Case No: C92LS191

Neutral Citation Number: [2017] EWHC 1517 (QB)
IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION

The Combined Court Centre, Oxford Row, Leeds

Date: 12 July 2017

Before:

HIS HONOUR JUDGE GOSNELL
sitting as a Deputy Judge of the High Court

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Between:

Deborah Magill (Executor of Colin Stuart Magill deceased)	**Claimant**
- and –	
Panel Systems (DB Limited)	**Defendant**

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Mr Jeremy Roussak (instructed by Irwin Mitchell LLP) for the Claimant
Ms Jane Adams QC (instructed by DWF LLP) for the Defendant

Hearing dates: 12th -14th June 2017

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Judgment

His Honour Judge Gosnell:

1. This claim is brought by Deborah Magill both in her own right and as personal representative of the estate of her late husband Colin Magill. Mr Magill (the deceased) died on 18th July 2016 when he was sixty years of age. The deceased had been employed by the Defendant as a saw man between 1975 and 1978. During that time the Defendant accepts that it exposed the deceased to asbestos dust and fibres in breach of their duty of care. The deceased developed mesothelioma which was diagnosed in June 2015. The Defendant accepts it is liable for any damage which flows from the contraction of mesothelioma by the deceased but not otherwise. The deceased did not die directly from the mesothelioma but from a cardiac arrest. The Claimant claims that were it not for the mesothelioma the deceased would have undergone a coronary artery bypass graft (CABG) which would then have prevented his death from hypoxia following cardiac arrest. The Defendant does not accept this and the purpose of the trial was to resolve this dispute and other related issues.

2. **The agreed factual background**

Counsel for the Claimant prepared a guide to the medical terminology in this case which I have included as an appendix to this judgment and have adopted to prevent me having to include the various explanations of the medical terms herein. The deceased was diagnosed with a dilated cardiomyopathy (enlarged heart) in 1993 probably caused by alcoholism. He had suffered from Atrial Fibrillation since 1993 but a coronary angiogram in 1994 had shown normal coronary arteries. He then developed diabetes mellitus in 2007 (a condition involving prolonged high blood sugar) which was partially controlled with oral medication. The deceased had diagnostic echocardiograms in 2004, 2008 and 2014 which have been commented upon by the expert witnesses. The deceased noticed a deterioration in his condition in 2014 when he started to become more breathless. He underwent a coronary angiography on 8th April 2014 which showed coronary artery disease. This led to his physicians recommending that he undertake coronary artery bypass graft surgery (CABG) which was originally arranged for 9th March 2015. This operation was postponed due to a chest infection and relisted for April 2015. A chest x-ray had revealed a large pleural effusion in the right lung which had to be drained using a chest drain.

3. The investigation of the pleural problem caused the second planned operation to be postponed and on 5th May 2015 a thoracoscopic biopsy led to the deceased being given a diagnosis of mesothelioma. He then underwent five cycles of chemotherapy over the following year. On 17th July 2015, he was at home and complained of breathlessness. He went outside to get some air and collapsed shortly afterwards. An ambulance was called and arrived within seven minutes. He was in cardiac arrest characterised by ventricular fibrillation but was shocked back into sinus rhythm. He had to be shocked again in the ambulance on route to the hospital. His heart was stabilised and performed adequately for twenty-four hours. He had suffered severe anoxic brain injury however and treatment was withdrawn. He died on 18th July 2016. A post-mortem was performed and both experts comment on the result of the post mortem.

4. **The factual evidence**

There were only two factual witnesses, the Claimant and the deceased. The deceased had signed two witness statements which were clearly admissible dated 18th July 2015 and 8th October 2015. In the main they dealt with his employment history and his family life with the Claimant. He stated that although he was diagnosed with cardiomyopathy many years ago he took medication for that to ensure his heart beat was controlled and he had been quite well since, although he had to reduce his activity. He was looking forward to undergoing the CABG surgery which he was told would improve his symptoms substantially and by then they had moved to a new home. He related the distressing progress of his condition and treatment following the diagnosis of mesothelioma. He had hoped to do some DIY, painting and decorating in their new home after surgery but this never took place and his condition worsened over time due to the mesothelioma.

5. The Claimant made two witness statements dated 18th March 2016 and 21st September 2016. She dealt with the distressing progression of her husband's condition but I do not have to deal with this issue in the light of the parties' agreement as to the quantum of general damages. Before he suffered from the effects of mesothelioma the deceased was not a well man but he could still perform some tasks like mowing the lawn but he would do it more slowly than the average person. He could go for walks with his wife although this became more difficult after he was diagnosed with coronary artery disease. He also used to do some of the cooking. After he was diagnosed with mesothelioma however he was much worse and he found it very difficult to walk any distance without breathlessness. She related how he collapsed at home and how she had to take the difficult decision to withdraw treatment when she was advised he had suffered irreversible brain damage due to lack of oxygen.

6. The Claimant gave evidence at the trial and was cross-examined briefly and sympathetically by leading counsel for the Defendant. She was taken to two documents which the deceased had completed in 2012 and 2014 called "Limited capability for work questionnaire" in connection with a claim for state benefits . This form revealed a considerable level of disability at a time well before the diagnosis of mesothelioma was made. She candidly conceded that she was giving the deceased a great deal of personal care in even those days amounting to about 35 hours per week. She agreed that the deceased would not have been capable of decorating a room at home given the level of disability. After the diagnosis of mesothelioma, the level of care was roughly the same but she had to spend more time dealing with and organising his medication which was much more complex than previously. She was a patently truthful and impressive witness.

7. **The issues in the case**

As often happens during a trial, and with the assistance of both counsel, the issues in the case have narrowed. The remaining issues requiring the decision of the court are as follows:

a) What was the cause of the cardiac arrest which precipitated the death of the deceased;

b) Whether the cardiac arrest would have taken place when it did if the deceased had undergone a CABG operation in March / April 2015 as planned;

c) What would have been the deceased's life expectancy had he not developed mesothelioma;

d) If the Claimant's claim is successful what the quantum of the awards for loss of love and affection and dependency on the deceased's services should be.

It is logical to deal with these issues in order.

8. **What was the cause of the cardiac arrest?**

This is an issue which can only be determined by analysis of the relevant expert evidence. I had the benefit of four expert reports in this case. Mr Patel, an actuary prepared a report which is relevant only to the issue of life expectancy. Dr Robin Rudd a chest physician prepared several reports and gave evidence at the trial. He deferred on most issues to the respective cardiologists, Dr Witte for the Claimant and Professor Channer for the Defendant.

9. Dr Robin Rudd was instructed initially to deal with the diagnosis of the deceased's condition of mesothelioma and his life expectancy but for the contraction of the disease. In relation to the issue concerning the cause of the deceased's cardiac arrest Dr Rudd agreed with Dr Witte that the cause of death was an arrhythmia leading to the loss of cardiac output and thereby perfusion to the brain. Dr Rudd doubted whether the deceased had acute left ventricular failure as opined by Professor Channer because this would have caused pulmonary oedema which was not described by the treating clinicians, nor found on autopsy. The deceased was not given intravenous diuretics which would have been standard treatment for a patient present with fluid on the lungs. Dr Rudd was also of the view that the anaemia caused by the reduced output from the right lung would have reduced the effectiveness of resuscitation after the cardia arrest. In most other respects, he deferred to the opinion of the cardiologists on this issue. It seems to me however that he can validly comment about the treatment for and effects of pulmonary oedema as a chest physician.

10. Dr Klaus Witte was called by the Claimant to give expert evidence on this issue. He is a consultant cardiologist at Leeds General Infirmary. He prepared four reports and contributed to the expert's joint statement. His overall view was that the deceased's cardiac condition had been stable and mild for the previous 10 years or more. On the 17th July 2016, the deceased suffered a cardiac arrest in ventricular fibrillation resulting from an acute myocardial infarction. Although the pathologist at post mortem diagnosed left ventricular failure Dr Witte disagrees because of the lack of pulmonary oedema found either by treating physicians or at post mortem. He felt that the symptoms of breathlessness reported by the Claimant shortly before the deceased's collapse could be evidence of a myocardial infarction in a patient with diabetes mellitus who often do not present with classic chest pain. An acute (permanent or transient) occlusion of a distal coronary artery would not be seen on autopsy, and with death occurring so abruptly, there would be no evidence of tissue damage. He felt the most common cause of ventricular fibrillation is cardiac

ischaemia and the raised troponin levels suggest that ischaemia or infarction was the origin of the arrhythmia and the cause of the ultimate death due to cerebral hypoxia.

