about that possibility.

26. I then heard evidence from Mrs Avtar Paul, the wife of Lakhbinder. Most of her witness statement was taken up dealing with the impact of Mr Paul's death on the children. However, in her witness statement she confirms that Mr Paul made great contributions around the house. She also confirmed there was a family discussion around the possibility of a live donation. In cross-examination, she confirmed there had been a conversation about Mr Paul returning to work the next day and she remembered it because she had told him not to cheat on her as they were both on Weightwatchers at the time. She acknowledged that Lakhbinder had never been tested for suitability as a donor.

27. I Turn to the Defendant's evidence. In chronological order of treatment but not chronological order of hearing I heard from Dr Velu who as I have said was a registrar at the time but is now a consultant cardiologist. His witness statement is simply an expansion upon the notes and inferences to be drawn from the notes. Neither he nor any of the treating clinicians profess to have any personal recollection of Mr Paul or his case. The essence of Mr Velu's witness statement is that any treatment was a matter which had to be considered by a multi-disciplinary team.

28. In cross-examination he was asked about Nurse Shelley's notes. He told me that the purpose of the triage was to see if there was any reasonable suspicion of cardiac symptoms requiring further consultant review. He agreed that Mr Paul had a lot of risk factors. He acknowledged that an ECG with a less than 1mm ST depression did show what could properly be described as a borderline abnormality but said that the significance of that depended on other factors. Mr Paul's Troponin was above normal and might in other circumstances have suggested an NSTEMI, but it had to be seen in the context of Mr Paul's CKD. He said that the rise and/or decline of troponin levels after an NSTEMI is very variable and it is not possible to say that any particular trajectory would occur in every patient. Troponin, he said, was not the end of the diagnostic process. It was just one factor.

29. He was clear that they had not ruled out ACS at that stage and indeed Mr Paul had been treated on the basis of ACS. He agreed that the description of the chest pain when he was walking across the car park was exertional, but he said his note was not intended to suggest that there were no exertional symptoms at all. His note recorded an independent review of the patient, and he had reviewed the notes in order to enable a recommendation to the consultant. He did not accept that his note was in any way deficient. He agreed with the proposition that he needed to be professionally curious. He told me that the troponin level as recorded in the note had been rounded down and that he would have seen the actual figures on the computer. He acknowledged that there was a further episode of pain which mandated a further ECG but the significance of this depends on the whole picture and of course the cause of the pain. He agreed that recurring episodes of chest pain meant increased risk. He said troponin levels would have been persuasive if they had been dynamic although, of course, they were still relevant. The decision to discharge Mr Paul was a team one. He considered the risks of angiography to be at least moderate.

30. Next, I heard from Mr Khogali who was then and is now an interventional consultant cardiologist then at Wolverhampton and now at the Royal Brompton. One again, his evidence

was based on his contemporaneous notes and is summarised in his witness statement in this way, at paragraph 24:

> *"In the light of these findings and in the absence of any other factors such as ongoing chest pain, rising troponin level or regional wall motion abnormality there was no need to proceed to a coronary angiogram. Mr Paul was a patient with chronic renal failure. There was no secondary rise in Mr Paul's troponin levels, no acute changes on the repeat ECGs and the echo cardiogram demonstrated good left ventricular function. Mr Paul's pain had subsided. These factors were a contra-indication for intervention and to recommend a coronary angiogram in the presence of the same would have been contrary to practice at the time."*

31. Further on at paragraph 34 he says this:

> *"Undergoing a coronary angiogram would not automatically lead to immediate revascularisation. It would be open to the interventional cardiologist performing the coronary angiogram to exercise their clinical judgment during the procedure…."* [I am paraphrasing very slightly] *"…Such a judgment being informed by the clinical risks, findings and risks of the patient it would be standard practice to discuss the possibility with a multi-disciplinary team. Had I discovered diffused moderate triple vessel disease, my standard practice would be to discuss the case at NDT."*

32. In cross-examination he described the clinical notes as a continuum. There was not necessarily a need to repeat all that had gone before and focus was on clinical change and progress. He said that the way histories are taken can in any event vary quite widely, as can the reporting by the patient and the interpretation of it. However, he did acknowledge that history taking was something of paramount importance.

33. He acknowledged that a working diagnosis of ACS required investigation and that some features of Mr Paul's chest pain were cardio in character. He agreed some exertional symptoms had been documented although anaemia therapy could have contributed to those symptoms. However, whilst pain is an important factor it was only one and did not assist in prognosis. A concern would only exist if pain was accompanied by ECG changes. Essentially, one needs two out of three of; recurrent cardiac pain, ECG changes and troponin changes.

34. He pointed out that the New Cross Hospital used high sensitivity troponin assays and therefore the intervals between the tests was correct. He said the raised level of troponin was not a concern on its own given Mr Paul's renal failure. His evidence was that a typical troponin level for a person with stage 3-5 CKD was around 100-129 and he said that there was data to support that. He said it was impossible to say whether the troponin level was also or may have been consistent with an NSTEMI on 7 November. He described the pain in this case as only off and on. He described the ECG as showing a borderline non-specific abnormality but that could be due to changes in electrolytes brought on by his renal failure. Here the patient settled without any unusual intervention.

35. He told me that he enjoyed doing angiography and he did them and CABGs on a day in day out basis. He would have been the first person to take that course, but it was not appropriate here given the risk and Mr Paul's wishes. He said it would have been very easy to do it, but it was not right. He accepted that persons with diabetes are at high risk of coronary artery disease, but he did not accept that the post-mortem was evidence of significant levels of such disease in November 2012. It was a disease which had clearly progressed. He was not willing to accept that the DSE had produced a false negative. It is all a question, he told me, of risk and benefit balance. He agreed that the risk could be reduced to some extent by reducing the volume of the contrast used during the angiogram, but it was difficult to predict exactly how much contrast would actually be needed.

