OUTER HOUSE, COURT OF SESSION

[2018] CSOH 84

A652/13

OPINION OF LORD ARMSTRONG

In the cause

(FIRST) EUPHEMIA BROWN, as an individual and as Executrix of the late WILLIAM BROWN; (SECOND) BRIAN BROWN; (THIRD) WILLIAM BROWN; (FOURTH) WILLIAM BROWN, as legal representative of his daughter IMOGEN BROWN; (FIFTH) WILLIAM BROWN, as legal representative of his son CHRISTOPHER BROWN; (SIXTH) DAVID BROWN; (SEVENTH) DAVID BROWN, as legal representative of his son AARON YARDLEY-BROWN; (EIGHTH) DAVID BROWN, as legal representative of his son CALLAN YARDLEY-BROWN; (NINTH) ELIZABETH AYDEMIR; AND (TENTH) MARSHALL BROWN

Pursuers

against

DR ALISON SMITH, DR NEIL WRIGHT AND DR JOHN WALLACE, General Practitioners practising in partnership as CRAIG NEVIS SURGERY

Defenders

Pursuers: Milligan QC, Smart; Drummond Miller LLP
Defenders: Smith QC, Pugh; Clyde & Co

15 August 2018

Introduction

[1] The pursuers are respectively the widow, the sons, the sister, and the brother of the late William Brown ("the deceased") who suffered a fatal cardiac event on 6 January 2011.

The defenders are general practitioners who practice together in partnership as Craig Nevis Surgery, Fort William.

[2] In the case as averred on Record, the pursuers sought damages from the defenders on the basis that the deceased's death was caused by negligence on the part of Dr Alison Smith in the course of the deceased's attendances with her on 29 December 2010, 31 December 2010, and 5 January 2011. In advance of the diet of proof, it was agreed between the parties that Dr Smith was negligent, on 5 January 2011, in failing, on that day, immediately to admit the deceased to hospital for further investigation. Subsequently, on the basis of evidence elicited, it was further agreed that, on 31 December 2010, Dr Smith was negligent to the extent that, on that day, she ought to have provided the deceased with advice that if his chest pain occurred more frequently with little or no exertion or lasted longer than 15 minutes, then he should call a 999 ambulance ("worsening advice"/"safety net advice"). In the light of that admission, the pursuers withdrew their allegations of negligence in relation to the deceased's attendance with Dr Smith on 29 December 2010.

[3] Beyond the admitted negligence on 31 December 2010 and 5 January 2011, the pursuers' cases of fault were that Dr Smith:

(i) failed to diagnose unstable angina on 31 December 2010;

(ii) did not refer the deceased to hospital on 31 December 2010;

(iii) did not prescribe appropriate medication for stable angina, namely low dose aspirin and beta blockers, on 31 December 2010

[4] In all respects, the issue of causation remained live. The pursuer's case on causation was (i) that admission to hospital on or before 2 January 2011 would have resulted in treatment that would have avoided the fatal cardiac event, and (ii) that admission to hospital

would have avoided exposure to the cold, a factor which could have contributed to the onset of the fatal cardiac event, on 6 January 2011. The quantum of damages was agreed.

The agreed facts

[5] The facts admitted on record, and agreed by joint minute, were to the following effect.

[6] As at 29 December 2010, the deceased had not previously suffered from chest pain. His first episode of chest pain commenced on Wednesday 29 December 2010, after he had been walking up and down stairs. His pain eased after five minutes of rest. He attended Dr Smith, his general practitioner ("GP"), on that day. She noted the circumstances of the onset of his chest pain. She did not note the site of the pain, nor whether there was any radiation of the pain. She noted that he was a non-smoker, and that a cholesterol test the previous year had been normal. She checked his blood pressure and pulse and found them to be normal. She arranged for him to have an electrocardiogram ("ECG") test. He had previously had an ECG in October 2009. Dr Smith's notes of the consultation were as follows:

> "chest pain this am - came on after going up and down stairs. Eased after sitting for 5 mins - never had anything like it before. felt like when breathing in when cycling in v cold weather. not sick or sweaty with it. just getting over flu, but no cough etc now. O/E chest clear, pulse reg 70 BP 120/80, heart sounds 1 + 2 + 0. father MI Willie never smoked and cholesterol ok last year - for ECG".

[7] On Thursday 30 December 2010, the deceased attempted to go for a walk but only got as far as his garden gate, due to chest pain. The pain resolved when he sat down and rested.

[8] On Friday 31 December 2010, the deceased suffered further chest pain while getting dressed. His wife arranged an emergency appointment with Dr Smith. The deceased

attended Dr Smith on that day, complaining of chest pain which she noted he had experienced after walking for five minutes the previous day. She noted the chest pain to be retrosternal and localised. She reviewed the deceased's ECG taken on Wednesday 29 December 2010. She prescribed a trial of glyceryl trinitrate ("GTN") spray and made an urgent referral for an exercise ECG or 'exercise tolerance test' ("ETT"). She made a review appointment for him. She did not prescribe low dose aspirin, a beta blocker and a statin. Dr Smith's notes from the consultation were as follows:

> "Chest pain, as last Wednesday - went for a walk yesterday - sharp retrosternal pain after 5 mins - had to slow right down. no feeling sick or sweaty or unwell - pain reasonable localised. no pain moving - has been practicing golf swing - has holiday to Florida booked for Jan. O/E chest clear PF 550; BP 130/80; pulse reg. Reviewed ECG from Wed no changes from 2009. for trial of GTN spray and urgent ref for exercise ECG. Review Wed as planned."