11. His initial view was not only that coronary artery bypass surgery (which was cancelled because of mesothelioma) would have prevented this acute coronary event and thereby the arrhythmia that led to his death but also the mesothelioma increased the chances that he would suffer hypoxic brain damage during the cardiac arrest.

12. Dr Witte was asked to comment on Professor Channer's report in particular his conclusion that the deceased had died of acute left ventricular failure. Dr Witte did not accept this as a diagnosis in that left ventricular failure, whether acute or chronic is always the consequence of a change. Professor Channer had not identified what change had caused the suggested left ventricular failure he opined.

13. Professor Channer also prepared four reports in this case and contributed to the experts' joint statement. He is a consultant cardiologist and physician formerly of the Royal Hallamshire Hospital. He was asked for his opinion on the cause of the cardiac arrest and he opined that from the history as recorded in the Claimant's statement of a sudden onset of breathlessness this almost certainly represented acute left ventricular failure. Although the treating physician had diagnosed a myocardial infarction Professor Channer felt this was unlikely because there was no preceding history of chest pain, and no evidence of acute thrombotic occlusion of a coronary artery found at post mortem. There was no evidence of acute changes within the myocardium at post mortem to suggest myocardial infarction and that the raised troponin could have been caused by the cardiac arrest. His overall opinion was that the cardiac arrest occurred because of acute left ventricular failure which occurred in association with the natural history of dilated cardiomyopathy in this case.

14. Professor Channer also pointed out the dismal survival rate from out of hospital cardiac arrests. The main reason why the survival rate is so poor is the time before defibrillation is done in cases of ventricular fibrillation. In the current case the earliest that defibrillation could have been done was at least seven minutes after the cardiac arrest. In Professor Channer's view hypoxic brain injury was inevitable given the cessation of circulation during that period.

15. In his fourth report Professor Channer replied to some Part 35 questions which had been put by the Claimant's solicitors. He was asked to explain why there would be a sudden deterioration in relation to the deceased's cardiomyopathy which had been diagnosed over 20 years previously and remained stable. His reply was that heart failure is relentlessly progressive. All individuals with impairment of cardiac contractile function will develop heart failure over time. This is called the "natural history" of the condition. The time to deterioration varies with the underlying condition and the progress of the aetiological factors. Professor Channer felt there was evidence of deterioration of the deceased's heart condition over time. He was diagnosed with severe obstructive coronary artery disease which can itself cause heart muscle damage from ischaemia. He relied on the enlargement of the heart found at post-mortem together with myocardial fibrosis which is the scar tissue left after muscle death, a sign of heart muscle cell deterioration. He relied on the fact that the heart surgeon treating the deceased described him as a "high risk" patient for surgery.

16. The expert's joint statement runs to some sixteen pages and I can only give a brief summary of the expert's views on the most relevant issues. Professor Channer was of the view that the deceased's heart failure was at least "moderate" prior to his death whereas Dr Witte described it as "mild". Professor Channer felt that his heart condition was deteriorating whereas Dr Witte felt it was stable. To the extent that his coronary artery disease was causing damage to his heart muscle Dr Witte felt this would be improved by revascularisation following CABG surgery. When surgery was being considered Professor Channer agreed that the deceased had a high peri-operative risk as his surgeon recorded whereas Dr Witte felt his risk was merely higher than average in that the pleural effusion which had been noted at that stage was actually due to the mesothelioma and not the heart condition (as the surgeon had understandably assumed). The chances of the deceased not surviving the surgery would have been 1.34% according to Dr Witte and more than 2% according to Professor Channer.

17. Professor Channer was asked to address the diagnosis of left ventricular failure which he did having seen the hospital records relating to the final admission for the first time. He opined that the cause of the acute left ventricular failure was an arrhythmia, probably ventricular tachycardia, which is supported by the recurrent ventricular tachycardia he had after initial successful resuscitation. He felt there was radiological evidence of pulmonary oedema on 17th July 2016 but treatment on the Intensive Therapy Unit (if only positive pressure artificial ventilation) would clear the lungs of oedema from left ventricular failure. He only had the necessary IV fluids to maintain insensible losses.

18. Dr Witte agreed that the cause of death was an arrhythmia leading to a loss of cardiac output but disagreed there was a left ventricular failure. Pulmonary oedema was not recorded by the treating clinicians nor on autopsy. He was not given intravenous diuretics which would have been standard treatment for pulmonary oedema and was given a litre of fluids over time, again treatment inconsistent with pulmonary oedema.

19. Professor Channer felt there was an acute arrhythmic event of ventricular tachycardia which then precipitated ventricular fibrillation. Dr Witte felt it was more likely that there was a myocardial ischaemic event which triggered ventricular fibrillation. Both agreed that the immediate cause of death was hypoxic brain damage.

20. Dr Witte conceded in the joint statement that a reduced haemoglobin level and the pro-thrombotic effect of both mesothelioma and chemotherapy did not play a material part in the development of the arrhythmic event which led to the death of the deceased. He had previously raised these issues in his reports as possible contributory factors.

21. When directly asked, what was the cause of the deceased's cardiac arrest Professor Channer replied "scar related VT (ventricular tachycardia) causing acute LVF (left ventricular failure) decompensation and degeneration to VF (ventricular fibrillation) and OOH (out of hospital) cardiac arrest". Dr Witte (who was addressing this explanation for the first time) considered that there was no evidence of a focal myocardial scar on the MRI scan that would increase the chance of an untriggered short circuit and it was far more likely that an ischaemic event triggered ventricular fibrillation.

22. Both experts gave evidence at the trial for some time and were robustly but fairly cross-examined by counsel for both parties. They are both experienced and respected in their field and eminently qualified to give expert evidence on this case. Neither expert made significant concessions in relation to the core issues in this case and were not shaken from their views. Dr Witte was asked why in the joint statement he had conceded that the reduced haemoglobin levels and the pro-thrombotic effects of mesothelioma and chemotherapy did not play a material part in the cause of the cardiac arrest. He replied "we all agreed that there were more major issues". I felt this was a sensible concession and an example of the concept of the joint meeting of experts working well to narrow the issues. These issues might have had a theoretical effect but it was not likely to be material and so it was right to exclude them. He had also argued in his report that the respiratory impairment caused by mesothelioma would have reduced the effectiveness of resuscitation and increased the risk and extent of brain damage. When it was put to him in cross-examination that after seven minutes' delay in an out of hospital cardiac arrest case the chance of resuscitation without brain damage was slim he accepted that on balance of probabilities the reduced respiratory effect would have made no difference. Again, I felt this was a sensible and reasonable concession to make and reflected well on Dr Witte's independence as an expert witness. Professor Channer was a more combative witness who seemed determined to put across his point of view forcibly. On the morning of the third day of trial he asked permission to make three extra points which had occurred to him since giving his evidence the previous day. It may have been the case that he wanted to make sure that his arguments had the necessary clarity to them but it did rather give the impression of a witness who wished to add weight to his client's cause.

23. **Analysis**

There is a significant issue between the experts as to the status of the deceased's underlying heart condition excluding his coronary artery disease. There are some echocardiograms in the deceased records which provide some assistance. On 13th December 2014, his LVEDD (left ventricular end diastolic diameter) was measured at 6.7 cm when the normal range is 3.5- 5.6 cm in men. This is a measurement of the size of the left ventricle at its greater diameter and shows an enlarged heart. On the same date his Left Ventricular Ejection Fraction (LVEF) was measured at 35-45%. This represents the fraction of the blood ejected from the ventricle on each beat. The normal range is evidently 55-70%. According to Dr Witte however 45% may well be considered on the borderline of normal in clinical practice. By 15th January 2008 the LVEDD was 6.4cm and the LVEF was 45%. On 16th July 2014, the LVEDD was 6.3 cm and the LVEF was not measured. The physiologist reported however that the left ventricle function was "at least moderately impaired" and the right ventricle was mildly dilated with impaired function of free wall motion. This should be compared with the result of an angiogram performed on 8th April 2014 which reported overall "reasonable function" in the left ventricle. There was also an MRI scan in July 2014 which, according to Dr Witte showed an LVEF of 48% and no area of distinct scar. Professor Channer felt the scan was barely of diagnostic quality.