36. He told me that an ECG measured heart function and heart abnormalities. It was good at telling you if an event had occurred but not so good at telling you how large the event was or when it happened. On that basis, it was unlikely in this case that any significant event had occurred. He was asked about the GRACE Score. He accepted that it was difficult to interpret and not commonly used. Management of patients is based on domestic and international guidance. He accepted that he had used (and appropriately so) the top and bottom boxes of the ESC stratification of risk.

37. Lastly, I heard from Dr Arya. Her witness statement again makes it clear that she has no direct recollection of this case. She was the discharging consultant then and remains a consultant now. She said that what she would have done was to have reviewed the whole clinical picture in order to assess the risk. I turn to her witness statement at page 204, paragraphs 22-23, where she says:

> *"Given his risk factors and certain characteristics noted during his admission, I considered his investigations did not suggest that he was at high risk of a myocardial infarction at that time. I based this assessment on the relatively stable, albeit elevated, troponin level, lack of significant ECG changes suggesting ischaemia in formulating my management plan as well as considering the ECG and troponin levels. I also considered the fact that anaemia can exacerbate cardiac symptoms. In the context of a patient whose pain had resolved where it was considered his troponin had not risen and where there were not significant ECG abnormalities but where there was a possibility of him having cardiac symptoms or cardiac risk, I considered it appropriate to prescribe cardio protective medication and organised an early stress investigation, namely a DSE.*
>
> *If the patient is reluctant to proceed to a coronary angiogram in different circumstances which are not present here, if there were significant ST segment changes on an ECG typical history, persistent or recurrent pain, raised troponin that could not be explained by other causes such as renal failure as in this case, it was and remains my standard practice to recommend a coronary angiogram and attempt to persuade the patient that they should undertake this."*

At page 207, paragraph 29, she said: *"Mr Paul was discharged with a working diagnosis of chest pain, suspected stable angina."* In her evidence in chief, she corrected that to read: "ACS."

38. In cross-examination she told me that you cannot diagnose ACS on troponin levels alone, particularly given renal failure. Their relevance has to be considered in the context of other factors, including ECGs and the patient profile. In terms of changes in troponin levels, you are looking for a 20% change or more. She said that potentially she considered that Mr Paul's troponin level to be his baseline. Mr Paul was treated as a high-risk patient with some cardiac symptoms, but he was being assessed to see how stable he was and how best to manage him. He would be at high risk if there were further episodes and ECG changes but given that he was settled, there were no changes and troponin levels remained static, conservative treatment was mandated. She was unable to explain why she had referred to stable angina in her witness statement, given that it was always clear that Mr Paul had been treated on the basis that he had ACS.

39. Like the other witnesses, she declined to offer an opinion on the characteristics and trajectory of troponin levels following an NSTEMI. She said some pain that he had reported was typical of cardiac pain, but that Mr Paul's reports varied, and the causes might be different. The history overall was not typical. Symptoms were suspicious but no more than that, although she acknowledged that the threshold was lower on a cardiac ward than it may otherwise be. She denied that she had wrongly categorised the risk and I made this note of her evidence:

> *"I do not accept that. There was chest discomfort in the early stages on admission only, hydrodynamically stable, ECT was normal, troponin was difficult to interpret, slightly elevated but unchanging. He had co-morbidity of chronic kidney disease at grade 4. I therefore agreed with the conservative approach and his pain settled after the haemoglobin provided."*

She acknowledged that the GRACE Score was mandated for use at the time, and she said there had been no guidance to say that it needed to be adjusted. Asked various questions in re-examination, I noted that she described Dr Khogali as an interventionist on the subject of angiography.

40. I turn now to, before I come to the expert evidence, to the medical literature. I was referred to a variety of papers but two in particular were the focus of attention. I will deal with the others briefly when I come on to the expert evidence. The first important piece of guidance that the Court needs to look at is of course the NICE Guidelines published for use in the United Kingdom. This is the 18 November 2020 version. No one has been able to find the version then in force, but any changes are identified by dates. They begin with this important heading:

> *"Your Responsibility. The recommendations in this guidance represent the view of NICE, arrived at after careful consideration of the evidence available. When exercising their judgment, professionals and practitioners are expected to take this guideline fully into account alongside the individual needs, preferences and values of their patients or the people using their service. It is not mandatory to apply the recommendations, and the guideline does not override the responsibility to make decisions appropriate to the circumstances of the individual in consultation with them and their families."*

41. Also relevant in the NICE Guidelines is this:

> *"As soon as a diagnosis of unstable angina or NSTEMI is made, formally assess individual risk of future adverse cardiovascular events using an established risk scoring system that predicts six-month mortality, for example GRACE."*
> [Global Registry of Acute Cardiac Events]

At page 157 these are the steps recommended:

> *"Take a full clinical history including age, previous myocardial infarction including whether there has been a previous MI or PCI or CABG, a physical examination including measurement of blood pressure and heart rate, resting ECG looking particularly for dynamic or unstable patterns that indicate myocardial ischaemia and blood tests such as troponin, creatinine, glucose and haemoglobin."*

That last one is from the 2010 edition and so was in force at the time.

42. The lowest risk is predicted rate of 1.5% or below followed by low at 1.5% to 3%. It says at 1.2.12:

> *"Offer immediate coronary angiography to people with unstable angina or NSTEMI, if their clinical condition is unstable. Consider coronary angiography with follow up PCI if indicated within 72 hours of first admission for people with unstable angina or NSTEMI who have an intermediate or high risk of adverse cardiovascular events according to the GRACE score, and no contraindications to angiography such as active bleeding or comorbidity."*

At 1.2.1:

> *"Be aware that some younger people with no risk causes of mortality within six months may still be at high risk of adverse cardiovascular events and may benefit from angiography."*

Finally:

> *"Where PCI is not indicated consider conservative management without early coronary angiography, people with unstable angina or NSTEMI who have a low risk of adverse cardiovascular events of 3% or less."*

43. The other key document has been the ESC Guidelines published by the European Society of Cardiology in 2011, so at about the time of events with which I am concerned. Relevant parts of that document begin at 697. This is the table that I have already referred to when dealing with Dr Khogali's evidence. The top box in the recommendations table on that page says:

> *"In patients with suspected NSTACS diagnosis and short term ischaemia bleeding, risk stratification should be based on a combination of clinical history, symptoms, physical findings, ECG, repeated or continuous ST monitoring and biomarkers."*

The bottom box says:

> *"In patients without recurrence of pain normal ECG findings, negative troponin test and a low risk score a non-invasive stress test for irreducible ischaemia is recommended before deciding on an invasive strategy."*

I anticipate that the reference there to negative troponin test is not that there is no troponin but that there is no change, although this is a matter the experts seem undecided about.