[9] On Wednesday 5 January 2011, the deceased attended Dr Smith complaining of continuing chest pains, and of an episode of chest pain while at rest. He complained of an episode of chest pain the previous night, while lying down. Dr Smith prescribed gaviscon and omeprazole for suspected acid reflux. Dr Smith's notes of the consultation were as follows:

> "Patient reviewed pains continue - last night worse lying down - still retrosternal. took NSAID while had flu O/E abdo NAD. try gaviscon and omeprazole. r/v Mon"

She failed to refer the deceased for admission to hospital, as an urgent referral.

[10] On Thursday 6 January 2011, at 10.45am the deceased attended at the Accident and Emergency Department ("A&E") of Belford Hospital, Fort William. His triage notes recorded:

> "67 year old man walked into department c/o chest pain.
> 1 week Hx of chest pain. Exercise induced. Seeing GP for this - prescribed GTN 1 week ago. Pain got much worse last night - '10/10' at worst point. Pain not radiating from chest. Pain at moment '1/10'. Awaiting exercise tolerance test apt."

On further examination, by Dr Bawa, the following was noted:

> "1 week history chest pain - on exertion. Has been referred for exercise tolerance test. Pain started following episode of flu-like symptoms. Pain last night worst at midnight - when at rest. GTN spray partially improved the pain. Lasted few minutes, returned again when climbing steps into hospital - now resolved. No nausea/SOB/sweaty. No cough."

His vital signs were normal. Troponin I testing was carried out, which was negative. Two ECGs were carried out, which were normal. He was prescribed gaviscon and discharged, at 2.30pm. At 3pm, after arriving home, the deceased developed chest pain when sitting down. He collapsed at around 3.30pm. His collapse was witnessed by paramedics from the Scottish Ambulance Service, who undertook attempts to defibrillate but, upon applying monitoring electrodes, they discovered that he was in ventricular fibrillation. He was defibrillated six times. His heart rhythm proceeded to pulseless electrical activity and then to asystole. At 4.30pm the deceased arrived at Belford Hospital, receiving cardiopulmonary resuscitation. He was asystolic. The resuscitation in A&E was unsuccessful. He was pronounced dead at 4.45pm.

A *post-mortem* examination was carried out on 11 January 2011. The report concluded:

> "*Post-mortem* disclosed an enlarged heart with significant narrowing of the left main coronary artery. There was also prominent pulmonary oedema in keeping with acute left ventricular failure. Microscopy has shown evidence of previous myocardial ischaemia ...
> Individuals with an enlarged heart are at increased risk of sudden death, most likely mechanism of death being the development of arrhythmia."

The evidence

[11] In all, I heard the evidence of ten witnesses: Dr Smith, Mrs Brown, and, Ewen Campbell, a family friend, who spoke to the facts of the deceased's presentation over the relevant period, and seven expert witnesses, of whom two, Professor Ian Wall and Dr Philip Gaskell, gave evidence from the perspective of a GP, two, Mr Neil Nichol and

Dr Graham Johnson, gave evidence from the perspective of an A&E clinician, two, Professor Stephen Brecker and Dr Kevin Channer, gave evidence from the perspective of a cardiologist, and Professor Derrick Pounder, who gave evidence relating to *post mortem* pathology.

The factual background

[12] (1) Dr Smith gave evidence under reference to her witness statement, dated 23 April 2018. In general terms, the deceased was a man who had enjoyed good health. He had been an active cyclist and had played golf.

[13] When he attended with her, on Wednesday 29 December 2010, she did not reach a diagnosis as to what was causing his chest pain. She had considered that one possibility was a cardiac problem, and for that reason had requested an ECG. She had advised the deceased to come back to her if his symptoms continued, and arranged a review in one weeks' time, in any event, on the basis that, after such a period, it would be clearer as to whether his chest pain was an isolated incident or was getting worse. Notwithstanding the terms of the NICE Guidelines on Chest Pain of Recent Onset, which set out the symptoms which might indicate an acute coronary syndrome ("ACS"), she conceded that she had not considered the possibly of ACS. Although she had not been considering ACS, she was considering a possible cardiac diagnosis of stable angina. She accepted that if her diagnosis had been one of unstable angina, she would have referred the deceased to hospital that very day. She confirmed that although she had not made any diagnosis, she had not ruled out one of angina.

[14] When the deceased returned to her on Friday 31 December 2010, that had been earlier than had been planned for her intended review. He was experiencing more chest

pain. It occurred with movement on the flat, rather than when going up and down stairs, and so could be characterised as having become worse. She accepted that in circumstances where he was experiencing chest pain after only five minutes walking, the pain could be characterised as being manifest after minimal exertion. Notwithstanding that, she did not consider ACS, on the basis that the pain was being caused by activity. Her view was that angina was more likely. When referred to the NICE Guidelines, which indicated that symptoms which might indicate ACS included:

> "new onset chest pain, or abrupt deterioration in previously stable angina, with recurrent chest pain occurring frequently and with little or no exertion, and with episodes often lasting longer than 15 minutes",

she accepted that the deceased's pain was new onset pain. It had started two days previously. On that basis he could have been characterised as having stable angina since Wednesday 29 December 2010. Although the pain could have been described as new onset pain and manifesting after little exertion, she did not consider ACS, on the basis that the pain lasted for less than 15 minutes.

[15] She maintained that she had advised the deceased to phone NHS 24 if his symptoms deteriorated, despite the fact that no mention was made of that in her consultation notes or in her written statement. She was sure that she would have advised him to that effect. She accepted that she had not advised him to phone for an ambulance if his symptoms became worse. Under reference to the report by Professor Wall, dated 12 September 2016, at paragraphs 8.5 and 8.6, in relation to the appropriate management, given the deceased's presentation, she agreed that at the consultation on Friday 31 December 2010 she should have given advice that an ambulance should be called in the event that the deceased's symptoms deteriorated. She did not agree that such advice would have been appropriate on 29 December 2010, on the basis that at that point she had not yet made a diagnosis. She

accepted, on the basis of her knowledge of the deceased, that if she had given the advice that an ambulance should be called as appropriate, he would have followed that advice.