24. Professor Channer opines that these readings show objective evidence of both deterioration in the deceased's cardiac status and of progressive ischaemic heart disease. Dr Witte opines that they show stable heart failure and left ventricular

function as there is no evidence of deterioration of renal function or change in diuretic requirement which would be expected if there was a marked deterioration in heart function. There seems to be no objective measured change in either the LVEDD or LVEF from 2004 to 2014. Whilst the reference to the mild dilation of the right ventricle and impaired function of free wall motion is a new symptom in 2014 this could be explained by ischaemia due to the coronary artery disease according to Dr Witte. Professor Channer disagreed with this due to the site of the damage to the free wall but this is not an issue I have the knowledge or capacity to resolve. Standing back from the technical issues' the measurements which these various scans reveal do not, in my view, appear to support a substantial worsening of the deceased's cardiac condition (other than due to the new coronary heart disease symptoms).

25. Professor Channer relies heavily on the conclusion expressed by Mr Javangula the consultant thoracic surgeon treating the deceased in his letter of 14th January 2015 (page 1571 of the trial bundle) where he stated "he has poor LV and he is a high-risk patient for surgery because of the previous MI's and very poor right ventricle and left ventricle with a diagnosis of? cardiomyopathy…". Just leaving aside the rather poorly expressed letter I agree with Dr Witte by this point the patient's clinical condition would have been deteriorating because of the undiagnosed mesothelioma and this would have been the cause of the pleural effusion reported on the cardiac MRI. He also appears to have concluded that the deceased had suffered two previous myocardial infarctions which was agreed during the trial to be incorrect. I accept the deceased's mortality risk was perhaps higher than average due to his diabetic status and heart condition but otherwise not indicative of a worsening cardiac status.

26. If I accept that there was no real objective evidence of a worsening in the deceased cardiac condition, then Dr Witte would say that a myocardial ischaemic event against a background of known coronary artery disease sufficient to justify CABG surgery is a much more likely explanation for the episode of ventricular fibrillation which caused the cardiac arrest than a sudden left ventricular failure triggered by a scar-related ventricular tachycardia leading to ventricular fibrillation. This was Professor Channer's alternative explanation at trial for the cause of the cardiac arrest. It had first seen the light of day in the joint statement. Leading counsel for the Defendant submitted that in effect Professor Channer had always been consistent in his explanation that the cardiac failure was due to the natural history of the deceased's heart disease. He explained this in his fourth report at p 568 of the trial bundle:

> *"Heart failure is relentlessly progressive. All individuals with impairment of contractile function will develop over heart failure over time. This is called the "natural history" of the condition"*

This suggests to me a gradual deterioration of the function of the patient's heart over time with consequent increase in symptoms until heart failure progressively occurs. As Dr Witte confirmed in evidence, he would expect to see a gradual increase in symptoms such as ankle swelling and increased breathlessness (not due to ischaemia from coronary artery disease).

27. At trial Professor Channer expanded on the brief explanation he gave in the joint statement by explaining how in his view an ectopic beat triggered ventricular

tachycardia. The heart must pump from the bottom upwards and an electrical impulse triggers the contraction of the heart muscles. The heart has its own pacemaker which is a modified cell which sends electrical impulses and causes the atrium to contact. There is a delay at the atrioventricular node to allow time for blood to go from atrium to the ventricle. There is then an impulse to the bottom of the heart which contracts from the bottom in a corkscrew fashion. The cells wait for the next electric signal to contract again. When they are electrically silent they are building up their battery and are exquisitely sensitive. If there is an extra beat the cells themselves spontaneously contract and can send the message to the next cell. It is called ventricular ectopy, which is this unexpected beat that can sometimes occur. If there is scar tissue where there is a dead cell the impulse must go around the scar to the next cell. When there is scar tissue the delay caused by the travelling of the impulse around the scar may cause it to hit the cell just when it is ready to depolarise. This can occur randomly, but it can trigger an unfortunate reaction such as ventricular tachycardia.

28. If this summary of the explanation is not clear it is because I was making a note of it for the first time at the trial as it had not actually figured in any of the expert evidence submitted in writing. Whilst I appreciate that this type of ectopic beat can only occur in the presence of scar tissue there was a debate between Professor Channer and counsel for the Claimant whether the scarring was caused by ischaemia. Eventually it appeared that Professor Channer accepted that ischaemia could be the cause of the fibrosis but he opined that ventricular tachycardia would not occur unless there was pre-existing cardiac muscle disease. This explanation which is complex and difficult for a layman to follow does not seem to me to be the same as the "natural history" of heart failure as previously expressed. Dr Witte considers this an unlikely explanation in any event because there was no evidence of a focal myocardial scar on the MRI that would increase the chance of an untriggered short circuit. He also appeared to opine that it would require an actual scar (dead tissue) to produce this reaction not merely fibrosis.

29. Another issue which separates the parties is whether there was pulmonary oedema when the deceased was admitted to hospital after his cardiac collapse. Both experts agreed that if there had been left ventricular failure then there would have been pulmonary oedema which would have been noticeable in the healthy left lung. Professor Channer is of the view that the deceased's complaints of breathlessness before his collapse are classic for pulmonary oedema and the chest X Ray taken on 17th July 2016 (page 2222 of the trial bundle) reveals patchy airspace shadowing at the left midzone (of the lung). Dr Witte was dismissive of the diagnostic quality of X rays particularly a supine view of an unwell patient. Dr Knights the consultant pathologist who conducted the post-mortem concluded that there was acute ventricular failure but not because of a finding pulmonary oedema in the healthy left lung which was normal (page 632 of the trial bundle). He perhaps reached his view because he found no evidence of coronary artery thrombosis or myocardial infarction. Dr Witte disagrees with this diagnosis and points out that the hospital medical team were not treating the deceased for pulmonary oedema as they were continuing to give him fluids (100 ml per hour) and did not prescribe diuretics which would be the normal treatment for pulmonary oedema. Dr Atkinson the consultant anaesthetist responsible for the deceased's treatment in the Intensive Care Unit opined that the cause of death was myocardial infarction caused by ischaemic heart disease the management of which condition was influenced by the mesothelioma as a contributory factor (page 638 of the trial bundle). He makes no mention of pulmonary oedema even though he presumably

had access to the chest X Ray carried out on 17th July 2016. Professor Channer feels that the positive pressure ventilation will have removed pulmonary oedema so that it is not detectable at post mortem. Dr Witte felt that whist the ventilation would have dilated pulmonary oedema it would not have removed it.

30. Dr Witte points out that whilst the deceased was defibrillated both at the scene and in the ambulance after application of amiodarone he settled into sinus rhythm and his cardiac condition was stable thereafter. It was only after extensive anoxic brain damage was discovered that treatment was withdrawn. This is not consistent with left ventricular failure. Professor Channer points out that the deceased kept returning to ventricular tachycardia after being shocked until amiodarone was administered adding weight to his theory of scar related tachycardia but I conclude that the relatively stable cardiac condition shown by the deceased for the 24 hours after his admission is not consistent with left ventricular failure.

31. I therefore conclude that on balance the deceased did not suffer from pulmonary oedema and I am not satisfied that there was left ventricular failure. The evidence that the deceased's heart condition (other than coronary artery disease) was deteriorating is not strong objectively. Whilst I have carefully considered the two competing theories, on balance I feel the theory put forward by Dr Witte is more likely. There was evidence of coronary artery disease which was worsening and Dr Witte has explained why evidence of a thrombotic event may not have been evident on post-mortem. The relative recovery of the deceased from a cardiac point of view in hospital is consistent with a transient thrombotic event creating a myocardial infarction which was successfully treated by defibrillation and medication. Professor Channer's theory as developed at trial is in my view overly complex and by continuing to embrace the notion of left ventricular failure in the absence of convincing evidence of pulmonary oedema less convincing than Dr Witte's explanation. I therefore decide the first issue that the cause of the deceased's cardiac arrest was an acute ischaemic event triggering ventricular fibrillation as proposed by Dr Witte.

32. **If the deceased had undergone CABG surgery in March / April 2015 would he have avoided the cardiac arrest in July 2016?**

The parties have agreed that, absent the mesothelioma it is probable that the deceased would have undergone CABG surgery in March/ April 2015 which would have substantially improved the condition of the vascular flow to the heart. Leading Counsel for the Defendant also accepted that if I preferred the explanation put forward by Dr Witte to that of Professor Channer then the mechanism of causation of the cardiac event was one which, potentially, may have been avoided by CABG surgery. The Defendant's case is that CABG surgery requires a period of time before it has a positive effect on the condition of the heart and that period of time had not been reached by the time the deceased had his cardiac arrest on 17th July 2016.

