OUTER HOUSE, COURT OF SESSION

[2017] CSOH 123

A474/14

OPINION OF LORD ARTHURSON

In the cause

HEATHER BRUCE AND OTHERS

Pursuers

against

TAYSIDE HEALTH BOARD

Defenders

Pursuer: A Galbraith; Thompsons
Defender: MacSporran; NHS Scotland Central Legal Office

19 September 2017

Introduction

[1] Mr David Bruce was born on 11 March 1949. On 12 August 2011, at the age of 62, he or someone on his behalf had cause to call for an ambulance and thereafter he was admitted to Ninewells Hospital, Dundee. He had been married to his wife Mrs Bruce, the first pursuer, since 1971, and they had a particularly close bond. At the time of his admission to hospital he was in fulltime employment and had just returned from a walking holiday to Austria. He had a close, loving and supportive family. Following his admission to hospital, which is timed in the records at 2125 hours, Mr Bruce ultimately died and was pronounced dead in the early hours of the next morning, 13 August 2011, at 0241 hours. In this case the

medical facts pertaining to Mr Bruce were not in material dispute, and with the exception of one very limited matter, quantum of damages was also agreed. Further, the defenders had at an earlier stage of pleading tendered a Minute of Admission setting out the parameters of the agreed scope of negligence pertaining to the treatment of Mr Bruce in the emergency department at Ninewells, which was in the following terms:

> "the defenders admit that on the night of 12/13 August 2011 at the emergency department of Ninewells Hospital, Dundee the defenders' staff failed to commence the late Alexander Bruce [the deceased] with IV fluids and hyperkalaemia treatment timeously and the deceased should have been commenced on (a) IV fluids (0.9% saline) by 2225 hours and received 2 litres within 2 hours, ie by 0025 hours; and (b) treatment for hyperkalaemia by 2325 hours by way of calcium gluconate (10 mls of 10% solution) and dextrose/insulin infusion (50mls at 200mls per hour, ie over 15 minutes)."

[2] The only issue on which the parties went to proof was accordingly that of causation. Each side led evidence from a single expert respectively to address the following single issue raised in this case, which was this: what would have been the likely outcome for Mr Bruce in terms of his survival had the admitted duties, referred to above, been complied with by the defenders' medical staff. No issue of law arose in this case. Instead, the parties, through the diligent and highly professional care of their counsel, had in terms agreed the facts and evidence of nursing staff and each simply invited the court to prefer the opinion of their respective expert on the single issue of likely outcome. I would like to commend counsel at this stage for their exemplary preparation for and conduct of the case. The proof before me, which although it was set down originally for two weeks, was completed, standing the scope of the agreements which I have mentioned, within three days, including submissions.

Hospital Admission, Co-morbidities and Post-mortem Findings

[3] The ambulance crew on 12 August 2011 arrived at Mr Bruce's home at 2045 hours. His skin colour was noted to be flushed. His respiratory rate was noted at 2050 hours to be 40bpm and at 2110 hours to be 30bpm. He presented with a seven or eight day history of diarrhoea and vomiting which had been treated by his GP. It was noted by the ambulance crew that Mr Bruce had in the course of the day become increasingly short of breath and was hyperventilating. He arrived at Ninewells at 2125 and was assessed by nursing staff at 2140 hours. It was recorded that he had been suffering from abdominal pain and diarrhoea for the past two days and was hyperventilating and agitated. He was talking in sentences and making sense. He was drinking from a bottle of water. He was able to slow his breathing down voluntarily on request but remained agitated and restless. On admission his respiratory rate was recorded as 50bpm. Triage was undertaken at 2150 hours when it was noted that Mr Bruce was voluntarily able to reduce his respiratory rate. He gave a five day history of his stomach problem. Mottling to his abdomen and legs was observed at 2201 hours, which mottling was described by a healthcare assistant as "from the bellybutton down and also on his shins" and "just from his bellybutton down to the top of his groin area and on his shins". At 2150 hours his respiratory rate was noted at 50bpm and his blood pressure at 180/81.