[16] On Friday 31 December 2010, she had prescribed GTN spray. She had considered other options such as aspirin, statins, or a beta blocker but had decided not to prescribe them. She did not prescribe a beta blocker because the pursuer was using salbutamol for wheezing, and she did not want to exacerbate that condition. Beta blockers were contra-indicated where there was asthma. In so far as alternatives to beta blockers were concerned, they all had side effects and she did not want him to feel worse. In her view, at that time he was stable. She had not prescribed statins because the previous year's test results had indicated that his cholesterol had not been raised. She did not prescribe aspirin because that can cause indigestion and she was still, at that point, not yet sure if he was suffering from heart disease, although she was considering it as a likely diagnosis.

[17] Under reference to SIGN 96 "Management of stable angina": "Pharmacological Management", she accepted that she had not followed the guidelines for drug intervention recommended for first line therapy, except in respect of GTN spray. She had not prescribed aspirin because it could cause indigestion and was properly viewed as a long-term prophylactic measure.

[18] At the time she had not made a 100 per cent diagnosis of angina, but rather was waiting for his response to the GTN spray. Under reference to the NICE Guidelines, at paragraph 1.2.1.4: "Do not use people's response to glyceryl trinitrate (GTN) to make a diagnosis", her approach had been contrary to the recommended guidelines.

[19] In relation to the deceased's consultation with her on Wednesday 5 January 2011, she accepted that, by that time, the deceased was manifesting unstable angina which she recognised as a form of ACS. On that basis, she accepted that she should have made a

referral to hospital. She accepted that she had not given advice to the deceased that he should go to the hospital if his symptoms became worse. She accepted that, at that time, ACS should have been actively considered and that the deceased should have been referred to hospital that day for cardiology assessment.

[20] In relation to the attendance on Friday 31 December 2010, under reference to the report by Professor Wall, at paragraph 8.9, in circumstances where she accepted that pain after walking for five minutes was consistent with angina, she agreed that the prescription of low dose aspirin and statins would have been an appropriate preventative measure, but that she had chosen not to prescribe them. She agreed that she should have made an urgent referral to hospital.

[21] Although the deceased had been describing pain after exercise which could be consistent with angina, his description of the pain was atypical of what a patient with angina would describe. On that basis, since her diagnosis was not certain, she chose not to prescribe statins. She accepted that she was thinking "he may well have angina", but she had not yet made that diagnosis. She accepted that if she had diagnosed angina, she would have prescribed differently. She would have prescribed low dose aspirin, and statins, and beta blockers or something similar. However on 31 December 2010 she had not done that because she was not yet certain that he was suffering from angina.

[22] In cross-examination, Dr Smith confirmed that she had practised consistently as a general practitioner since 1985, when she graduated MBChB. The practice was situated two miles from the centre of Fort William, and two miles from the local hospital, Belford Hospital. It was a district general hospital with medical and surgical consultative services, an A&E department, and allied health professional services. It had no cardiology

department. Cases requiring cardiology assessment would be referred to a hospital in the central belt, most probably in Edinburgh.

[23] She was familiar with the NICE Guidelines which she recognised as providing guidance on particular illnesses, including angina and chest pain. She did not routinely refer to them as part of a first line checklist. She was also familiar with the SIGN Guidelines which were the Scottish equivalent. Her practice, if checking, was to refer to them rather than the NICE Guidelines. Under reference to SIGN 96 "Management of stable angina": "Pharmacological Management", she agreed that the prescription of beta blockers was an appropriate first line therapy for relief of stable angina. Under reference to "Management of stable angina": "Diagnosis and Assessment", she recognised that a patient with chest pain might not necessarily be suffering from angina, and that there could be possible alternative diagnoses. She confirmed that, when assessing the deceased, she had in mind the type of discomfort described, its location, its relation to exertion, its duration and other factors relating to the deceased's pain, all consistent with the terms of SIGN 96 "Management of stable angina": "Clinical assessment". In relation to his general lifestyle, there had been a number of factors which it was necessary to take properly into account.

[24] In relation to her consultation with the deceased on Wednesday 29 December 2010, her recollection was that in describing his chest pain after climbing up and down stairs, he had indicated that he had been carrying cases at the time. On that basis, she had assessed that activity as involving more exertion, and more increased effort, than normal.

[25] She had noted the deceased as not feeling sick or sweaty. That was to be considered in the context of the nature of pain being subjective and often difficult for a patient to describe. Generally, where there was angina, the patient found the pain difficult to describe, but often complained of being sweaty or of feeling sick or having a heavy feeling. Patients

with angina often did not describe pain well. In contrast, the deceased had complained of a sharp pain which was clearly localised. That was an unusual description from a patient suffering from angina. She confirmed her understanding, that angina did not usually present as involving a sharp or stabbing pain, as being consistent with the terms of SIGN 96 "Management of stable angina": "Non-cardiac chest pain", at paragraph 2.1.2.

[26] She had considered it significant that he was "just getting over flu". He had been unwell, had been in bed for two weeks, had developed a cough, and probably had weaker musculature as a result. She had taken into account his family history ("father MI"), and the fact that the deceased had never smoked and had normal cholesterol levels. She had referred him for an ECG examination, and had considered the possibility that the pain could be muscular. She accepted however that angina was a common complaint.

[27] She had been aware that, on Thursday 30 December 2010, the deceased had attempted to go for a walk but had only reached as far as his garden gate before suffering pain which subsided with rest. In relation to her consultation with him on Friday 31 December 2010, she was aware that he had suffered pain when getting dressed, but that it had been a sharp pain and that he had not felt sick or sweaty. She had prescribed GTN spray, requested an urgent ETT, and arranged a review on the following Wednesday. She confirmed that, at that point, her thoughts were that the deceased was describing the symptoms of stable angina, involving pain which came on with exercise.