44. At page 692, we have a table of possible non-acute coronary symptom causes of troponin elevation. The first of those is chronic or acute renal disfunction. At page 715 the guidance goes on in this way:

> *"Recommendation: an invasive strategy within 72 hours after the first presentation is indicated in patients with at least one high risk criterion, see table nine, and recurrent symptoms."*

Table nine states that; *"The primary symptoms are a relevant rise or fall in troponin or a dynamic ST or T wave change."* Neither of those are suggested to be present in this case but the secondary criteria are these: *"Diabetes and mellitus…"* which was present, *"…renal insufficiency…"* which was present, *"…reduced left ventricle function, early post-infarction angina, recent PCI, prior CABG, intermediate to high GRACE score."*

45. In terms of the guidance insofar as it relates to patients with diabetes, the relevant extracts are at page 719:

> *"Patients with chronic kidney disease more frequently present with heart failure and without typical chest pain. Patients with NSTACS and CKD often do not receive guideline recommended therapy. CKD is associated with a very adverse prognosis and is an independent predictor of short to long-term mortality and of major bleeding in patients with NSTACS. Despite the fact that patients with NSTACS and CKD are frequently underrepresented in clinical trials there is no particular reason not to treat these patients just like patients devoid of renal disfunction."*
>
> *"Data on the impact of an invasive strategy on clinical end points in patients with NSTACS and CKD are not available as many trials of revascularisation excluded patients with CKD. In large registry as well as in sub-studies of trials given setting of NSTACS the outcome of CKD patients should improve with basic management, not only at end stage renal failure but also at the stage of moderate CKD. In observational studies, early invasive therapies associated with a better than one year survival in patients with mild to moderate renal insufficiency but the benefit decreases with worse renal function and is uncertain in those with renal failure or on dialysis. Patients with CKD are at risk of contrast induced nephropathy. This risk is increased in patients with older age and diabetes. In the case of urgent angiography, the risk of contrast induced nephropathy must be balanced against the ischaemic risk. Hydration before and following angiography and angioplasty is*

a strategy which is said to have the greatest impact on reducing the risk of nephropathy."

It goes on about the amount of contrast that might be used.

46. I also wish to read a section of page 727 which is under the heading "Management Strategy."

> *"Step 2. Diagnosis, validation and risk assessment. After the patient is assigned to the NSTACS IV oral anti-thrombotic treatments will be started according to table 13 and further management of the patient will be based on the following; responsiveness to anti-angina treatment, routine clinical chemistry, particularly troponins on presentation and after 6-9 hours that is without the highly sensitive troponin available. A fast-track rule out protocol three hours may be implemented…"* [as happened here] *"…Repeat or continuous XT segment monitoring where available assuming risks scores, assessment GRACE score and an echocardiogram."*

All of those steps were those taken by the Defendant in this case.

47. I finally wish to note this, at page 729, under the heading "Invasive Strategies":

> *"Patients with less acute risks according to table nine and without the recurrence of symptoms, angiography may be performed within a time window of 72 hours, thus such patients should undergo elected invasive evaluation at the first opportunity depending on the local circumstances."*

I will deal in some more detail with some of the other papers in the course of considering the expert evidence, to which I now turn.

48. The first expert to give evidence was Dr McCance, the Claimant's cardiology expert. The crux of his opinion can be found at page 222 of the bundle at paragraph 5.2 of his report:

> *"In my opinion there is a breach of duty by New Cross Hospital in not arranging coronary angiography during his emergency admission in November 2012. Although troponin levels are unreliable in the setting of CKD, his typical presentation of abnormal troponins, borderline ECG and highly adverse risk factor profile should, in my opinion, have prompted inpatient coronary angiography which would have been performed on a 'quaere proceed to PCI' basis. In these circumstances no competent body of cardiologists would have discharged him without a planned coronary angiography. This should ideally have been as an inpatient but given there was no clear rise in his troponin levels, early outpatient angiography within four weeks would have been acceptable had there been some delay in obtaining inpatient treatment."*

49. At paragraph 5.3 of his report, he is critical of the timing of the troponin tests, describing them as being too close together and expressing the view that further troponin tests should have been taken over the following 24-48 hours. He was critical of the failure to proceed to angiography after the exercise test, although in fact that is not alleged as an act of negligence

in the particulars of claim. He considered that the use of a DSE test was appropriate. In Dr McCance's opinion had the angiography been performed, it would have revealed severe three vessel coronary artery disease and Mr Paul would have been offered a CABG. He does not consider that the angiography would have precipitated dialysis and the risks of that could have been managed, although he acknowledges that CABG itself would have had that consequence. The outlook for Mr Paul would have been improved, although in his report he gives no figures for that.

50. Responding to the defence, he says that the history of pain on its own was enough for an angiograph to be performed, given the risk factors. As far as the troponin is concerned, he acknowledged that it is difficult to assess a patient with renal failure, but the levels were consistent with an NSTEMI that had taken place on the previous Wednesday. In respect of the patient's wishes, his views were these:

> *"This should not be a significant factor. The job of his cardiologist was to explain relative risks of an invasive versus a conservative approach. Given the guidelines, in my opinion the vast majority of intervention cardiologists would favour an invasive approach here, explaining to the patient the risks including those to his kidneys were outweighed by the benefits. In other words, no reasonable and responsible body of intervention cardiologists would not have recommended coronary angiography after due discussion of the risks."*

At paragraph 8.1, Dr McCance acknowledges that the NICE Guidelines are as he puts it "the most appropriate to use" and he also acknowledges the GRACE score by itself "did not mandate intervention" but he considers it needs to be adjusted to reflect Mr Paul's relative youth.