[4] At 2235 hours Dr Paterson recorded an eight day history of diarrhoea and observed mottling on Mr Bruce's legs. His legs were of different skin tones, with shades of darker and lighter patches described by Dr Paterson as "blotchy". Dr Paterson ordered a sample to take blood gases and prescribed IV fluids. Parties had helpfully agreed by way of Joint Minute that Mr Bruce was commenced on IV fluids (0.9% saline) and received the following amounts: (a) 500ml between 2335 hours and 0030 hours; (b) 500ml between 0035 hours and

about 0135 hours; (c) 1000ml between 0005 hours and 0100 hours; and (d) 1000ml between 0100 hours and about 0145 hours. At 0145 hours Mr Bruce was commenced on 500mls of gelofusine and at about 0115 hours he was commenced on hyperkalaemia treatment and received (a) 10mls of 10% solution of calcium gluconate and (b) 50mls of 50% dextrose/insulin infusion at 200mls per hour. At 2340 hours Dr Thakore met Mr Bruce in the accident and emergency department. On examination he noted cool peripheries and mottled skin. He noted an impression of diarrhoea leading to dehydration leading into renal failure. At 2350 hours Mr Bruce was surgically reviewed in accident and emergency. His blood pressure was noted at 99/54 and his respiratory rate at 45bpm.

[5] I turn now to the events after midnight, that is in the early hours of 13 August 2011. At 0020 hours Dr Thakore spoke to Mr Bruce's family. He apologised for the delay in recognising Mr Bruce's illness and advised that treatment should have commenced on his arrival at Ninewells. Dr Thakore recorded the following note at this time: "Mr Bruce's condition was so poor that I do not believe earlier recog(nition) would necessarily have affected outcome". While venous blood results at 2321 had shown pH at 6.757, arterial blood results at 0027 disclosed a pH figure of 6.793. Biochemical blood results at 0002 hours showed figures of 7.7 potassium, 1441 creatinine, and 14.5 lactate. At 0155 hours Mr Bruce suffered a cardiac arrest. He was transferred to the intensive care unit at 0210 hours and pronounced dead at 0241 hours.

[6] The post-mortem examination report prepared by Dr Sadler followed examination of Mr Bruce's body on 22 August 2011. The only medical cause of death was recorded as atherosclerotic coronary artery disease. Dr Sadler made the following comments in his report:

> "Death is attributed to atherosclerotic coronary artery disease. This would account for sudden death due to a heart attack or a disturbance in the electrical rhythm of the heart. The presence of minor gastritis might account for his abdominal symptoms. No adequate explanation for shortness of breath was detected at autopsy."

Dr Sadler further stated:

> "Autopsy disclosed severe hardening and narrowing of one of the three main blood vessels supplying blood to the heart (atherosclerotic coronary artery disease). This portion of vessel had been previously surgically treated by the insertion of a wire tubular stent. The degree of stenosis and narrowing at this point was sufficient in itself to account for sudden death due to a heart attack or a disturbance in the electrical rhythm of the heart. There was no evidence of pneumonia to explain his shortness of breath prior to death."

It should be noted that the respective medical experts for each side in this case of course had the benefit of the post-mortem examination findings to inform their opinion on outcome.

[7] Before considering the expert opinion evidence, I now wish to record the general health of Mr Bruce as at his admission to Ninewells Hospital in August 2011. He had, on any view, significant co-morbidities. In October 1995 Mr Bruce was referred to a diabetic clinic for Type 2 diabetes mellitus. In 2001 it was recorded that he had some early background retinopathy in both eyes. In July 2011 his height and weight were noted as 185 centimetres and 109 kilogrammes with a body mass index of 31.85. In July 2006 he was admitted for angioplasty to his proximal left anterior descending coronary artery, being the artery referred to by Dr Sadler as the stented artery in the post-mortem examination report. Hypertension is recorded in his medical history from October 1995. Medication taken by Mr Bruce included bisoprolol, losartan and metformin.