[28] In relation to her consultation with the deceased on Wednesday 5 January 2011, at which she had noted that he had experienced pain lying down during the night, she had prescribed gaviscon and omeprazole as measures to counteract indigestion and reduce stomach acid. She had done so because she was considering acid reflux and dyspepsia in

the context of his recent viral illness. Her view had been that the pain he described on Wednesday 5 January was the same pain he had been complaining of earlier.

[29] Where an urgent referral to hospital was required, the mechanism involved would require Dr Smith to liaise with the hospital by phone, draft a written letter, and arrange a lift for the patient as transport to the hospital, two miles away, on the basis that an ambulance would in fact take longer to make the journey.

[30] Where an ETT was required, that could be provided by the local hospital. As at Friday 31 December 2010, an urgent referral carried with it the expectation that the patient would be seen within one week, or within ten days, but certainly after the New Year public holidays. The referral which she had made on Friday 31 December 2010 had been an urgent referral.

[31] Where there was an abnormal ETT result, further investigation, most probably in the form of an angiogram, would be required. For that purpose referral to a cardiologist would be required. That would be necessary before a diagnosis of coronary heart disease ("CHD") could be reached. Dr Smith's view was that, had the deceased been admitted to hospital on Friday 31 December 2010, and the decision taken that an angiogram was required, it would have been necessary for the angiogram to be carried out by a cardiologist, most probably in Edinburgh, which would have been unlikely to happen before several weeks had elapsed.

[32] In re-examination, Dr Smith confirmed her understanding that retrosternal pain was typical of angina.

[33] She confirmed that she had in the past, on several occasions, referred patients to Belford Hospital as emergency ACS cases.

[34] (2) Mrs Euphemia Brown, the widow of the deceased, described her former husband as an active man who played sport, and in particular golf, skiing, walking and cycling. He

had been in the habit of cycling every Sunday from Fort William to Spean Bridge and back, a distance of some 20 miles. In December 2010, he had suffered a bout of the flu, but otherwise, in the recent past, he had experienced no serious health complaints. On Wednesday 29 December 2010, her husband had gone to his GP, Dr Smith, complaining of chest pain. He had not had any previous chest pain problems. He had first mentioned his chest pain to her the day before. He had been moving the dissembled parts of a cot into the loft of their home, access to which was by a ladder. When he had come downstairs he had been complaining of pain in his chest. She had phoned the GP and had arranged an emergency appointment. The fact that he was complaining at all suggested to her that it must be something significant. He had appeared to be worried, and so she also had become worried. She did not attend the GP with him on that day. When he returned, he told her that an ECG had been carried out, but that nothing had showed up. He said that his doctor had said to him that it could be angina.

[35] On the next day, Thursday 30 December 2010, her husband had not done much at all. He had not been his usual active self. Whenever he moved, he experienced pain. He had attempted to go for a walk, but had returned within minutes, complaining that he could not get further than the garden gate. She had told him to sit down, and when he did so the pain quickly subsided. Each time he rose from a seated position, the pain returned. When he sat down, the pain resolved.

[36] On the next day, Friday 31 December 2010, he was complaining of pain again and went to his GP. That morning he had experienced pain after getting up from bed. He had not been doing much. Any movement brought on the pain, and so she had phoned his GP. That evening, which was Hogmanay, she and her husband had not participated in any

celebrations, but had stayed at home and had gone to bed, having brought in the New Year on their own.

[37] On New Year's Day, her husband had done nothing other than to sit on a chair all day. If he rose from the chair, his pain came back, but subsided when he sat down again. He was in pain whenever he was not sitting down. Pain was generated even by moving from his chair to the toilet. On New Year's Day, it had been the couple's custom to visit friends and they did so on that day. They visited their friend Mr Ewan Campbell. Her husband had struggled to get there on foot, even although Mr Campbell's home was only seven to eight minutes' walk from their own. On the way he kept experiencing pain, and had to stop several times before being able to carry on. It had been the same pain as that from which he had been suffering in the previous days.

[38] Mrs Brown had no doubt that if the GP had told him that he should go to the hospital if things got worse, he would have done so. The weather at that time had been cold and had remained cold in the days following New Year's Day. In the days up to 5 January 2011, her husband had done nothing involving any activity.

[39] On Thursday 6 January 2011, Mrs Brown had called an ambulance which had taken her husband to A&E at Belford Hospital. Her sister-in-law had gone with them. Access to the A&E at Belford Hospital was by 12 or 13 steps; there was no ground floor entrance. He had required to ascend and descend the steps when entering and leaving A&E. They had been dropped off at home, at the garden gate, at 2.30pm. Very quickly, after enough time only for a cup of tea, her husband had collapsed, and an ambulance was called at 3.00pm.

[40] (3) Mr Ewan Campbell had known the deceased for 44 years, and described him as an outdoor man with an active life. He had been a keen golfer, hillwalker, cyclist and skier. He had seen the deceased a few days before his death. The deceased and his wife had

visited him just after New Year. During the visit, the deceased had not seemed to be his usual chirpy self. He was a man who had customarily enjoyed New Year, but that was not the case on this occasion. He seemed slightly downcast. He sat down very quickly on entering the house. His wife had told Mr Campbell that the deceased had required to stop two or three times in the course of the journey, which was only a quarter of a mile or so, because of pain in his chest which was not normal. Mr Campbell described the deceased as a man who would not normally make a fuss, but rather kept himself to himself. The couple had stayed with Mr Campbell for two to two and a half hours. Throughout that time, the deceased had remained seated. He had insisted on having only one dram - which was uncharacteristic of him. The couple had left at 10.00pm. The weather had been cold and dry. The temperature had been above freezing, but there had been a cold wind. After that meeting, Mr Campbell never saw the deceased again.