51. In his report, Dr Cripps took a different view. The core of his opinion is that given that Mr Paul had settled on medication, the lack of any significant rise in troponin, the absence of any dynamic ST changes and taking into account Mr Paul's CKD and the potential consequences of angiography (as well as of course Mr Paul's wishes), that it was reasonable to adopt a conservative approach and proceed to a DSE test which, as it happens, validated that approach. There was no failure to appreciate the risks, no failure to diagnose the possibility of ACS and indeed Mr Paul was treated on that basis. None of that by itself mandated performing angiography. He relies on various extracts from NICE and from the ESC Guidelines. In his opinion, even if the angiography had been performed, it would have only shown diffuse three vessel disease and that would have had to have been something considered by the multi-disciplinary team. The probability is that he would not have been offered for revascularisation given that CABG would almost certainly put him into renal failure. He described the survival rates of patients like Mr Paul with CABG as being "disappointing" with about a 50% four-year survival rate. If CABG had been performed, his view was that Mr Paul might have lived for a further four years with increasing levels of disability.

52. In their joint statement, the experts maintained their positions and there was not an enormous amount of agreement. There was agreement that the pain reported by Mr Paul on presentation was, or appeared to have been, cardiac in nature and that he was properly considered and treated as having ACS on admission. However, they disagreed on the significance of troponin, except that they did agree that there had been no material rise in troponin levels. They disagreed on the interpretation of the ECG. They disagreed on the significance of recurrence

of pain. However, at section four they did agree that a responsible body of clinicians would regard raised troponin in a patient with renal failure to be of uncertain significance.

53. In answer to question 10, they agreed that there was no randomised data to guide the treatment of patients with end stage renal failure and they reviewed literature, including the American Heart Association Guidelines. They agreed vAryations for individual patients was appropriate. At page 775 of the bundle, they agreed this:

> *"The American Heart Association Guidelines notes 'class 2A reasonable to perform' rather than 'class 1 should be performed' indication for an invasive strategy. An invasive strategy is reasonable with patients with mild stage two and moderate stage three CKD. There is insufficient data on the benefit risks of invasive strategy in NSTEMI patients with advanced CKD stages four and five."*

54. They cite the Swedeheart study where no statistically significant benefit was seen with an early invasive strategy at stage four or five renal failure. It is common ground that the Swedeheart study does not necessarily produce high quality data. Dr Cripps however draws attention to the editorial by a Dr Cannon who is, as I think everyone accepts, a world-renowned cardiologist. He said this:

> *"With this study in the back of my mind, when next seeing a patient with NSTEMI and severe renal disfunction who was not on dialysis, I likely would not rush to the cardiac catheterisation laboratory but rather would use intensive medical therapy to stabilise the patient but I would proceed to angiography if the patient were unstable or had significant ischaemia on provocative testing."*

55. In relation to causation, it was agreed that the data was inconclusive on benefit or indeed on survival rates. It was agreed however that CABG would have avoided Mr Paul's death in 2014. Dr McCance's view, having now put a time on it, was that the survival might be 5-10 years. Dr Cripps was prepared to go to five years. Both would discount those figures by 25% if Mr Paul did not comply with his prescription of statins.

56. Giving live evidence, Dr McCance had an unfortunate beginning in that he struggled to correctly formulate the so-called *Bolam* test for negligence, to which I shall return. He was reluctant to acknowledge the significance of a patient's wishes, describing them as secondary in his report before subsequently it seemed to me recanting after being referred to the NICE Guidelines. He also acknowledged that his reference to younger patients benefiting from early angiography was not in the NICE Guidance at the time. Having clearly categorised Mr Paul's GRACE score as being low, he then in evidence described it as being in reality low to intermediate. He accepted that his analysis of the recurrence of pain may have been inaccurate in some respects and/or that the notes could be open to different interpretations.

57. Turning to the American guidance, he agreed that there was no mandate for revascularisation in severe CKD cases. He acknowledged that Professor Cannon was a highly respected cardiologist but described his editorial as being generalised. I noted him give this evidence: *"CKD patients are difficult and the guidelines are not very clear about what should be done and therefore you have to make an individual decision in each case."* Dr McCance was also

of the view that guidelines could only be properly applied to patients who present themselves immediately after a myocardial infarction.

58. Asked about troponin tests, he backed away from his report on the question of repeated tests saying only that they may have been helpful. He agreed that the intervals used by the Defendant were in fact in accordance with the *guidance* paper because they were high sensitivity arrays. He also acknowledged there was no literary support with the proposition that extended troponin testing over 24-48 hours was an appropriate course to take. He acknowledged that the defendant did undertake further tests, GRACE scores, ECGs, echocardiograms all as suggested by the ESC paper. However, Mr Paul was, in his view, in the top category in the table at page 715 of the bundle. He had at least two secondary Table 9 risk factors. In his own personal experience, there was only a 5% risk of an adverse response to angiography, and he was reluctant to defer to the opinion of the nephrology experts. Even if he did, angiography was still indicated in this case. He denied attempting to minimise the risks.

59. So far as the DSE was concerned, he agreed that if Mr Paul had been suffering from severe three vessel CAD at the time he would have expected this to show on the DSE results because of the wall abnormalities. However, he was of the view that this must be an example of a false negative. He was clear that CABG would have been chosen as the way forward if revascularisation was appropriate. He maintained that life expectancy of 10 years was about right, notwithstanding non-compliance with statins.

60. Dr Cripps followed him into the witness box. Asked about ECGs, he said that what was shown on the ones taken by the Defendants were insignificant. He would not describe them as borderline. There will always be a vAryation, even in healthy people. What you are looking for are marked or dynamic changes. He accepted that it was difficult to interpret troponin levels. He acknowledged that those levels could be attributed to NSTEMI or they could be attributable to baseline. He said that renal patients often have high troponin levels. Mr Paul's history was consistent with either interpretation. He would not accept the proposition that troponin levels were prognostically important. He said what mattered was whether you treated someone as having ACS and that is precisely how Mr Paul was managed.