33. Professor Channer opined that there is a time lag for the benefits of coronary artery bypass graft surgery of about three years and accordingly the deceased would not have reached the stage of experiencing the benefit from surgery before he died. Even if the death had occurred in association with coronary artery disease the scientific evidence is that coronary artery bypass does not reduce the risk of death initially. There is a lag time of nearly three years before the survival curves separate indicating a benefit from

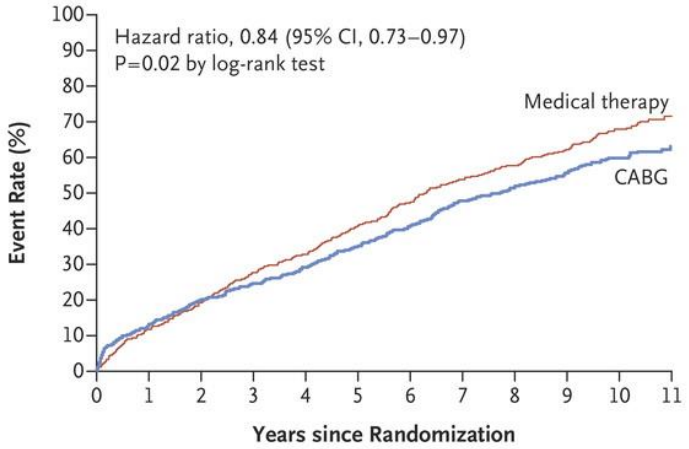
surgery[1]. Because the deceased died less than two years after he would have undergone bypass surgery the effect of mesothelioma made no difference to his prospects of avoiding the fatal cardiac arrest which he experienced in July 2016.

34. Dr Witte accepted that all treatments, especially serious surgical interventions are associated with a lag time to benefit since each is associated with side effects or complications which are more common in those patients with more severe disease. The Kaplan-Meier survival curve relied on by Professor Channer described the survival rate of a population with a worse underlying heart condition than the deceased and with a range of co-morbidities not shared by the deceased. Dr Witte felt that the deceased would have benefitted much earlier than three years from the coronary bypass surgery.

35. Professor Channer's opinion is based upon a study entitled "Coronary Artery Bypass Surgery in Patients with Ischaemic Cardiomyopathy" first published in the New England Journal of Medicine in April 2016. It sought to assess the survival benefit of a strategy of CABG added to guideline-directed medical therapy as compared with medical therapy alone, in patients with coronary artery disease, heart failure, and severe ventricular systolic dysfunction. The conclusion of the study was that the rates of death were significantly lower over ten years among patients who underwent CABG in addition to receiving therapy than those who received medical therapy alone. His conclusions can perhaps best be understood by considering certain graphs which were included as part of the study to illustrate the results:

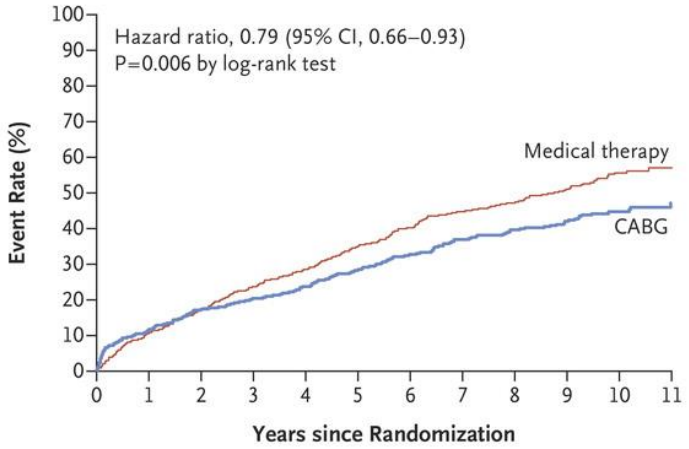
[1] Velazquez EJ et al NEJM 2016 ;374;1511-20

A Death from Any Cause (Primary Outcome)



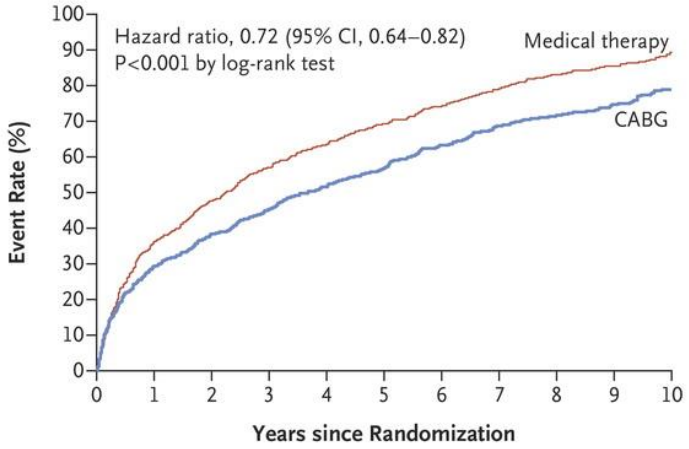
No. at Risk												
Medical therapy	602	532	487	435	404	357	315	274	248	164	82	37
CABG	610	532	487	460	432	392	356	312	286	205	103	42

B Death from Cardiovascular Causes



No. at Risk												
Medical therapy	602	532	487	435	404	357	315	274	248	164	82	37
CABG	610	532	487	460	432	392	356	312	286	205	103	42

C Death from Any Cause or Cardiovascular Hospitalization



No. at Risk											
Medical therapy	602	385	314	259	219	185	152	123	98	57	19
CABG	610	431	376	334	293	259	218	184	166	106	43

36. Professor Channer relied on the first of the graphs reproduced above to show that whilst CABG improved mortality rates over the lifetime of the study (58.9% after bypass surgery compared with 66.1% in those treated medically only) the Kaplan Meier survival curves show that the improvement in survival occurred only after nearly three years following randomisation.

37. Dr Witte had several principled objections to this analysis, the first one being that the patients who were eligible for this trial were significantly unhealthier than the deceased. Patients were only eligible for this trial if they had coronary artery disease that was amenable to CABG and an ejection fraction of 35% or lower. Dr Witte considers that the deceased who had a stable ejection fraction of 45% could not have qualified to be part of the trial. In addition, there were patients who were part of the trial who had co-morbidities not shared by the deceased which would have been poor prognostic indicators of mortality. The characteristics of the patients who were the subject of the test are revealed in the following table:

Table 1. Characteristics of the Patients at Baseline.*

Characteristic	CABG Group (N=610)	Medical-Therapy Group (N=602)
Median age (IQR) — yr	60 (54–68)	59 (53–67)
Female sex — no. (%)	73 (12)	75 (12)
Race or ethnic group — no. (%)†		
Hispanic, Latino, or nonwhite	221 (36)	200 (33)
White	389 (64)	402 (67)
Median body-mass index (IQR)‡	27 (24–30)	27 (24–30)
Medical history — no. (%)		
Previous myocardial infarction	462 (76)	472 (78)
Hyperlipidemia	360 (59)	370 (62)§
Hypertension	358 (59)	370 (61)
Diabetes	240 (39)	238 (40)
Previous stroke	51 (8)	41 (7)
Chronic renal insufficiency	49 (8)	45 (7)
Previous percutaneous coronary intervention	82 (13)	74 (12)
Previous CABG	22 (4)	14 (2)
Current smoker — no. (%)	130 (21)	122 (20)
CCS angina class — no. (%)¶		
No angina	217 (36)	225 (37)
I	96 (16)	91 (15)
II	265 (43)	260 (43)
III	25 (4)	23 (4)
IV	7 (1)	3 (<1)
NYHA heart failure class — no. (%)¶		
I	65 (11)	74 (12)
II	319 (52)	307 (51)
III	207 (34)	205 (34)
IV	19 (3)	16 (3)
Median systolic blood pressure (IQR) — mm Hg	120 (110–130)	120 (110–130)
Median pulse rate (IQR) — beats/min	74 (66–82)	72 (65–80)
Median 6-min walk distance (IQR) — ft‖	1145 (863–1320)	1115 (840–1345)

* There were no significant differences in baseline characteristics between the treatment groups. CABG denotes coronary-artery bypass grafting, and IQR interquartile range.
† Race and ethnic group were self-reported.
‡ The body-mass index is the weight in kilograms divided by the square of the height in meters.
§ Data on hyperlipidemia were missing for 1 patient.
¶ The Canadian Cardiovascular Society (CCS) angina classes range from I to IV, with higher classes indicating more disabling pain due to angina. New York Heart Association (NYHA) heart failure classes range from I to IV, with higher values indicating greater disability.
‖ To convert the values for the 6-minute walk distance to meters, multiply by 0.305.