The expert evidence

[41] It was agreed that in the case of each of the expert witnesses, his report was to be treated as his evidence in chief, subject to such further examination in chief as the court might allow.

(i) Diagnosis and treatment by a general practitioner of ordinary competence

[42] (1) Professor Ian Wall gave expert evidence for the pursuers, under reference to his report dated 12 September 2016. His view was that, in respect of the attendance on Friday 31 December 2010, given the history of chest pain on minimal exertion, and on that history alone, Dr Smith should have diagnosed ACS until proved otherwise. Such a diagnosis would have mandated the prescription of a loaded dose (300mgs) of aspirin, and an

immediate 999 emergency admission to hospital. Such a diagnosis would have been consistent with guidance given on the Patient.co.uk website: "Assessment for possible acute coronary syndrome", where it was stated that:

> "Symptoms that may indicate ACS include:
> New-onset chest pain or abrupt deterioration in stable angina, with recurrent pain occurring frequently with little or no exertion and often lasting longer than 15 minutes."

That description was consistent with what had been noted by Dr Smith in her consultation notes.

[43] On the hypothesis that it would have been reasonable for her to have reached the alternative diagnosis of stable angina, then the appropriate treatment would have been the prescription of GTN spray, low dose aspirin, a beta blocker if no contra-indication, statins, and "worsening advice" to the effect that in the event of further chest pain with minimal exertion or at rest, a 999 ambulance should be called.

[44] In relation to the drug therapy offered to the deceased, it was clear that Dr Smith had prescribed GTN spray. However, Professor Wall did not accept, as having been reasonable, Dr Smith's reasons for not prescribing low dose aspirin on the basis that it might cause indigestion, and that she had not yet reached a certain diagnosis in relation to angina. There was a need to balance risk and, in that regard, the risk posed by the condition of angina was potentially very great. To have prescribed low dose aspirin, in these circumstances would have been standard treatment. No competent general practitioner would not have prescribed low dose aspirin in these circumstances. If necessary, low dose aspirin could have been prescribed with other gastro-protective medication to protect the lining of the stomach. The only reason not to have prescribed low dose aspirin would have been in the case of the patient being allergic to it.

[45] In terms of the guidance set out in SIGN 96 "Management of stable angina", beta blockers should be used as a first line therapy for the relief of symptoms of stable angina. That was normal procedure. Professor Wall did not accept, as reasonable, Dr Smith's reason for not prescribing beta blockers on the basis that the deceased had been taking salbutamol. Where the patient was suffering from asthma, that was a contra-indicator for the prescription of beta blockers, but it was accepted, contrary to the notes made in error in the deceased's records, that he had never suffered from chronic obstructive pulmonary disease ("COPD"). In any event, even if the patient was intolerant of beta blockers, in terms of the SIGN 96 Guidance, alternatives could be prescribed in the form of rate limiting calcium channel blockers, long-acting nitrates or nicorandil. There had been no reason not to prescribe any of these drugs. No reasonably competent general practitioner would have failed to do so. To do so would have constituted standard management of angina. Although Dr Smith had been aware that the deceased did not suffer from COPD, she had noted that he had been "wheezy". If indeed the deceased had been wheezy at the relevant time, that would have constituted a contra-indicator for beta blockers, subject to the available alternatives.

[46] In cross-examination, Professor Wall accepted that before a GP could consider relevant treatment options, it was necessary to reach a working diagnosis, or a suspected diagnosis. He did not accept that the deceased's chest pain could have had a muscular cause, for the reason that there was no basis for it in the deceased's notes.

[47] Under reference to his report, at paragraph 6.15, where there was reference to Dr Smith's note: "Chest pain - this am - came on after going up and down stairs", Professor Wall expressed his view that such activity amounted to minimal exertion. He accepted that it was possible, in circumstances where the deceased had been carrying cases

up and down stairs, that muscular pain might have resulted, but, notwithstanding that, he maintained his view that the fact that chest pain had resulted from exertion was indicative of angina.

[48] Dr Smith had noted that at that time the deceased had been "not sick or sweaty with it". Professor Wall confirmed that such symptoms were associated with heart attack and ACS, as was vomiting. He accepted that such symptoms were also associated with angina. The incidence of angina in Scotland was higher than in England and Wales. Under reference to the SIGN 96 Guidance, the Scottish Health Survey (2003) had reported the prevalence of angina in males aged 65 - 74 (the deceased's age group), as 6.7 per cent.

[49] Professor Wall accepted that in determining whether a patient might or might not be suffering from angina, the history of the patient was critical to an appropriate judgement. He accepted that Dr Smith had had an advantage in that respect, in that she had had the opportunity to speak to the deceased, in circumstances where, having been in his presence, she could assess his presentation and how he was behaving, and had the opportunity to pick up unspoken signals. He accepted that Dr Smith's note that the deceased was "just getting over flu but no cough etc now." was indicative that she had been considering a chest infection. He accepted that it was also clear that Dr Smith had been considering the possibility of a cardiac issue, on the basis that she had noted that the deceased's father had suffered a myocardial infarction, and that she had, in the event, referred him for an ECG.

[50] In relation to Dr Smith's notes of the attendance on Friday 31 December 2010, in which she had described the pain suffered by the deceased as being "sharp" chest pain, Professor Wall accepted that the pain associated with angina was often described by patients as being dull, but in the centre of the chest. He accepted that sharp pain differed from the classic presentation, but maintained that it did not exclude a diagnosis of angina. In relation

to Dr Smith's note that the pain was reasonably localised and was not moving, Professor Wall accepted that angina was classically associated with radiating pain.