The Evidence of the Experts

[8] Turning to the expert evidence presented at proof, I observe at the outset that I found both clinicians to be highly impressive and more than qualified to give opinion evidence in a

clinical negligence dispute of this nature. Professor Patrick Nee, a consultant in emergency medicine and critical care medicine, had 23 years of practical experience as a consultant, had been involved in important research and was a member of a number of advisory bodies. He had published a paper in 2016 concerning critical care in the emergency department with reference to acute kidney injury and had considerable clinical experience of the management of critically ill patients with acute kidney injury. Rather intriguingly, in his CV it was recorded, and confirmed by the witness in his evidence in court, that he prepared on average about 600 medical legal reports per annum. His current practice involved approximately one third NHS clinical work and two thirds medical legal work. Dr Monika Beatty, a consultant in critical care at Edinburgh Royal Infirmary, was the regional advisor in training for the south east Scotland area in critical care. She had been an examiner for the fellowship exam for the Faculty of Intensive Care Medicine since October 2016, and had been admitted as a fellow of the Royal College of Physicians in 2010. Her unit in Edinburgh was the second largest critical care centre in Scotland and was a regional centre for renal disease and transplants. Her unit dealt with 1317 ICU admissions per annum and 13% of her patients required renal replacement therapy. In stark contrast with Professor Nee, the present case represented her first instruction as an expert witness.

[9] Professor Nee's evidence can be summarised in its salient points in the following terms. With regard to outcome, he highlighted that a capacity to work, exercise, self-care and a reasonable exercise tolerance, all of which he identified in Mr Bruce, were good survivor indicators. He accordingly ascribed significance to Mr Bruce's functional status prior to his admission to Ninewells. He was still working as an engineer and had been on a walking holiday in Austria. On arrival at Ninewells he was mobile and independent. These were indicators that there was no severe organ failure on admission. Mr Bruce's death had

occurred because of profound shock. Fluid had been lost from the gastrointestinal tract and accordingly other fluid had shifted within Mr Bruce's body to maintain cardiovascular volume by way of a compensatory mechanism. The object of the heart circulating blood was to deliver oxygenated blood to other body organs. A failure in circulation led to cells metabolising without oxygen, and, in particular, cell membranes would break down and ions, especially potassium, would spill out into the general circulation. High potassium could affect the electrical circuitry in the heart and cause cardiac arrest. Mr Bruce's cardiac arrest had been produced by acidaemia, which amounted here to an inability of the relevant electrical signals to reach the heart muscles. The kidney is one of the first organs to feel the deprivation of a lack of blood circulation. Professor Nee spent some time in his evidence discussing a five stage progression of shock model, emphasising that shock represented a deprivation of oxygenated blood and noting that in the context of Mr Bruce's presentation volume resuscitation was likely to be effective right up until the final stage of the five stage model, which he termed as terminal or irreversible shock. There had been in this case a three hour window of opportunity from Mr Bruce's initial presentation at Ninewells until approximately 0030 hours the next morning, at which stage Professor Nee accepted, Mr Bruce's terminal decline was underway. During that window of opportunity, in the opinion of the witness in terms of his written report:

> "vigorous intravenous volume resuscitation in an appropriate setting would have prevented his decline to cardiac arrest. Correct treatment with 3 or 4 litres of warmed fluids within that interval would have made a significant difference in my opinion and, on the balance of probabilities, he would have survived this episode sufficient to be transferred to a critical care unit for haemofiltration."

This volume figure is not of course the volume referred to in the Minute of Admission for the defenders which represents the agreed normative treatment by the defenders' medical

staff in this case. The pursuers' counsel accordingly put the terms of the Minute in detail to Professor Nee. The witness responded that if adequate volume had been received in a timely manner, Mr Bruce's condition would have improved sufficiently for him to be transferred to intensive care where he would have received treatment for his acute kidney injury. Sick as he was, he would have stabilised with the level of treatment and care referred to in the Minute. What was important was timely intervention in the resuscitation room within the three hour window. Although that remained Professor Nee's core position throughout his evidence, he very frankly described the provision of 2 litres of IV fluids within 2 hours as a "fairly modest ambition" and at one point, when asked what difference this would have made, observed "one would hope his clinical condition would have improved". He stated that it would not be right to view any decline here as linear in form. Mr Bruce was extremely acidaemic at presentation and his eight day decline was reaching a crisis point by the time he arrived at Ninewells; the failure to treat at Ninewells, however, contributed to what the witness described as an accelerated decline. This was a potentially reversible illness. The witness was critical of certain articles referred to by Dr Beatty at paragraph 3.19 of her principal report and of her use of the APACHE II (Acute Physiology and Chronic Health Evaluation) Scoring System from which she reached a score of 28 points which she described as representing severe critical illness. Although Professor Nee was critical of this model, he himself reached a score of 27 points using it. He described it as a very old-fashioned model which was not properly used to determine a prognosis in individual patients, and should never be used prospectively. At the end of his examination-in-chief he expressed the view that choosing the correct approach to questions of survival and outcome was best achieved by an experienced physician and intensivist.