61. He accepted that on admission, the pain appeared to be cardiac in nature. It did not however follow that the pain he had post-admission was necessarily in the same category. It had to be evaluated in the context of the repeat ECGs. The important point to his mind was that the pain had settled after medical intervention. The existence of one occasion of exertional pain was not inconsistent with the expression "no clear exertional symptoms." He denied having misunderstood the dates of the various incidents of pain in his report.

62. Taken to the ESC guidelines, he agreed that there had been a recurrence of pain but all the guidelines needed to be interpreted in the context of the individual, considering the lack of change in troponin levels and the fact that ESC guidelines were generally not appropriate to patients with end stage renal function as opposed to merely being diabetic. The GRACE score he said was not routinely used, but the components were important and they were negative. He declined to agree with Ms Stamp that the assessments made by the treating clinicians were deficient. There were no primary factors in table nine in the ESC guidelines and whilst there were some secondary ones, and whilst with an ordinary patient an angiography might have

been indicated within 72 hours, that was not the case with this patient. He was managed correctly.

63. He rejected the suggestion that Mr Paul had been deprived of an angiogram. He said that in reality what had happened was that further investigation had been suggested before reaching a final conclusion. If angiography had been done, then he said it was not possible to know how much contrast would have had to have been used and therefore how much risk there would be. He said he would have to consult a nephrology colleague before considering revascularisation. He said that it was likely given the passage of time to the point of death and the outcome of the DSE test that coronary artery disease was not sufficiently severe to mandate revascularisation in 2012. He said that medical management can be a successful alternative and that the DSE test is long-established and reliable. You would expect it to detect triple vessel coronary artery disease, and he rejected any suggestion to the contrary. To the extent that any literature suggested otherwise, that was based on asymptomatic patients who might be expected to have a lesser and therefore less detectable level of disease.

64. As far as the Swedeheart study was concerned, he recognised that there were limitations in terms of the numbers, but he did not agree that they were necessarily small. In any event, what mattered was not the numbers for each stage but the clear trend towards poor outcomes with more advanced CKD. He said that there were no studies that showed any better results for younger patients. He also recognised that the cohort in *O'Hara* was older but at four-year survival he considered that age was less significant and younger patients may in any event have a more aggressive disease. He reminded me that Mr Paul had higher BMI than normal and of course had had a TIA. He said again it was a question of what the trends showed rather than the absolute figures. He drew a line on plan, at page 745, which he said justified his estimate of five-year survival rate. He also said that that accorded with the registry data. He rejected the use of the *Kumar* paper as that was for a completely different cohort.

65. The nephrology evidence was shorter and less controversial. Dr Frankel noted that Mr Paul had had poor control of diabetes in the most important first 10 years of his disease. He said that evidence from Imperial College where he works is that 70% of CKD patients have significant coronary artery disease. He considered that life expectancy after a CABG was about eight years. The waiting list for a cadaverous transplant would have been about three to four years. After transplant, his life expectancy would have been 11 years.

66. In his report Dr Andrews accepted that if Mr Paul had had CABG it is likely that he would have activated for transplant in late 2013. He described the 'waiting list' (both experts denigrated the use of this term) as being about five to six years. He said that in his professional opinion it was unlikely that Mr Paul would have survived long enough or at least been in good enough a condition to have received a transplant. If he had had a transplant, he might have lived a further one to two years. He doubted that Mr Paul would have been able to return to work or re-engage with his usual activities during his dialysis.

67. At the time of the joint report the nephrology experts did not know about Mrs Paul's blood type. They agreed that angiography was not absolutely contraindicated in the case of Mr Paul, but it was relatively contraindicated due to his late-stage renal failure. They were agreed on some percentage risks, including a 30% risk of permanent partial loss of kidney function and a 15% risk of precipitating dialysis. They noted that Mr Paul's cardiac condition was stable and therefore urgent revascularisation would have been unlikely considering it might have

taken place at some time in 2013. They differed on the survival benefit of CABG but agreed that Mr Paul would need to have survived more than five years in order to have achieved a cadaveric transplant. In reality, he would probably have become unfit to receive a transplant within two to three years. They agreed that he might have received a live transplant in 2014 using the swap scheme, some six to 12 months later, but there was no agreement on life expectancy following a transplant.

68. Following the joint statement, Dr Frankel wrote two letters after becoming aware of the Claimant's blood type. He confirmed that only the sharing scheme would be available in those circumstances but there was no reason, he said, to believe that the Claimant would not then have been assessed as suitable, noting that she was not diagnosed herself as suffering from diabetes until February 2022.

69. Mr Frankel gave evidence and was cross-examined. He maintained that the registry data was the only reliable source and he was particularly critical of *O'Hara* as being based on a limited sample and a different cohort although he did agree that it did show that outcomes depended on the stage of CKD. He agreed it was standard practice to discuss potential donors in the years leading up to a transplant and he was surprised to see no such discussion. He said 99% of medical letters on this subject would have included some form of leaflet about the options. So far as testing the Claimant was concerned, he maintained that only an HbA1c test would have been used. Given that the first possibility of the Claimant suffering from diabetes arose in July 2021 he had no reason to believe it would have been detected in 2014, particularly given that her BMI was much better then. Shown the PowerPoint presentation relied upon by Dr Andrews, he said that changes in the altruistic donor scheme in 2015 would have meant an improvement in waiting times and indeed the prospects overall. He was not inclined to accept that Mr Paul was sensitised by blood transfusions, although he said that if he had multiple transfusions, things might be, as he put it, a bit more difficult.

70. Dr Andrews told me that he would have expected there to have been discussions about a live donation. Albeit that not all conversations are documented in clinical letters, he would nevertheless have expected any positive offer of any live donation to be documented. He described the approach taken at Imperial College by Dr Frankel and his colleagues as being an 'outlier'. This was certainly so in terms of the practice of performing angiography pre-emptively on all transplant patients. That was not part of common clinical guidance.