[51] He accepted that on the basis of her conclusions, Dr Smith had put in place a treatment plan, but he disagreed that its content was reasonable, on the basis that the deceased had been a man who was physically active, and who could cycle 20 miles regularly, but was now suffering from chest pain when entering and leaving a loft. These facts were indicative of something which could be a serious problem.

[52] When producing his report, Professor Wall had been aware of the content of Mrs Brown's statement, in particular to the effect that, on Thursday 30 December 2010, the deceased had attempted to go for a walk but had returned after about two minutes because of pain and had not gone further than his garden gate, which Professor Wall characterised as minimal exertion, that, when the deceased had sat down, the pain had gone, that, when he got up again, the pain returned, and that, on Friday 31 December 2010, the deceased had again experienced the same pain when getting dressed first thing in the morning. Professor Wall's view was that the account by Mrs Brown was a basis for recognition that the deceased had been continuing to suffer chest pain.

[53] He accepted that Dr Smith's reference in her notes to the deceased practising his golf swing, was indicative that she was considering a possible musculo-skeletal failure, and that her plan to review him on the following Wednesday was part of her treatment plan.

[54] He accepted that, when referring the deceased for an ETT, Dr Smith had indicated that one reason for urgency was the fact that the symptoms experienced by him had come on fairly rapidly. In his view, however, when angina was suspected, the referral should have indicated that the position was urgent rather than "fairly urgent", as Dr Smith had stated. Where such referrals were concerned, the standard expectation was that a patient

might be seen within two weeks or so. On the basis that the need was urgent, the patient would normally have been seen within a few days. In circumstances in which the timing of treatment was dependent on the availability of facilities, and the imminent holiday period would extend from 31 December 2010 until 4 January 2011, Wednesday 5 January 2011 being the first business day of the year, there had been all the more reason for the provision of clear worsening advice.

[55] Under reference to Mrs Brown's description of the deceased's walk to Mr Campbell's home after New Year, he characterised the facts of someone, who could cycle 20 miles regularly, having to stop because of the onset of pain when walking the relatively short distance of about quarter of a mile, as being indicative of the onset of chest pain with minimal exertion.

[56] In response to the suggestion that, in the following days, the deceased's problems had reached a plateau in their manifestation, Professor Wall's view was that the pattern, correctly stated, was that the deceased had been suffering from chest pain, caused by minimal exertion, over a period of several days. He accepted that the notes from A&E, at Belford Hospital, from 6 January 2011, recording one week of chest pain, "much worse last night", indicated a period of time extending backwards prior to 31 December 2010, during which the deceased had been struggling with pain on minimal exertion in circumstances where the pain resolved with rest, and during which the pattern continued until 1 or 2 January 2011 and possibly until 5 January 2011.

[57] He confirmed that in his view the appropriate diagnosis, as at Friday 31 December 2010, would have been that the deceased had been suffering from chest pain on minimal exertion and was to be considered as suffering from ACS until proved otherwise, necessitating the prescription of aspirin, and admission to hospital by 999 ambulance.

[58] Although in his report, at paragraph 8.9, he had stated that, if Dr Smith's evidence that the pain came on after walking for five minutes was accepted, the appropriate diagnosis would have been one of angina, which would have required referral for an ETT on an urgent basis, his expressed opinion, that the diagnosis should in fact have been one of ACS, with immediate 999 admission, was based on the fact that he had taken account of the evidence of Mrs Brown in relation to her descriptions of the symptoms being experienced by the deceased. Under reference to his report, at paragraph 8.10, his view was that, had a full history been taken at the relevant time, the appropriate diagnosis would indeed have been ACS, provoking an emergency 999 admission. Taking into account Mrs Brown's evidence, the deceased had been suffering from chest pain on minimal exertion which fulfilled a criterion for the diagnosis of ACS. In addition to that, he was critical of Dr Smith on the basis that she had not given any worsening advice.

[59] He accepted that Dr Smith's diagnosis, that being the first step necessary before putting in place a care pathway, was predicated on the information made available to her. He accepted that, on the information which Dr Smith had noted, that pain was coming on after walking for five minutes, her diagnosis of angina had been reasonable. Against that however, on the basis of the additional information provided by Mrs Brown's evidence, his view was that Dr Smith had failed to take a full history from the deceased. That additional information included the fact that the deceased had been suffering from pain on minimal exertion, as illustrated by his inability to walk beyond the garden gate, and his experience of pain when getting dressed. He reiterated that his criticisms of Dr Smith were that she had failed to take a full history from the deceased, in particular in relation to the identification of precipitating factors, and had failed to give him worsening advice. Dr Smith had failed to elicit from the deceased what was apparent from Mrs Brown's evidence. In that context, he

accepted that he did not know what questions had been asked of the deceased by Dr Smith, and did not know what the deceased's actual responses had been.

[60] He accepted that the SIGN Guidelines were more relevant for doctors in Scotland than the NICE Guidelines, but indicated that the matter was often one of personal preference. Under reference to SIGN 96 "Management of stable angina": "Diagnosis and Assessment", in a passage which stated:

> "Those patients who should be considered for early referral to secondary care include those with new onset angina and those with established coronary heart disease with an increase in symptoms.",

he noted that the reference was to "early" referral, rather than to urgent or immediate referral. He also accepted that further investigations and treatment, such as angiography and revascularisation were matters to be considered in the context of secondary care settings. He also accepted that SIGN 96: "Management of stable angina": "Establishing a diagnosis" included the statement that:

> "A significant proportion of patients with chest pain may not have angina and assessment should also try to identify alternative diagnoses at an early stage."

and further:

> "Patients with stable angina are usually managed in the primary care setting, but may present in a number of health care settings. An initial diagnosis of angina can be made within primary care but this should be supported by further assessment and risk stratification, which will normally require specialist input".