[10] In cross-examination Professor Nee accepted that Mr Bruce's medications, namely losartan and metformin, were nephrotoxic, that is to say potentially damaging to the kidneys. He further accepted that in the case of a severely dehydrated patient taking such medication, this dehydration would compound the effect of such nephrotoxic drugs. He accepted that when Dr Thakore examined Mr Bruce at 2340 hours on 12 August, Mr Bruce had been profoundly dehydrated. He accepted that during admission Mr Bruce suffered from severe or very severe acute kidney injury. The creatinine level of 1441 recorded at 0002 hours on 13 August was very high. The level of creatinine on admission must also have been extremely high and indeed would have been close to the figure recorded at 0002 hours of 1441. The witness noted that he had seen this level of acute kidney injury resolve in an elderly male patient with gastrointestinal losses. He agreed that respiratory rate was a very sensitive marker of critical illness and that the respiratory rate of 50 recorded in the SEWS (Scottish Early Warning Scores) chart in the records on admission was exceptionally high. The level of lactate of 14.5 recorded at 0002 hours was high and was consistent with severe shock and metabolic acidosis. The level of potassium recorded at 0002 hours of 7.7 was very high and would have been very high on admission also. When counsel for the defenders put the Minute of Admission again to Professor Nee, the witness reiterated that the figure of 2 litres within 2 hours was a very modest one. When asked whether the potential infusion set out in the Minute of Admission would or would not have been enough to change the outcome for Mr Bruce, the witness observed that this was "very difficult to say". In his view, 2 litres was better than 1 litre but was not sufficient. Anyone would find this matter difficult, he observed. His nuanced approach to the matter was as follows: rather than considering whether a 2 litre infusion would lead to survival, to consider instead whether it would lead to a widening of the window of opportunity to lead

Mr Bruce to the intensive care unit and survival beyond that Unit to discharge. The witness expressly agreed that he had a difficulty in confirming that the extra litre would have made a difference, but couched his final position thus: he was "not content to say that it would have made no difference".

[11] Professor Nee addressed finally the minor outstanding quantum issue between the parties, namely whether Mr Bruce would, had he survived this episode, have returned to work. He expressed the general view that survivors tended to be free of dialysis in the longer term but very properly accepted that one would need the opinion of a nephrologist on renal outcome. Here, however, Mr Bruce was a maintenance engineer. He had no cancer and no overwhelming sepsis. The witness accepted that medical literature could be a useful tool on return to work, but offered none himself.

[12] I now turn to summarise the position in evidence of the defenders' expert, Dr Monika Beatty. The respiratory rate of 50bpm on admission was an indication that Mr Bruce was a critically ill patient on arrival at Ninewells. His high creatinine level was consistent with a very severe kidney injury; in effect, his kidneys had shut down and he was relying on respiratory compensation to eliminate acid by breathing methods. His high lactate indicated a high level of organ dysfunction and tissue hypoxia. He was profoundly dehydrated on admission with an accumulative loss, in the witness's opinion, of 10 litres of fluid over a number of days, standing the creatinine level. This loss had led to acidosis and a high lactate reading. The five-stage shock model spoken to by Professor Nee was not applicable in a case such as that of Mr Bruce, as that model related to patients presenting in respect of a traumatic incident leading to an acute loss of blood. Further, it was not correct to view function level as the best indicator of post-admission recovery; instead, the most important factors in this matter were diagnosis on presentation, the degree of organ

dysfunction at presentation, and the response to organ resuscitation. Pre-morbid functioning would be a relevant factor, but would not be the major determinant. Mr Bruce's diabetes predisposed him to renal disease and his hypertension disposed him to acute kidney injury. His diabetes was not optimally controlled, and complications such as retinopathy had obviously been developing. Mottling was a clinical sign of shock but did not determine the response of a patient to shock; it was simply a warning sign that someone was unwell. It was accordingly not accurate for Professor Nee to characterise her position, as he did at page 4 of his report, to the effect that Dr Beatty pointed "to mottling as evidence of shock of such a severe nature that treatment with intravenous volume resuscitation would not have been effective".