71. He accepted that he UK Registry data was the most appropriate source of information and that there were issues with the papers in *O'Hara* and *Kumar*. However, in his view the relevant papers, including the *Gill* paper, confirmed that there was an approximately 1/3 life expectancy advantage. He acknowledged that there had been improvements in the swap scheme in 2015 but, speaking from his position as a member of the Council of Transplants Society, it was still a very grave problem. Whilst the system was more successful after 2015 there were more candidates and therefore the waiting times were really not much different.

72. He said that given that Mr Paul had had more than one transfusion, this was the greatest sensitising event outside of transplant itself and pregnancy. It was however a question of specificity rather than the absolute quantity of antibodies. On the question of whether the Claimant would have been a suitable donor, he disagreed strongly with Dr Frankel on how she would have been tested. He said he had commissioned and authored the transplant

guidelines, and the fasting glucose test was specifically mandated. It is very possible, he said, that if it had been conducted in 2014 it would have been positive.

The Law

73. That concluded the evidence and I turn to the law. The law in this case is not in dispute, although inevitably the emphasis placed upon it differs. It is trite law that in a general sense a professional person is negligent if they fail to demonstrate the care and skill of an ordinary member of their profession. However, there is a refinement in cases of clinical negligence, based on a recognition that clinical judgment is as much an art as a science. A clinician will not be considered to have been negligent if his diagnosis or his treatment would have, or could have, been supported by a reasonable and responsible body of medical opinion even if another reasonable and responsible body of medical opinion would have taken a different view.

74. Some further refinements are needed to that general statement, and they are largely taken from the case of *C v North Cumbria University Hospitals NHS Trust* [2014] EWHC 61. They are these:

(1) The fact that one or more treating clinicians took the course they did does not by itself constitute the necessary body of opinion, nor does the calling of an expert who endorses their decision to take that view have that effect; that would be to delegate the Court's task. However it is also right to say that the responsible body need not be a large one.

(2) The Court has to assess the expert and other evidence to see whether it is reasonable and whether the giver is competent and whether the conclusions drawn are logical.

(3) A responsible expert is one who is willing to make concessions, does not appear to be adopting an unsustainable or partial standpoint and is prepared to change or alter their opinion in the face of changing facts or knowledge.

(4) An expert's logic must be tested for internal contraindications and contradictions in the face of known facts and published guidance and learning. It is to be expected that an expert will accurately record contemporaneous evidence and summarise the applicable medical knowledge accurately.

(5) The standard of care is not one of some hypothetical gold standard. It is an assessment rooted in reality. I must consider how things appeared at the New Cross Hospital in November 2012, not how they appear in the cold light of forensic hindsight 13 years later.

Causation is of course a binary issue. The burden of proof is on the claimant to prove on a balance of probabilities that each successive step in the chain that leads to loss or damage can be made out.

Submissions

75. I have received extremely helpful and (this is not to be critical) lengthy written submissions from Counsel both in advance of and following the trial. I have also had the benefit of their oral submissions. No summary can do justice to them and what follows is a very brief

summary. I repeat however that I have taken the full breadth of the submissions into account in reaching my decision.

76. Having reminded me that all the Claimant's witnesses were themselves consultant cardiologists, Ms Wolstenholme submitted that I should regard Dr McCance as unreliable. She reminded me that he appeared not to understand the legal test and instead appeared to be simply asking himself whether the care was reasonable. She submitted that his views on the significance of patient wishes was inappropriate and submitted that there were a number of other important errors in his report. She drew attention to Dr McCance's refusal to revise his assessment of the risks of angiography in the face of the nephrology joint statement. She said his assessment of the treatment provided lacked balance. As one example, he seemed to have overlooked the significance of high sensitivity troponin. She reminded me that his insistence on repeated troponin tests was not supported in the literature. She submitted that he had failed to properly address the guidelines or guidance and referred to parts of the NICE guidelines which were not in force at the time. By contrast she said that Dr Cripps had made concessions where appropriate and his evidence should be preferred.

77. She took me to the particulars of claim and addressed the four allegations of breach *seriatim*. She said that the treating clinicians had plainly and demonstrably considered and heeded the presence of Mr Paul's multiple risk factors. She reminded me that they had, and demonstrably had, appropriately treated him on the basis that he had ACS. There is nothing wrong with Dr Velu's assessment that there were no clear exertional symptoms. She reminded me that this was a man who had been using the treadmill and the stairs without any other similar results. In any event, there were no repeats in the last 45 hours or so before he was discharged. No one at the time, she said, had assessed the ECGs as being significantly abnormal and in any event, there were no dynamic changes as required by the ESC Guidelines. She submitted that the evidence established that the benefits of angiography for patients like Mr Paul were small and/or uncertain and therefore it could not be said to outweigh the risks. Indeed, she submitted, had angiography been performed and an adverse outcome had been occasioned, that in itself might have been the subject of criticism.

78. Turning to causation, she reminded me that 14 months passed between the events with which I am concerned and the post-mortem. It cannot be proved, she argued, that severe coronary artery disease existed at the time and certainly not to a level which would have justified revascularisation. This was especially so given the DSE test which could not be shown on the balance of probabilities to have given a false negative. In any event, she argued Mr Paul might have declined revascularisation given the potential adverse outcomes.

79. She invited me to reject the evidence that Mr Paul was intending to and/or would have returned to work. He had been off work for seven months with multiple health issues and at very best he would only have returned to limited part-time work. It is unlikely his earnings would ever have crossed the threshold which would have triggered a loss. She submitted that I should accept the nephrologists' assessment of life expectancy and then reduce for his non-compliance with statins and an early start. In reality, Mr Paul would never have achieved a cadaveric transplant. There is no record, she argued, of the Claimant offering herself for transplant, no evidence from Lakhbinder and a limited prospect of a match even under the altruistic donor scheme. She reminded me that Mr Paul had apparently had at least two if not more transfusions. In any event, there was a distinct possibility, if not probability, that the Claimant herself would have been diagnosed with diabetes at about the time the transplant

came to be offered. She made detailed submissions on aspects of the special damages claim which I do not propose to delve into.