He accepted that Dr Smith's actions in relation to the deceased were consistent with these passages.

[61] Under reference to another passage in the same document, at paragraph 2.1.2 to the effect that: "Angina pain is not usually sharp or stabbing in nature.", Professor Wall accepted that Dr Smith's note of the type of pain being suffered by the deceased was not suggestive of angina. However, given the deceased's age, and his experience of central chest

pain, on exertion, which was relieved at rest, his presentation was so typical of angina that a diagnosis of angina should have been made until proved otherwise, and his case managed as such.

[62] He accepted that, on Wednesday 29 December 2010, Dr Smith had apparently asked the right questions, but maintained that she should have reached a diagnosis of angina until proved otherwise. On that basis, she should have prescribed aspirin, beta blockers - if not beta blockers, because of prior aspiratory problems, then appropriate alternative medication - and statins to lower cholesterol in the pulmonary artery. He accepted that the prescription of statins might take months to be fully effective. Aspirin acted to stop platelets clumping and causing clots. It would not be immediately fully effective, but its effect was more rapid than that of statins. It was recognised that, in such cases, there was a general benefit to be obtained from the prescription of aspirin.

[63] In relation to a report by Dr Gaskell, dated August 2016, in which it was stated that Dr Smith had been entitled to diagnose stable angina on the basis that the pain was not occurring frequently and with little or no exertion, Professor Wall's view was that Dr Gaskell's consideration of the matter had been based only on the evidence of Dr Smith, who had not taken account of the facts referred to in the evidence of Mrs Brown which indicated minimal exertion, as illustrated by the deceased's attempt to reach his garden gate and his pain on getting dressed. Professor Wall accepted that the reference by Dr Smith to "sharp retrosternal pain after five minutes" indicated pain on exertion, rather than pain on minimal exertion. Minimal exertion was characterised by activity less than five minutes' walk. Whereas Dr Smith had noted a history of pain after five minutes, Mrs Brown had given a history of the deceased suffering from pain on getting dressed and within the limited period necessary to reach the garden gate. Any reasonable person would consider

such activity to be minimal exertion. He disagreed with the suggestion that it would only be reasonable to expect Dr Smith to have reached a diagnosis of ACS with hindsight. In his view, had she elicited a full history, she would have reached a proper diagnosis. Her failure to have taken a full history was significant. Professor Wall's view was that no doctor of ordinary competence would have reached a diagnosis of stable angina on the basis of the facts comprising the evidence of Mrs Brown. Although he accepted that patients would not always give full answers to questions, he would expect a doctor of ordinary competence to elicit all the necessary and relevant information.

[64] In re-examination, he accepted that, in essence, the difference between him and Dr Gaskell on the issue of whether or not Dr Smith was negligent, was the fact that in his report, at paragraph 5.3, Dr Gaskell had attached weight to the statement that "The pain was not occurring frequently and with little or no exertion".

[65] On the question of the efficacy of aspirin, Professor Wall agreed with the statement in the report by Professor Channer, dated 22 February 2016, at paragraph 4.5, to the effect that the drug reduced mortality by about 25 per cent when taken by patients in the first 24 hours of acute myocardial infarction, and that the benefit is seen almost immediately on starting the drug.

[66] He considered that, on the basis of the test set out in the case of *Hunter* v *Hanley* 1955 SC 200, if Dr Smith had thought that the deceased was suffering from pain after minimal exertion, then, that being the definition of what constituted ACS, she should not have reached any other diagnosis.

[67] (2) Dr Philip Gaskell, General Practitioner, gave expert evidence on behalf of the defenders, under reference to his report, dated August 2016. He confirmed that, in his

opinion, in relation to the consultation on Friday 31 December 2010, on the basis of the notes taken by her, Dr Smith had acted appropriately.

[68] He agreed that the SIGN Guidelines were intended for the Scottish context, and that the NICE Guidelines had no status in Scotland, but were generally useful. Both sets of guidelines were in effect appraisals of current medical evidence.

[69] In relation to the consultation on Friday 31 December 2010, he confirmed his opinion that Dr Smith had been entitled to diagnose stable angina, on the basis that the pain was not occurring frequently and with little or no exertion. He qualified that by suggesting that in his view, rather than having reached a working diagnosis of stable angina, she had placed the possibility that the deceased was suffering from that condition at the top of her list of differential diagnoses. On that basis, he would have expected her, in 2010, to have referred the deceased for an ETT as a means of confirmation of such a diagnosis. Her referral in that regard had been appropriate, as had her prescription of GTN spray. In his view the effect of an "urgent" referral carried with it the expectation that the patient would be seen within two weeks. In his view the characterisation of the referral as such, was appropriate. In contrast an "emergency" referral would have involved the patient being seen on the same day.

[70] On the basis of the deceased's records, Dr Gaskell's understanding of the level of exertion which provoked the pain being suffered by him, as at Friday 31 December 2010, in the context of the pain remaining in the same area of the chest, being sharp, and not a dull ache, being the same as on the previous day, and coming on after five minutes, was of the onset of pain after exercise, but not after minimal exercise. On that basis the history was suggestive of stable angina. Although it was necessary for a GP to clarify with a patient the level of exertion which provoked pain, equally, it was reasonable to assume that not all the

detail would be recorded. Dr Smith should have checked with the deceased whether, when walking, he was walking briskly or slowly, or was carrying anything. Generally, however, if pain commenced after five minutes, in a previously fit man, that was suggestive of angina.

[71] In relation to the medication prescribed by Dr Smith, in 2010, it was the practice to prescribe GTN spray, in that context, as a test of diagnosis. If the GTN spray relieved pain within two to three minutes, then that provided confirmation of the existence of stable angina. Although there had been changes in the recommendations since then, such use of GTN spray was reasonable at that time.