[13] In more detail, Dr Beatty highlighted the respiratory rate of 50bpm recorded early in the records and considered this to be a marker of the severity of Mr Bruce's illness on presentation. In addition she highlighted the creatinine level of 1441, noting that it was extremely rare for anyone to present with a level of over 1000. She herself had never seen an acute kidney injury patient presenting with creatinine at this level. By way of comparison, on 2 August 2011, the figure was 128. Her own position was that, working back to admission, Mr Bruce's creatinine figure would have been exceptionally high on admission. A pH level of under 7 represented exceptionally severe acidosis, the normal range being 7.35 to 7.45. In her view Mr Bruce's pH level would have been under 7 on admission. The base excess level recorded at 2321 hours of -32.2 was consistent with very severe metabolic acidosis, the normal range being -2 to +2. The elevated lactate signified hypoxia and equated to a higher mortality and poor prognosis. The terms of the post-mortem report were also significant. In the witness's view, Mr Bruce was at much higher risk of cardiac arrest than someone who had not had stenosis of the artery referred to in that report. Issues of the

passage of time were also significant. To perform renal replacement therapy it was necessary to place a specially designed central venous catheter into a major vein in the neck or leg, which would have taken time; Dr Beatty elaborated on this point at paragraphs 3.14 to 3.15 of her report, summarising her position in terms that it was extremely unlikely, even had Mr Bruce been assessed formally by senior medical staff on admission, that it would have been possible to have commenced him on renal replacement therapy much before the acute deterioration at 0040 hours. Her own assessment on a percentage basis of Mr Bruce's predicted mortality from acute kidney injury was in the region of 60% to 80% (para 3.19). Dr Beatty referenced certain articles in coming to this view, which articles had been the subject of criticism by Professor Nee.

[14] Dr Beatty was asked about Professor Nee's preferred infusion of 3 to 4 litres of fluids. In her view a large volume of fluid resuscitation was undoubtedly indicated. Nevertheless this would not have corrected the acidosis sufficiently to prevent Mr Bruce's subsequent cardiac arrest. What was needed was in fact renal replacement therapy, a severe acute kidney injury having already been established. Renal replacement therapy was urgently required to correct the acidosis and treat the hyperkalemia, being the raised concentration of potassium in the blood stream. She accepted that APACHE II had its limitations but described it as a useful descriptor of how critically unwell the patient was and indeed it was routine in Scottish practice to use this tool for that purpose. This was, further, the longest established of all scoring systems. In no sense was it her view, however, that it should be used for individuals to predict the likelihood of outcome or survival.

[15] On the disputed issue of employment, the witness was taken to certain entries in the medical records concerning Mr Bruce's work with heavy tools as a mechanical engineer. She spoke to a recently evolving post-intensive care unit syndrome from which she drew the

inference that intensive care patients of longstanding had a reduced ability to return to past employments. She referred to a series of papers which had been produced by the defenders in process and from which she concluded it was very unlikely that Mr Bruce would return to work, given his age at the time of admission.

Submissions for the Pursuers

[16] Counsel for the pursuers invited the court to prefer Professor Nee and to accept his opinion of Mr Bruce's prognosis, commending him as an expert witness in a case of this nature. It was submitted that the extent and breadth of Dr Beatty's experience was considerably more limited, in contrast. The court was asked to agree with Professor Nee and to conclude that in considering the assessment of the likely prognosis for Mr Bruce on 12 August 2011, the best, and arguably only, methodology was one based on practice, knowledge and experience as applied to the particular circumstances of the individual case. It was submitted in contrast that Dr Beatty had not carried out such a task but instead her assessment of Mr Bruce's predicted mortality came from articles that she had referred to at paragraph 3.19. In one of the articles (*Uchino*), only one hospital out of 54 was in the United Kingdom and it could not be said what other health conditions the patients in that study had which could have affected the prognosis. The second paper (*Tolwani*) was also criticised as not representing independent research at all but simply a commentary on other papers. According to Professor Nee's analysis of the progress of Mr Bruce's condition as at the point of his arrival at hospital, he had acidosis and kidney injury and had been compensating; nevertheless his consciousness level was intact and he had not yet reached a stage that appropriate treatment would have been unlikely to succeed.