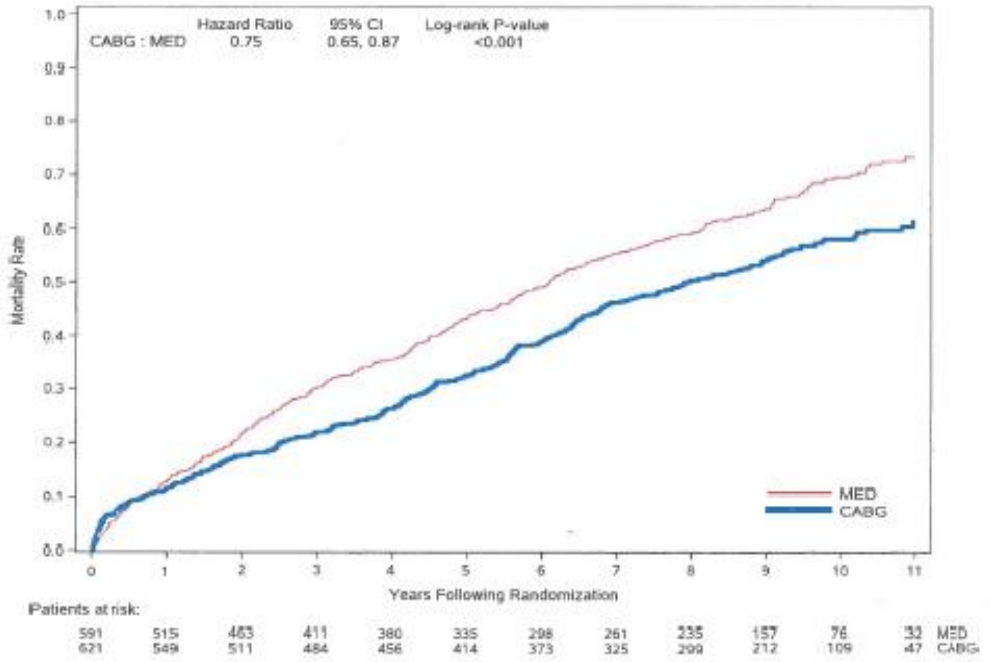
38. It can be seen from this table that there were several patients with diabetes (as did the deceased) but other complicating factors such as previous myocardial infarctions,

hypertension, strokes, renal problems and current smokers (not shared by the deceased). The most significant distinguishing factor according to Dr Witte is that these were patients effectively in heart failure whereas the deceased's condition was virtually stable (save for the coronary disease) based upon his EF of 45% and theirs of 35% or less.

39. It was also suggested that the graph which Professor Channer utilised was not the most accurate or appropriate from those published in the study. He used the primary outcome graph whereas the "as treated analysis" would produce a more accurate view of mortality rates because this analysis shows the results in accordance with the treatment they received, not determined by the sample group they were originally allocated to:

Supplementary Appendix Page 10

Figure S1. Kaplan-Meier rates of death from any cause for CABG vs. MED (As-treated analysis)



It can be seen from this graph that the change in the mortality rates starts to occur shortly after one year rather than after two and a half years in the primary outcome group.

40. Another point made by counsel for the Claimant and endorsed by Dr Witte is that the time lag before the benefits of surgery are seen is caused by the increased mortality involved in surgery itself compared with medicine alone. Even the graph used by Professor Channer shows the very early increased mortality and the fact that once the mortality surrounding the original surgery is removed the death rate of the CABG

group is consistently lower for the entire 10 years of the study, than of the medically treated group. It was also argued that the second of the graphs shown in the group above "death from cardiovascular cause" is the more appropriate graph to use when looking at whether CABG helps reduce the prospects of future cardiovascular failure. In this graph if the initial peak caused by those patients who died within 30 days of surgery is removed (presumably as result of complications surrounding surgery) the angle of both the CABG group and medical therapy group is relatively linear with the mortality rate in the CABG group being consistently lower. Counsel for the claimant illustrated this point by producing a graph excluding those patients who died within 30 days of surgery based otherwise on the Velazquez study:

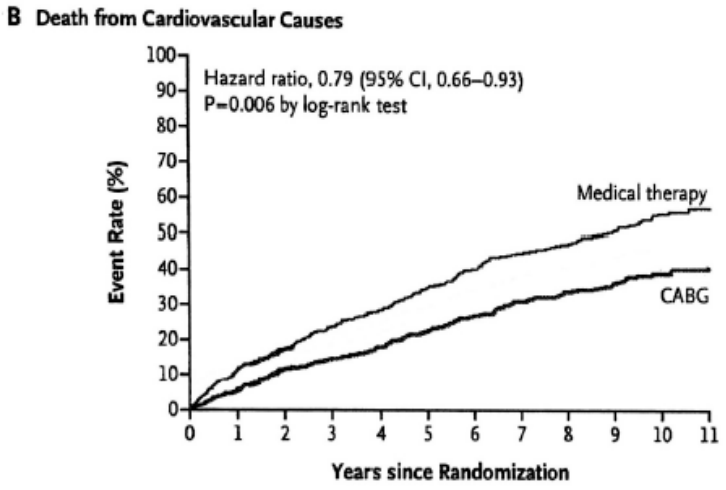


Figure B from Velazquez et al, NEJM, after removal of 30-day CABG mortality

It is clear from this amended graph that the benefits of surgery are obvious almost immediately (i.e. after the 30-day period post operation) and at 15 months post-surgery (which the deceased would have been in July 2016) the mortality rate for CABG patients is roughly half that of those receiving medical therapy alone.

41. Professor Channer was not able to express any particular medical reason why it would take three years to see any benefit from CABG, for example that it would take that long for ischaemic heart tissue to re-vascularise. Dr Witte pointed out that heart surgeons find the operation very satisfying because the moment the clamp is removed it is possible to see the blood flowing freely into the heart whereas previously it had enjoyed interrupted flow due to stenosis of the vessels or arteries. Logically, once the obstruction is removed the heart will immediate benefit from an uninterrupted supply of blood which, if properly oxygenated, should prevent any further damage from anaemia. I was told that previously anaemic cardiac muscle may possibly recover if is re-vascularised provided the cells are not already dead. Both experts confirmed that a CABG operation tends to have an immediate benefit for patients in the removal of angina pectoris pain. These arguments were put to Professor Channer who merely pointed out that there was no improvement in the heart function at rest, which may be relevant to the removal of angina pectoris pain (which would only occur on exertion) but seems to ignore the benefit both of increased oxygenated blood supply to the heart and the re-vascularisation of ischaemic tissue.

42. The graph prepared by counsel for the Claimant was shown to Professor Channer but he said it was scientifically inappropriate to manipulate the figures in this way. The authors of the report may agree with that but I accept that the less than 10% of the 600 patients in the CABG group who died within 30 days may well be distorting the position if the court wishes to look at the graph to see if there would be any benefit to this particular deceased if he had been given the opportunity to have CABG surgery in early 2015. According to Dr Witte his chances of not surviving the operation were only 2.4 % and Professor Channer accepted that a bare statistical analysis would be no more than 4%. As he was overwhelmingly likely to survive the surgery in my view it is permissible to look at those patients who survived for longer than 30 days to establish whether his chances of survival would have increased if he had undergone CABG surgery 15 months before his death. The answer appears to be that his chances of survival would have been twice as good as without the surgery.

43. Even ignoring the statistical analysis a logical examination of what the surgery is intended to achieve leads to the conclusion that there are very likely to be early and significant benefits from CABG surgery, a proposition that Professor Channer did not seem able to accept. On balance, I prefer Dr Witte's view that the deceased would have benefitted from surgery and the prospects of his suffering a cardiac arrest due to an acute ischaemic event would have been significantly reduced.

44. I therefore accept that there was a doubling of the risk of death due to the absence of CABG surgery caused by the deceased contraction of mesothelioma. Both counsel agreed that if I made this finding, I would inevitably find that the Defendant had caused or contributed to an indivisible injury, namely the arrhythmia, which caused the death of the deceased. This means the Defendant is liable in law for the death of the deceased and any consequences which flowed from that event even though there may have been other competing causes.