80. Ms Stamp began by reminding me that the clinical records showed that multiple risk factors were present. Troponin levels were high and prognostic even if they were exacerbated by CKD. What the cardiac ward did following Nurse Shelley's assessment was to, as she put it, downgrade the risk. Dr Velu had failed to demonstrate the required professional curiosity as illustrated by his rather skimpy notes. Whilst Mr Paul may have expressed concern about the risks of angiography, that could equally have been the product of the inadequate advice he was receiving. Given that the notes were apparently a continuum, she queried whether any of the subsequent clinicians did more than accept Dr Velu's assessment. Certainly, Dr Khogali did not appear to have considered the evidence that a NSTEMI had occurred and appears instead to have placed Mr Paul in the bottom category of risk. In Dr Arya's case, she ruled out NSTEMI by attributing his troponin to CKD and her conclusion is difficult to support. It was not reasonable to consider that Mr Paul's cardiac symptoms were possible as opposed to probable. If she had properly assessed the risk, she would have referred him for angiography.

81. Ms Stamp described Dr Cripps' evidence as an exercise in *ex-post facto* rationalisation and as constructing a defence, rather than being an independent appraisal. She asserted that he had snipped extracts from the ESC Guidelines selectively to make his case for the appropriate treatment. She said he also gave an imbalanced account of Mr Paul's diabetic control by referring only to parts of the clinical correspondence at the time. He had conflated separate reports of pain and appeared to have mixed up his dates. His exercise of drawing on the chart at page 745 showed that he was taking a partial view. His reference to *O'Hara* was unhelpful and inappropriate. Dr Cannon's editorial was no more than a generalisation and, perhaps, a provocative challenge to conventional wisdom.

82. In relation to causation, she invited me generally to adopt the benevolent approach suggested in *JAH v Burne and Others* [2018] EWHC 3461 QB. There was no reason to think that Mr Paul would have declined angiography or indeed revascularisation or that he would have been unsuitable for either of those treatments. There were good reasons why he had expressed reluctance in relation to other forms of treatment. The *Kumar* paper supports Dr McCance's opinion that angiography would have shown severe three vessel disease mandating CABG. She argued I should find that Mr Paul and his wife had discussed a transplant, that she would have made the offer, and that he would have accepted it. There was no basis for suggesting that the Claimant would have been diabetic at the time. She reminded me that any change in her BMI came later and in very difficult personal circumstances following Mr Paul's death. She acknowledged that the prospects of success in the swap scheme were, as she put it, problematic but they were improving at this time. The records so far as sensitivity is concerned do not suggest any more than one or possibly two transfusions. Again, she too addressed me on the question of special damages.

Conclusions

83. My conclusions in this matter are these. First, I must assess the evidence that I heard. In my judgment the Claimant gave her evidence in a clear manner. Her affection for her late husband and her belief that he had been the victim of clinical failures were clear. It seemed to me that this was the perspective from which she viewed events that happened, as I have already said, now some 13 years ago. One illustration of this was her unwillingness to acknowledge the

fact that Mr Paul was not always good (and I find he was not always good) at keeping appointments and taking his medication. Another aspect was her evidence about Mr Paul planning to return to full time work the day after he died. Leaving aside minor inconsistencies between her evidence and that of her sister-in-law, there is unfortunately a real improbability in the proposition that someone employed by a major company who had been off work for a very considerable period of time, who was still very unwell, who was unable to work more than two days per week and who was changing work location, would simply 'go back to work'. It is curious in these circumstances that there is no HR or other employment documentation to suggest that was what was going to happen. It may be that Mr Paul was going to attend a meeting about going back to work. It may even be that he reassured his wife of that fact. I fear however that the passage of time has conflated what might have been the first tentative step into a leap.

84. In my judgment, something similar probably has to be said about discussions relating to a live transplant. I do not doubt that both the Claimant and Mr Paul were aware that live donation was one option. I acknowledge that the absence of mention of the identity of a donor in the correspondence is not conclusive given it is at a relatively early stage. There may have been some discussion, but I am not persuaded on the balance of probabilities that it went much further than that. I acknowledge that the Claimant's evidence derives some support in this respect from her sister-in-law, but I suspect the latter's recollection is very much conditioned by what she has heard from the Claimant herself and does not persuade me to take a different view. In any event, I fear that it is academic for reasons that will become clear.

85. I turn to the Defendant's evidence. It would not be expected that any of the treating clinicians had any memory of Mr Paul and none professed to do so. It is important therefore to recognise at the outset that their evidence is in reality no more than a hindsight interpretation of their contemporaneous clinical notes viewed through the lens of their general practice "what I would do in these cases." To that extent one might say it has relatively limited value. I felt Dr Velu gave his evidence in a measured and honest manner. He made some appropriate concessions although I acknowledge that he did have some difficulty in dealing with the issue of exertional symptoms. However, I was not persuaded that he lacked professional curiosity; it was very clear from his evidence that he regarded this as the first step in a team decision in which he was deferring to his consultants.

86. At the other end of the clinical history chronologically speaking we had Dr Arya. I fear her evidence was less impressive. Quite aside from the unfortunate change of her categorisation of Mr Paul's symptoms, I formed the impression that she did not always answer Ms Stamp's questions directly (for example in relation to troponin levels). She seemed to me on occasion to be providing an *ex-post facto* justification for her understandably sensitive decision to discharge Mr Paul. That said, it is important to recognise that this was a continuum. The course of treatment had been set by Dr Khogali. Whilst as a professional she needed to make her own assessment, there was no reason for Dr Arya to reach any different conclusion, provided of course that there was no change in Mr Paul's condition. As she said in her evidence, it was for this reason that she agreed with the conservative approach that had already been set.