[17] Counsel emphasised the importance ascribed by Professor Nee to prior functional status when considering prognosis. She further noted the particular example given by the witness of treating the same level of acute kidney injury in an elderly man who had survived, in the context of a high level of creatinine. It was to Professor Nee's credit that he had accepted that, given his view that more ought to have been done than was set out in the Minute of Admission, this was a difficult assessment. Nevertheless, on careful consideration his view was that on a balance of probability the infusion referred to in the Minute would have allowed Mr Bruce the opportunity to maintain treatment such that he could have been transferred to the intensive care unit in order to receive renal replacement therapy. When giving evidence on the new parameters set in the Minute of Admission, his position was that the matter was finely balanced, but his position remained that it would have been enough to point to survival. Counsel noted that due to the failure by the defenders to appreciate Mr Bruce's condition on arrival, there were no test results before 2321 hours. It was simply not a valid exercise to compare results taken at 2321 and 0027 with a hypothetical outcome of fluids being given at 2225. Mr Bruce's condition was deteriorating rapidly over the course of the few hours that he was in hospital, and it was accepted by counsel for the pursuer that by midnight he was essentially beyond help. There was a significant deterioration in his condition between 2225 and 2335 hours. It was not a logical approach to assume that the change in his condition between 2335 and 0027 hours was an appropriate comparator with a likely outcome had he had treatment between 2225 hours and 2335 hours.

[18] With regard to capacity for work, it was accepted by counsel that it was uncertain what the future would have held for Mr Bruce, but the court was asked to find that he would have worked again before retirement, following a lengthy period of recovery, standing his established work history, albeit it was accepted that Professor Nee had

indicated that it was a matter properly for a nephrologist. Counsel addressed the court on all of the quantum figures, which were ultimately agreed by the defenders' counsel and which I do not propose to rehearse here, on the basis of a return to work on 1 March 2012 until retirement at 65.

Submissions for the Defenders

[19] Counsel for the defenders submitted that it had in terms been agreed by both experts that Mr Bruce suffered from profound dehydration on admission and further that he was suffering from a very severe acute kidney injury at that time. The experts further agreed, it was submitted, that the venous gas results at 2321 showed a very profound metabolic acidosis and he pointed out that Dr Beatty had been confident that on admission the pH level would have been less than 7. The respiratory rate recorded on admission of 50 was exceedingly high and both experts agreed that this was a very sensitive marker of critical illness. The lactate result at 0002 hours was agreed by the experts as being very high at 14.5. Professor Nee had accepted that this was consistent with severe shock, severe dehydration and severe metabolic acidosis. The experts of course parted company as to the conclusion to be drawn from this largely agreed background.

[20] Counsel for the defenders invited the court to prefer the evidence of Dr Beatty and to conclude that even with the agreed non-negligent treatment set out in the Minute of Admission, Mr Bruce, on a balance of probabilities, would not have survived. It was submitted that the defenders' expert should be preferred for a number of reasons. Her report on causation was detailed, researched and reasoned; in addition it was supported by medical literature and cross-checked with the APACHE II scoring system. Dr Beatty did not consider functional status prior to admission to be an important determinant of survival.