45. **What was the deceased's life expectancy had he not developed mesothelioma?**

It perhaps not unusual to have disputes about life expectancy in disease claims but it is unusual to have a debate about the appropriate methodology to be used. Counsel for the claimant points out that the deceased was born on 8th July 1956 and his life expectancy would be 25.06 years according to Table 1 of the Ogden Tables using 0% discount rate. Using 2014 ONS data the figure would be 25.5 years which is the most accurate current statistical information

46. In his report dated December 2015 Dr Witte adopts what might be termed as the conventional approach in personal injury litigation when there are co-morbidities which might mean that it would be appropriate to deviate from the life expectancy of the population. He reduces the deceased's expectation of life by two years because of his coronary artery disease, a further two years because of his diabetes and in his 2016 report he agreed to deduct a further two years to reflect the mild left ventricular failure. This would make the deceased's life expectancy 19.5 years. Professor Channer does not criticise these individual discounts (although he may well have chosen to do so) but would prefer to approach the problem from a completely different perspective.

47. Professor Channer is critical of the method of discounting from the average population as used by Dr Witte which he describes as a retrospective assessment. It

fails to take into account geographical and differences due to social class; it fails to consider the increased risk of cumulative causes; it does not distinguish smokers from non-smokers and does not allow for the change of circumstances (and therefore risk) over time. Professor Channer espouses a prospective assessment in which the risk per year of a fatal event is calculated and the time taken to reach a 51% chance of the event taking place is taken as the average survival. Many epidemiological studies have calculated the risk of death in association with individual risk factors and when they are combined. It is therefore possible to estimate the annual risk of a fatal and non-fatal event by multiplying the individualised risk against the background risk in the population. It is also possible to estimate survival from long-term follow up of large cohorts of patients with a particular disease process.

48. In the case of the deceased Professor Channer says there are data on survival after triple bypass surgery in association with impaired left ventricular function, and in the presence of diabetes and atrial fibrillation and so it is possible to predict average survival from those data sets. The data from coronary artery bypass surgery suggest the mortality rate is 4% per annum [2]. Patients with diabetes do better with CABG than without but still have double the mortality of those without diabetes[3]. Generally, the lower the ejection fraction measured on echocardiography the higher the annual mortality [4]. He also relied on the Velazquez study that I have referred to earlier which showed a mortality at 6 years of follow up after CABG of over 40%. On this basis Professor Channer calculates that if the deceased had not developed mesothelioma when he did he would have survived for about eight years after bypass surgery in 2015.

49. I had some assistance on this issue in the form of an expert report from Mr Chinu Patel who is a consulting actuary and a member of the Ogden Working Party. Mr Patel explained the general approach of the UK courts in dealing with impairments to life expectancy. He also explains how the Ogden Tables work by providing multipliers based on the mortality experience of the general UK population as a whole. These are not in dispute in this case and well understood by both counsel. If a Claimant is atypical and the court decides that he has a reduced life expectancy and this is expressed as a reduction in normal life expectancy, then the adjustments to the standard multipliers are relatively easy and described in the notes to the Ogden Tables. He describes Professor Channer's approach as an absolute assessment in that it is not relative to that of a reference population. He was unable to find anything in the Ogden Tables to enable a reference point to connect to Professor Channer's opinion to the underlying base mortality used in the Ogden Tables. In essence, the methodology used by Professor Channer cannot be combined with the methodology in the Ogden Tables although Mr Patel accepts that the multiplier could be arrived at by a different method outside the Tables. Whilst I accept this is a potential difficulty if I accept Professor Channer's views it is not an insurmountable problem and this alone would not persuade me to reject his views.

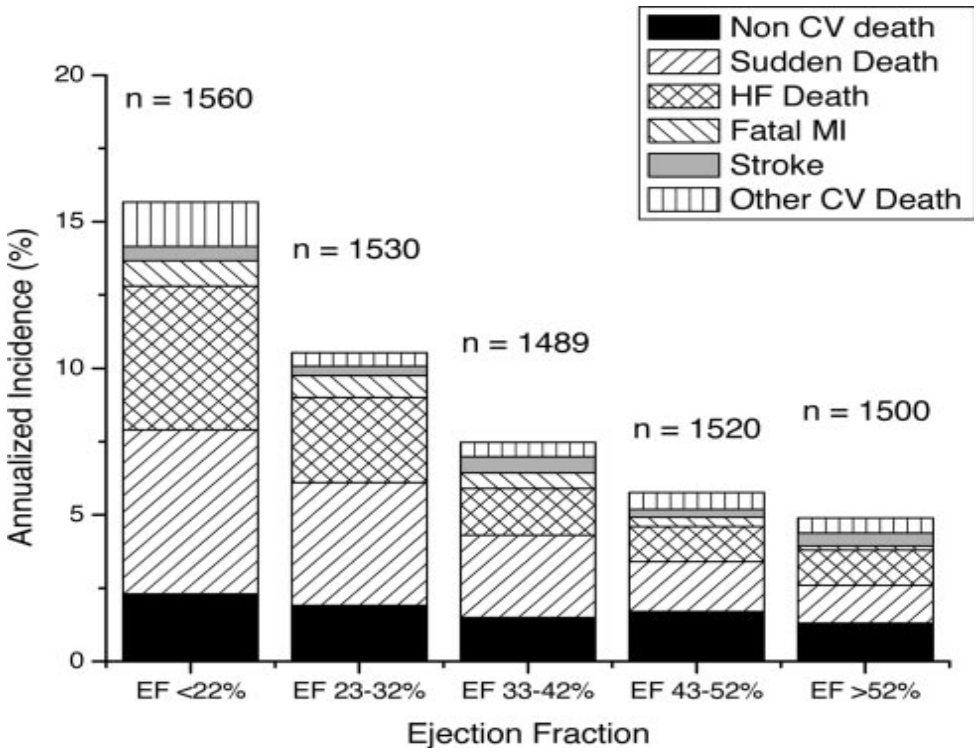
50. I also had some assistance on this issue from Dr Robin Rudd the consultant chest physician. He accepted that on the issue of life expectancy he had deferred to Dr Witte in relation to the cardiac components of the assessment. He accepted that he had

[2] Weintraub WS et al NEJM 2012 ,316:1467-76
[3] Hlatky MA et al Lancet 2009 373; 1190-97
[4] Solomon SC et al Circulation 2005:112;3738-44

prospectively assessed the deceased actual life expectancy as result of the contraction of mesothelioma at between 3 – 9 months. He said that when life expectancy is very short the best way to approach it is by using the median survival period for patients with that condition coupled with any particular factors that might affect the individual patient. He accepted that Professor Channer's method was a legitimate way to approach life expectancy for someone who had the characteristics of the study which was being used to fix the median. His view was that where there were multiple co-morbidities it was more difficult to find a study which reasonably matched the patient under consideration and it was therefore more sensible to use the retrospective method and to make deductions from the normal life expectancy to reflect each factor. I gave him an example of dealing with a mesothelioma patient who smoked and he confirmed that he would deal with his theoretical life expectancy absent mesothelioma by the retrospective method making a deduction to reflect the additional health risks appropriate for patients who smoke.

51. Whilst I accept from what the experts have told me that both the prospective and retrospective methods of assessment are scientifically legitimate I reach the view that the prospective method is very much dependant on finding data or studies which accurately match the patient concerned. Where this is difficult, the retrospective method would appear to be more appropriate method, accepting as I do that it is very rough and ready method depending mainly on the clinical judgment of the physician making the assessment. The issue in this case then is whether the data relied on by Professor Channer are a close enough match to the deceased to mean that the prospective method of assessment is likely to be more accurate than the retrospective method.

52. I am satisfied that the studies relied on by Professor Channer prove the individual points that he wishes them to make but they are not wholly supportive, in my judgment, of his overall conclusion. For example, whilst the Weintraub paper quoted in paragraph 48 above does suggest a mortality rate of 4% per annum for CABG patients this would not lead to a 50% mortality in the population for 17 years. Similarly, whilst the Hlatky paper does show that CABG patients with diabetes do half as well as those who do not have diabetes the graph shown at paragraph 4.6 of Professor Channer's first report seems to show mortality of around 20% at 8 years in the "CABG diabetes group" which would suggest perhaps that 20 years might pass before 50% mortality was reached. Professor Channer relied on the paper by Solomon and others referred to in paragraph 48 to show that the lower the ejection fraction measured on echocardiography the higher the mortality. This is borne out by the graph he exhibited at page 535 of the trial bundle:



From this graph, it can be seen that the patients in the 43-52% category into which the deceased would have fallen have an annual mortality of about 4% when non-cardiovascular causes are removed. One of the conclusions of the Solomon paper was:

> *"All cause mortality, cardiovascular death, and all components of cardiovascular death declined with increasing ejection fraction of 45%, after which the risk of these outcomes remained relatively stable with increasing LVEF"*

The importance of the ejection fraction as a tool for assessing the condition of the heart is emphasised by this study.