87. That brings me to Dr Khogali. I considered he gave his evidence in a calm, assured, fair and I must say deeply impressive manner. He made fair concessions to some of the questions asked. He gave convincing answers to others, for example about the potential source of

Mr Paul's limited exertional symptoms, as to the expected troponin levels in late-stage CKD patients and as to the significance of ECGs. However, curiously the key value of his evidence was that this was a consultant known by his colleagues to be interventionist rather than conservative, who said himself that he was experienced in and enjoyed angiography and revascularisation. Yet coming to Mr Paul from that perspective, he did not consider that intervention was mandated in his case and that in all the circumstances it was better to adopt a wait, look and see approach.

88. I turn then to the expert evidence. It is undeniable that Dr McCance did not have a good start. I might in other circumstances have been prepared to recognise that this was a forensic rather than a substantive victory for Ms Wolstenholme. Unfortunately, the real difficulty with Dr McCance's evidence was that I was left with the impression that he was in fact applying a higher standard than the *Bolam* test that I have already enunciated. I was left with the impression that what Dr McCance was telling me was what he thinks he would have done rather than what others might have done. In my judgment, he also gave a clear impression that he was unwilling to concede any ground that might adversely affect the Claimant's case. That was marked particularly in questions about the patient's wishes, where he had to be forced reluctantly to concede that they were of more significance than he had at first suggested. I noted also his attempt to recategorize the GRACE score, his initial description of ECGs as being not valuable and his evidence that CKD patients should be treated like any other patient, before then acknowledging that they did present difficulties.

89. The proposition that repeat troponin tests should have been carried out was a mainstay of his initial report. However, in his evidence he rowed back from that recognising that there was no support for it in the guidance, stating only that it may have been helpful. Confronted with passages in the various different sources of guidance that appear to support the treatment that had been given by the Defendant, his response always was to say that the treatment of Mr Paul should have been tailored to the individual risk. There was an element, I felt, of picking and choosing. Finally, I noted his disinclination to accept the nephrologist combined joint assessment of the risk of angiography, a risk which at nearly one in seven, or worse than one in seven, was on a Russian roulette basis really quite significant.

90. I acknowledge that Dr Cripps' evidence was also not without its problems. I acknowledge that his line drawing on the plan at page 745 might have been on the optimistic end of the spectrum, although it is actually very difficult to know exactly where the line should be drawn. I also note that he struggled to a degree with Ms Stamp's criticism of him as having 'snipped' selectively from the ESC Guidelines. However, I do not accept that he was genuinely confused about the timings of the reports of pain. In other areas he made sensible concessions. To give one example, he conceded that there was other correspondence congratulating Mr Paul on his compliance with his diabetes treatment. He was impressive with his explanations of aspects of his evidence, for example as regards the probability of the DSE test providing a false negative and the reliability of trends as opposed to absolute figures in the various studies. Overall, Dr Cripps stuck to his basic proposition. Taking this patient as a whole, particularly his progress and the lack of any adverse signs in the last 45 hours of his admission (I find as a fact that the last pain was at 8pm on the Saturday evening), a conservative approach was not only reasonable but appropriate. Generally, he gave to me a much clearer sense of addressing the practicalities of treating patients like Mr Paul in an NHS setting than a more academic dissection of the rights and wrongs of any particular course of action or academic paper.

91. The one area of common ground between both experts is that clinicians treat the individual patient in front of them. There is no checklist. They do not hold the ESC Guidelines or the American Guidelines in one hand as they decide how to treat an individual patient. Those guidelines are not conclusive as to how to treat any individual. They are just part of the background knowledge that informs clinical judgment. I remind myself of course that Mr Paul's treatment was strictly in accordance with the NICE Guidelines. I note and agree with Dr McCance's evidence that I have already read, and I will read again; "CKD patients are difficult and the guidelines are not very clear about what should be done. Individual decisions have to be made."

92. The nub of this case is that in this kind of situation there will always be a variety of approaches that are possible. Picking bits and pieces from guidance and criticism of the adequacy or otherwise of notes is not always terribly helpful. It is clear to me that the Defendant did treat Mr Paul as an ACS risk patient. It is clear to me that they did have in mind his various risk factors. The Claimant fails to satisfy me on a balance of probabilities that Dr Khogali did not belong to a body of responsible clinicians practising at the time who would probably have taken the same approach. The editorial by Dr Cannon merely provides some reinforcement. Even if Dr McCance is right that the vast majority of treating clinicians would not have taken the course that the Defendant took, that of course does not exclude the possibility that a significant minority might well have done. That would be sufficient to satisfy the *Bolam* test.

93. It follows I am afraid that it is unnecessary for me to deal at any great length with the remainder of the evidence but in deference to the arguments I will offer a few brief conclusions. Both of the expert nephrologists were in my judgment impressive. They both made appropriate concessions. The area of dispute between them was quite limited but where there was dispute, I prefer the evidence of Dr Andrews. I felt that Dr Frankel came to this case with the quite limited perspective of his work at University College and the particular practices adopted there. Dr Andrews struck me as having a much wider experience, being on the Transplant Council, authoring guidelines and so on.

94. This is particularly so it seems to me as regards the prospects for a live transplant. Even allowing for the benevolent approach that Ms Stamp urges upon me, I consider that if angiography had been performed it would have demonstrated some diffuse but not severe coronary artery disease in 2012. I reject the submission that on a balance of probabilities that it can be shown that the DSE test provided a false negative, particularly in the context of the death coming over a year later. Accordingly, this was something that would have been referred to the multi-disciplinary team. Whilst I am not satisfied that Mr Paul would have refused any recommended treatment, I am also not persuaded on the balance of probabilities that that treatment would have been recommended. I am further not satisfied that the Claimant has proved on a balance of probabilities that, whether a transplant was offered on the altruistic donor swap scheme or on a cadaverous basis, that he would have been able to obtain such a transplant either before he died or perhaps more pertinently before he reached a point at which he would no longer have been considered viable to receive that transplant. I find that Mr Paul's compliance with statins would probably have been intermittent. In the light of those conclusions, I do not propose to say anything about quantum as it is hypothetical.

End of Judgment.

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This transcript has been approved by the judge.