The three important factors which she highlighted were diagnosis at presentation, degree of organ dysfunction and response. In this case it was clear that there was a very severe organ derangement with many readings described as exceedingly high (namely respiratory rate, creatinine, acidosis, potassium and lactate). Professor Nee's reliance on pre-admission functioning was not a reliable gauge and paid insufficient regard to this derangement. The other cornerstone of Professor Nee's contention was the stages of trauma shock scale which did not feature anywhere in his report and in any event related to acute haemorrhaging rather than hypovolaemia, being fluid lost from the body. Professor Nee provided no contrary literature supporting a lower mortality rate percentage to set up a contrast with Dr Beatty's medical papers produced in process. Dr Beatty sought support from the APACHE II Scoring System with the clear caveat that such a scoring system was not a predictor of individual mortality but instead was an additional source of information. Dr Beatty, on the principal issue of survival, was clear, on a balance of probabilities, with particular reference to the underlying acidosis, that Mr Bruce would not have survived. She was unshaken under cross-examination. Professor Nee's personal position, in contrast, was that correct treatment involved an infusion of 3 or 4 litres of warm fluids. In cross-examination he accepted that the issue of whether the agreed fluid would have made a difference was a very difficult question. He seemed uncomfortable with the agreed amounts of fluid. Professor Nee's position was over-simplistic and amounted to concentrating on the pre-admission functioning issue at the expense of the accepted profound derangement, ignoring the literature and belittling the scoring system used throughout Scotland. In addition he had paid insufficient regard to the agreed amount of IV resuscitation. He had overstated Dr Beatty's reliance on mottling and misunderstood her approach to the use of the APACHE II Scoring System. It was of note finally that Dr Beatty gave unchallenged

evidence that it would be extremely unlikely that it would have been possible to commence Mr Bruce on renal replacement therapy much before the acute deterioration, which in itself was fatal to the pursuers' case.

[21] On the issue of fitness to return to work, Dr Beatty had addressed the matter in some detail with support from medical literature. It was of note that the pursuers had led no detailed evidence as to the nature of Mr Bruce's work. No award should be made, it was submitted, for past loss of support in relation to earnings.

Discussion and Decision

[22] I found the presentation and content of the evidence of each expert to be wholly absorbing. Notwithstanding that this case represents Dr Beatty's first instruction in a medical legal matter, I am more than content to regard her as an expert who was properly instructed in this case. It goes without saying that Professor Nee, standing his qualifications, experience and highly impressive *curriculum vitae*, was also properly instructed as an expert. I do not propose to rehearse here in detail the facts founded upon by the experts but instead simply record that the material areas of agreement which I have attempted to summarise above accurately reflect the evidential position of the experts. The only area of dispute between them, of course, concerned the outcome for Mr Bruce in terms of his survival.

[23] I have decided in this case to prefer the opinion of the defenders' expert, Dr Beatty on this single critical issue. My reasons for so doing can be summarised as follows. It was clear to me from the content and tenor of Professor Nee's responses to the agreed level of infusion, as set out in the Minute of Admission, that he appeared to have had in his own mind throughout his instruction a much quicker and more aggressive treatment regime than that agreed in this case. When pressed on the level of creatinine present, he was able to refer only

to a single patient, an elderly gentleman with gastrointestinal losses, who had presented with this level of acute kidney injury and gone on to survival. Dr Beatty herself had never in her career seen an acute kidney injury patient presenting with a creatinine figure of that level. Professor Nee accepted that on admission Mr Bruce's creatinine level would have been over 1000 and indeed close to 1441. In particular, when the pursuers' counsel asked him what difference the agreed infusion in the Minute would have made, his response was that "one would hope his clinical condition would have improved". In cross-examination, when asked whether the infusion proposed in the Minute would have been enough to change the outcome, to his credit Professor Nee expressed the view that this was very difficult to say. Ultimately his position became a rather more nuanced one to the effect that, as he put it, "We are talking 2 litres leading not to survival, but to widening of the window of opportunity to lead Mr Bruce to the intensive care unit, meaning that he survives beyond that unit and onto discharge". He agreed that he had a difficulty on the matter, but couched his position in the double negative format of indicating that he was "not content to say that it would have made no difference." This was not an expression of confident opinion but was rather, in my view, standing the content of his evidence and indeed his at times hesitant demeanour in giving it to the court, an opinion properly but in the end reluctantly expressed. He himself appeared to lack confidence in his own opinion, and on that basis, so did I.