53. The Claimant relies on a paper by Michael E Farkouh and others[5] which was a study about patients with diabetes and the relative benefits of CABG compared with percutaneous coronary intervention. The study found that CABG had a lower overall mortality rate which is perhaps best illustrated by the following table:

[5] Farkouh et al. Strategies for multivessel revascularisation in patients with diabetes. New England J Med. 2012; 367; 2375-84

Table 2. Kaplan–Meier Estimates of Key Outcomes at 2 Years and 5 Years after Randomization.

Outcome	2 Years after Randomization		5 Years after Randomization		Patients with Event		P Value*
	PCI	CABG	PCI	CABG	PCI	CABG	
	number (percent)				*number*		
Primary composite†	121 (13.0)	108 (11.9)	200 (26.6)	146 (18.7)	205	147	0.005‡
Death from any cause	62 (6.7)	57 (6.3)	114 (16.3)	83 (10.9)	118	86	0.049
Myocardial infarction	62 (6.7)	42 (4.7)	98 (13.9)	48 (6.0)	99	48	<0.001
Stroke	14 (1.5)	24 (2.7)	20 (2.4)	37 (5.2)	22	37	0.03§
Cardiovascular death	9 (0.9)	12 (1.3)	73 (10.9)	52 (6.8)	75	55	0.12

* P values were calculated with the use of the log-rank test on the basis of all available follow-up data (i.e., more than 5 years).
† The primary composite outcome was the rate of death from any cause, myocardial infarction, or stroke.
‡ P=0.006 in the as-treated (non–intention-to-treat) analysis.
§ P=0.16 by the Wald test of the Cox regression estimate for study-group assignment in 1712 patients after adjustment for the average glucose level after the procedure.

According to this data, death from any cause resulted in 10.9% of patients five years after randomisation. This again is more suggestive of a longer period of survival on average than eight years. I know that most of the patients in this trial had a LVEF of more than 40% but this only shows to emphasise the relevance of the LVEF to prognostic factors.

54. It against this background that I must consider Professor Channer's reliance on the Velazquez paper that I considered in more detail in paragraphs 35-40 of this judgment. The concerns I explored about the applicability of that paper to the deceased are clearly relevant to the issue of life expectancy as well. The fact that the deceased had an LVEF of 45 % whereas all the patients in the study had an LVEF of less than 35% is clearly relevant particularly given the issues I have explored in the last few paragraphs. Professor Channer had to concede that the deceased could not have participated in the Velazquez trial because his LVEF reading was too high (and therefore he had better cardiac function than the subjects). Professor Channer was asked in cross-examination why he chose that study and he replied that it was recent and it was the study which was the best fit for the deceased constellations of problems. The difficulty with this argument is that all of the patients in that study had worse LVEF readings than the deceased and many of them had other co-morbidities that he did not have.

55. In my judgment, the prospective method of life expectancy can only be preferable to the retrospective method where the data or studies used to make the assessment are as near a fit as possible to the actual medical condition of the Claimant or deceased being assessed. In this case the constellation of the deceased's symptoms and co-morbidities mean that it is impossible to approach the prospective method of assessment with any real confidence given the material I have considered in the preceding paragraphs of this judgment. The retrospective method of assessment is a rough and ready approach based not upon scientific study but on clinical judgment of experienced and qualified practitioners. I doubt it is a method they use in their clinical practice but it is a genuine attempt to answer a question which lawyers put to them to assist the court in a way it can understand. In this case I prefer the retrospective method of assessment as put forward by Dr Witte for the reasons I have expressed.

56. Counsel for the Claimant submitted that it would be open to me to make a finding of life expectancy which is somewhere between the two estimates. This is tempting as my gut reaction is that Dr Witte's estimate may be too optimistic whereas Professor Channer's may be too pessimistic. I have however chosen a method of assessment which is reliant on the clinical experience and judgment of a properly qualified clinician. I have no experience or expertise in this area and I believe it would be wrong for me to substitute my view for his where I have no sound basis for doing so. I accordingly find that the deceased's life expectancy, but for the mesothelioma would have been 19.5 years from the date of trial.

57. Counsel for the Defendant addressed me briefly about the application of the various tables when dealing with a fixed period of life expectancy but I got the impression that this would only be an issue if I preferred Professor Channer's view about life expectancy. On this basis, I will merely say that I prefer the use of Table 1 of the Ogden Tables as adjusted for the reasons expressed by Mr Justice Cranston in paragraphs 40-43 of his judgment in *Smith v LC Windows Fashions Ltd* [2009] EWHC 1532 (QB).

58. **The claim for dependency on the deceased's services**

Almost all the claims relating to quantum are agreed subject to my findings on the issues dealt with above. There are however two issues which require the court's determination. The Claimant makes a claim for both her past and future dependency on the deceased's services and the Defendant denies that the Claimant is entitled to succeed on the facts of this case. In the schedule of special damages the claim is expressed as follows:

> *"As a result of her husband's death, the Claimant has required assistance with tasks such as gardening, DIY and decorating. A nominal figure of £500 per year is claimed from the date of death until trial"*

A further claim was made following trial for the same claim at the same annual amount for a period of 15 years.

59. In his first witness statement dated 18th July 2015 the deceased stated:

> "*We also have a nice garden at the front and a large garden at the rear. I mainly take care of this and mow the lawn and look after the plants but I don't know how I am going to do this now"*

This suggests he had been mowing the lawn up to his mesothelioma diagnosis. His statement also suggested that he and the Claimant were planning on doing quite a lot of decorating and DIY in their new home. The Claimant had to concede however that prior to the mesothelioma diagnosis she was giving him a lot of personal care, up to 35 hours per week. She said "he could do a few things but very, very slowly" and she also agreed that he was incapable of decorating the house at this stage. Whilst I accept the deceased did mow the lawn at some point in the past I am not convinced he was doing this before his diagnosis of mesothelioma. The two questionnaires he completed in 2012 and 2014 reveal extensive disability such that the Claimant was awarded a carer's

allowance. In January 2014, he was asked if he could safely move 50 metres (page 715 of the trial bundle). His reply was:

> "I *cannot go any distance unless I take it slowly and stop for rests. Any kind of effort can bring on shortness of breath and chest pain. I do need a walking stick sometimes"*

He was then asked about going up or down two steps and his reply was:

> *"I can manage two steps as long as I take it slowly. I can go upstairs ok but I usually have to stop and catch my breath a few times"*

60. In my view these answers are not consistent with a man who is carrying out DIY decorating and gardening at home. The impression I got was that the Claimant was acting as the deceased's full time carer before the diagnosis of mesothelioma and that she carried on doing just the same afterwards, albeit with the need to devote more time to his complicated medication regime. The burden of proof is on the Claimant and I am not satisfied on balance of probability that she has lost the benefit of the deceased services in any meaningful way as a consequence of his death.

61. **Loss of partner's care and attention**

This claim is expressed simply in the schedule of special damages as follows:

> *"It is claimed that compensation should be awarded for loss of partner's care and attention and an appropriate award under this heading is £4500 see Bath v Highgate [2004] EWHC 707 (Ch)"*

This is uprated in the schedule to £6500.27 by application of the Retail Prices Index since 2004. Counsel for the Claimant seeks however £5000 in his skeleton argument for the purposes of the trial. Leading Counsel for the Defendant relies on the authority of *Mosson v Spousal (London) Ltd* [2015] EWHC and contends that this is what the bereavement award is for and no further award should be made. Counsel for the Claimant suggests that the decision in *Mosson* is an outlier which appears not to have been followed perhaps because of the curious way in which it was argued to be merited in that case.