[24] Dr Beatty, by contrast, grew in confidence on her opinion on survival as her evidence progressed, and ably defended her use of medical literature and of cross-checking with the APACHE II Scoring System under skilful cross-examination by counsel for the pursuers. The blood results approximately two hours after admission at 2321 hours showed a significant acidosis that simply could not have developed after Mr Bruce's admission to Ninewells. She supported her opinion by reference to the cumulative factors referred to by

her in the records, together with observations by staff on matters such as mottling of the skin, as mentioned above, and the agreed factual background of Mr Bruce's significant co-morbidities. There was then at admission a severe acidosis with acute kidney injury, and renal replacement therapy was indicated at the outset. Further, and in any event, it would have been extremely unlikely, in Dr Beatty's view, that it would have been possible to commence Mr Bruce on renal replacement therapy much before the acute deterioration which she records at paragraph 3.15 of her report as at 0040 hours. In addition, Professor Nee did not seem to be at all familiar with the current Scottish use of the APACHE II Scoring System and criticised it as, in terms, obsolete. Dr Beatty, in contrast, was highly familiar with its use in everyday practice in Scotland, and noted that it was the longest established of all scoring systems. In no sense was it being used as a predictor for individual patients but was, rather, a cross check tool which could be considered retrospectively. On all of these matters I have chosen to prefer the measured and evidence-based approach of Dr Beatty as one soundly rooted in current Scottish practice and more in keeping with the whole picture which emerged at the proof regarding Mr Bruce's true position on his admission to Ninewells on 12 August 2011.

[25] In so far as counsel for the pursuers criticised Dr Beatty's reference to medical literature, there may or may not be merit in these criticisms, but for present purposes I simply note that Dr Beatty did attempt to support her position not just with that literature but by reference to the APACHE II Scoring System, with which she was highly familiar. In contrast, Professor Nee offered no literature to support his position, but as he put it at the end of his examination in chief, to which I have already made reference, the best approach to a consideration of outcome for individual cases was the assessment and opinion of an experienced physician and intensivist. This line is perilously close to an *ipse dixit* approach,

which in my view cannot be a proper foundation for a supportable and reason-based clinical opinion. In so far as Dr Beatty offered methodology and literature to the court to support her reasoning, that is in my view all the more to her credit and, in the absence of anything else put forward by Professor Nee to gainsay that, I am content to regard it as being of some, albeit perhaps limited, value. The critical part of each expert's evidence in this case comprised the presentation and content of their respective evidence on outcome, and, as I have already indicated, Professor Nee's position in support of the pursuers contention on that matter was reluctantly delivered after what appeared to be considerable internal wrestling in the light of what he clearly considered to be the far too limited terms of the infusion set out in the Minute as the agreed non-negligent treatment in this case.

[26] I noted that Professor Nee's reliance on the five stage shock scale was not in the end materially relied on by the pursuers' counsel in her submissions. Further, on the matter of Professor Nee's opinion on the relevance of an individual's prior functional status, I prefer once more the evidence of Dr Beatty, which was in my view again more consistent with Mr Bruce's considerable co-morbidities, his recorded data at Ninewells on presentation and of course the important but only retrospectively known feature of the post-mortem examination report of Dr Sadler in respect of the narrowing of the stented blood vessel referred to in his discussion in that report on cause of death.

[27] On the matter of return to work, in the absence of the opinion of a nephrologist, as was properly observed by Professor Nee, I find the pursuers' contention *non probatum.*

[28] For these reasons I find that, notwithstanding the agreement of parties that there was indeed negligence on the part of the defenders' staff at Ninewells Hospital on 12 August 2011, the non-negligent infusion as set out and agreed between the parties in the Minute of Admission would have made no difference to the tragic outcome for Mr Bruce and of course

for his family. I am satisfied on the evidence led that this negligence accordingly did not cause the death of Mr Bruce, which was, instead, from the point in time of his admission to hospital an inevitable end-point of his medical journey there.

Disposal

[29] I accordingly repel the pleas in law for the pursuers and sustain the third plea-in-law for the defenders, pronounce decree of absolvitor in favour of the defenders and reserve all questions of expenses meantime. Finally, I should record that, had I found in favour of the pursuers on the question of causation, I would have awarded damages, other than making no award for past loss of support in relation to earnings, in the full terms agreed between counsel for the parties, whom I once more commend for their conduct and presentation of this litigation.