62. This type of claim found its origins in *Regan v Williamson* [1976] 1 W.L.R. 305 where it was held that, in determining the pecuniary value to be put on the services of the deceased, acknowledgement should be given to the constant attendance of a mother on her children, and accordingly the value placed on such services should not be limited to a mere computation of the cost of services of a housekeeper less the cost of the deceased wife's maintenance. So, too in *Mehmet v Perry* [1977] 2 All E.R. 529 an additional sum was awarded because "the children have lost the attention of their mother and …now have only one parent to look after them instead of two". In the same case the husband was awarded a modest sum because he had lost the care and attention of his wife.

63. The development of this principle to widows and widowers in addition to children was recognised by Mr Justice Hamblen (as the then was) in *Beesley v New Century Group Ltd* [2008] EWHC 3033 (QB):

> *83. In my judgment the principle of making awards for loss of intangible benefits is now well established – see Kemp and Kemp [29-052]. It reflects the fact that services may be provided by a mother, wife, father or husband over and above that which may be provided by a paid replacement. In principle there is no reason for differentiating between the position of children and spouses in connection with the availability of such awards.*
>
> *84. In relation to services provided by a husband or father the position is summarised in Kemp and Kemp at p29074 as follows:*
>
> > *'Awards of this kind have also been made to a widow or child for the loss of services provided by a deceased husband or father. There is no reason in principle why such awards should not be made where the services provided by a husband or father justify it on the facts. Such awards ought to be in proportion to the more conventional awards already noted for wives/mothers. This will mean that they will be lower in the average claim where the deceased husband/father was the family breadwinner.'*
>
> *85. The present case is a good illustration of why it may be appropriate to make such an award to a widow. So, for example, there are considerable advantages in having jobs around the house and garden done by a husband at his own time and convenience rather than having to go out to find and choose commercial providers, and to have to work around the hours that suit them for the work in question.*

64. Whilst recognising that awards of this nature have become commonplace in fatal accident claims Mr Justice Garnham in *Mosson* questioned the jurisprudential foundation for this type of claim:

> *"71. I have had careful regard to these previous cases, in particular to the reasoning of Hamblen J and Mackay J. I take on board the fact that the making of awards of this sort has become increasingly commonplace. However I regret to say that, for two reasons, I find myself in disagreement with the conclusions of the other judges of this Court to whom I have referred. I can see no proper jurisprudential foundation for this claim.*
>
> *72. First, damages for personal injuries are intended, so far as money can achieve it, to put the claimant in the position he or she would have been in had the tort not occurred. But that is an*

art not a science, especially in the case of claims for future loss. There can be no precise equivalence in money terms of every loss that flows from an injury or a death. The Court fixes on a sum, often by reference to commercial costs, but recognises that that is unlikely to be a precise reflection of all the future consequences of the event. In the case of claims for services, the award is the Court's best estimate of the value, rather than the cost, of the services lost.

73. I have already made an award in respect of the services the deceased would have provided his family had he not contracted mesothelioma. I have done so by seeking to estimate the cost of providing commercially what would otherwise have been provided by the deceased. Obtaining services commercially, rather than from a member of the family, has both advantages and disadvantages. The disadvantages are those to which Mr Steinberg refers. But since few individuals, even caretakers, possess all the skills of all trades people, there are also advantages in having work carried out commercially. The work can be provided by specialist contractors at times that are convenient to the claimant; it may be of a better quality; its provision does not necessarily detract from other activities the family may wish to carry out. The award I have already made recognises both the advantages and disadvantages of having services provided commercially rather than by the deceased.

74. In my judgment, there is no room here for an additional award for the loss of intangible benefits over and above the claim for the lost services.

75. Second, what the claimant seeks is further financial compensation for the inconvenience of having to pay someone to do what her husband would have done voluntarily. In other words, she seeks financial compensation for what is a non-financial loss consequent upon her husband's death. That, it seems to me, is a claim of the sort which bereavement damages were intended to cover."

65. I recognise that these claims have become commonplace but I find myself in agreement with Mr Justice Garnham for the same reasons he gives. My personal view on the jurisprudential basis of this type of claim is however irrelevant in this case. This is a case where the deceased was unwell at the time he was diagnosed with mesothelioma. He was receiving personal care from the Claimant for at least 35 hours per week as he was too unwell to manage without that care. I have already found that his medical condition was such that he was unable to provide services such as DIY, gardening and decorating by 2014 and made no award for the loss of those services. As a consequence of this finding, even if a claim pursuant to ***Regan v Williamson*** could be validly made it could not be justified on the facts of this case as the deceased would not have been fit enough to do the various "jobs round the house" which formed the factual foundation for the successful claim made by the Claimant in ***Beesley v New Century Group Ltd.*** I have no doubt that the Claimant has lost the care

and attention of the deceased in the emotional sense and the loss of that cannot be minimised but it does not sound in additional damages because this is exactly the loss that the bereavement award (modest though it is) is intended to compensate for.

66. Counsel for the Claimant felt that Mr Justice Garnham had been led into error by claimant's counsel in *Mosson* expressing the claim as "loss of intangible benefits" rather than loss of love and affection. It is clear however from my analysis above that the genesis of this claim is based upon the perceived advantages of having a service performed by a member of the family rather than a commercial provider (and compensation for that loss). If a claim was in fact put forward, simply for loss of love and affection, in my view it would fail for being encompassed in the bereavement award whichever school of thought prevails about claims pursuant to *Regan v Williamson*.

67. For the reasons I have expressed I make no award for this head of loss.

68. Both counsel kindly indicated that if I ruled on the two outstanding issues in relation to quantum they would perform and agree a calculation of the Claimant's claim which could be incorporated in an agreed order. I would like to express my thanks to them for this help and their assistance generally.

69. This Judgment will be handed down on a date to be fixed by the court in public. The time for appealing the Judgment shall not start to run until it is handed down. CPR Practice Direction 40E shall apply. If the parties can agree the form of an order and any consequential directions arising from this Judgment then the attendance of leading and junior counsel and solicitors will be excused.

Appendix: a guide to terminology

1 The heart comprises four chambers: two upper collecting chambers (atria) and two pumping chambers (ventricles). Blood from the body enters the right atrium and flows to the right ventricle, from where it is pumped to the lungs. It returns to the left atrium, whence it passes to the left ventricle to be pumped into the aorta and round the body.

2 A cardiac cycle comprises the relaxation phase, diastole, during which the chambers fill passively, and the ejection phase, systole. In systole, the atria contract first, contributing about 20% to ventricular filling; the ventricles contract a fraction of a second later. The proportion of blood in the left ventricle (LV) which is ejected during systole is the ejection fraction (EF), normally around 55 to 70%.

3 Cardiac muscle fibres normally contract in unison. A state of uncoordinated contraction of the muscle fibres is known as fibrillation. Atrial fibrillation (AF) causes loss of the atrial contribution to ventricular filling and may lead to a rapid heart rate but is compatible with long-term survival. Ventricular fibrillation (VF) causes complete loss of cardiac output and death within minutes: it is one rhythm seen in cardiac arrest, the other being asystole (absence of any ventricular activity).

4 The heart itself is supplied with blood from the coronary arteries (left and right; the left divides after a short main stem into anterior descending and circumflex branches). Coronary artery disease (CAD; also ischaemic heart disease, IHD), in which the coronary arteries are narrowed (stenosed) by atheroma (fatty deposits in the walls), is a cause of angina (pain in the chest arising from inadequate oxygen supply to heart muscle). Angina usually occurs when the heart is called upon to work harder than normal, for example on exertion. The stenoses may be seen on coronary angiography, a procedure in which contrast medium is injected into the coronary arteries and a video x-ray taken. If a branch of a coronary artery becomes blocked, for example by rupture of an atheromatous plaque, the portion of heart muscle it supplies may die (myocardial infarction, MI) and be replaced by a scar. In coronary artery bypass grafting (CABG), vein is attached to the aorta and to the coronary artery, bypassing the narrowed section and improving distal blood flow. Arterial conduit may also be used.

5 IHD can cause diminution in function of the LV (left ventricular failure, or heart failure). The EF is diminished and the left ventricle dilates: the latter is reflected in an increased left ventricular end-diastolic diameter (LVEDD) seen usually on echocardiography (cardiac ultrasound). Increased pressures required to fill the failing LV can lead to fluid leaking into the lung (pulmonary oedema) and breathlessness.

6 Oxygen is carried in the blood bound to haemoglobin contained in the red blood cells. Only minimal quantities are dissolved in the plasma. A reduction in the haemoglobin concentration in the blood (anaemia) causes a proportionate reduction in the capacity of the blood to carry oxygen.