[72] In relation to the fact that Dr Smith had not prescribed beta blockers, Dr Gaskell confirmed that would have been the appropriate course where a patient was taking salbutamol in order to relieve breathing difficulties. There was a risk that, where a patient had asthma, beta blockers would make the condition worse. In a situation where the deceased was "wheezy", it was possible that he was suffering from asthma. His view was that Dr Smith had chosen to avoid the possibility of complicating the presentational picture.

[73] In cross-examination, Dr Gaskell accepted that although the SIGN Guidelines were specifically preferable to the Scottish context, the NICE Guidelines were nevertheless still relevant. He had relied on the terms of both the NICE Guidelines and the SIGN Guidelines in his own report.

[74] In relation to his conclusion that Dr Smith had been entitled to diagnose stable angina, he accepted that, had the deceased's pain been occurring with little or no exertion, then his opinion would have been different. He accepted that if the deceased had been suffering pain after getting dressed, that would amount to minimal exertion, which would suggest a diagnosis of ACS, requiring immediate referral to hospital for same day assessment. Such a set of facts would be regarded as a major warning.

[75] He confirmed that it would be normal to ask a patient to return, for early review, if symptoms worsened. In other words, it would be normal to give the patient "worsening advice". He would have expected that advice to have been given on each of the consultations of 29 December 2010, 31 December 2010 and 5 January 2011. That was normal practice. In the context of this case, he had simply assumed that it had been done. Although it was usual to do it, in his view it was not something which was normally recorded. At paragraph 4.18 of his report, he had stated his conclusion that Dr Smith had acted as an ordinarily competent doctor exercising reasonable care. In response to a question as to whether in coming to that conclusion, he had included consideration of the need to give "worsening advice", his response was that, in his view, the giving of such advice was not a requirement for the purposes of ordinary competence. It was normal, but was not a 100 per cent requirement, particularly in circumstances where GPs often worked under pressure.

[76] In relation to the consultation of Friday 31 December 2010, his understanding was that Dr Smith had placed stable angina at the top of her list of possible differential diagnoses.

[77] In relation to the SIGN guidance that beta blockers should be used as a first line therapy, he agreed with that approach, but qualified it to the extent that such an approach was only appropriate once a diagnosis had been made.

[78] He did not accept that the prescription of aspirin in such circumstances was the usual practice in 2010. He indicated that the practice had changed since then.

(ii) Accident and Emergency: diagnosis and treatment

[79] (1) Mr Neil Nichol, consultant in emergency medicine at Ninewells Hospital, Dundee, and at Perth Royal Infirmary, gave expert evidence for the pursuers under

reference to his report dated 19 April 2018. Amongst his qualifications, he was the lead author in the chapter "Causes and Pathophysiology of Cardiac Arrest" in the Oxford Textbook of Critical Care (1999 ed).

[80] On the hypothesis that the deceased had been first admitted to hospital on Friday 31 December 2010, suffering from chest pain brought on by minimal exertion, his view was that, since the pain was brought on by minimal exercise, and relieved by rest, the obvious diagnosis would have been one of ACS, which carried with it the risk of sudden death. In these circumstances initial treatment within A&E would have comprised the prescription of aspirin, heparin, GTN spray, together with morphine if the patient was in pain. Thereafter the patient would have been transferred to an acute medical unit within the hospital. On the basis that the presentation indicated that blood flow to the heart muscle was critically impaired, with the risk of progression to acute myocardial infarction (heart attack) or cardiac arrest due to abnormal heart rhythm, the patient would have been kept in hospital. Blood tests would have been carried out, beta blockers and statins prescribed, high blood pressure treated, and an ETT carried out.

[81] If later, say on Sunday 2, or Monday 3 January 2011, the deceased, acting on worsening advice, had been admitted to hospital following attendance on his own account, the treatment would have been the same, although, against a background that he had already been seen by his GP who had given worsening advice, the symptoms exhibited would have presented an even stronger case for admission.

[82] Under reference to the report by Mr Johnson, consultant in emergency medicine, and expert witness for the defenders, dated 22 September 2016, at "Opinion": paragraph (ix), to the effect that:

> "He should also have been provided with appropriate safety netting advice, specifically to ring 999 and return to hospital if chest pain returned and did not respond to GTN and persisted more than 20 minutes or came on frequently or with no exertion.",

Mr Nichol agreed that had the deceased presented at hospital with stable angina then on discharge, he should have been given appropriate safety netting advice. He would have expected the same advice to have been given by an ordinarily competent general practitioner. The requirement to do so had been reflected in standard teaching from the 1980s, and the recommended practice had not changed since then.

[83] He confirmed that the SIGN Guidelines were more specifically tailored for the Scottish context, than the NICE Guidelines. Both sets of guidelines provided recommendations for the management of common clinical conditions, based on review of best practice, on the basis of evidence-based current practice, over a period of up to five years prior to the guidelines coming into force. Their content provided the baseline standard for the appropriate management of the clinical conditions to which they referred.

[84] Under reference to the deceased's hospital records from A&E at Belford Hospital, when admitted at 10.45am on 6 January 2011, it was noted that his blood pressure (170/80) was higher than the norm (120/80). The explanation for that was that blood pressure readings were variable in any event, but were influenced, in particular, by stress, pain, and anxiety, or even simply by the experience of being in hospital. The fact that the deceased was experiencing chest pain on admission would have been an explanation for his elevated blood pressure. Where stenosis (narrowing of the coronary artery) was present, there was a danger associated with raised blood pressure. The effect of stenosis would be to increase the work rate of the heart, and therefore increase the necessary oxygen delivery requirement. Where there was stenosis, the supply of oxygen would be inadequate, which could lead to

ischaemia and chest pain, in turn increasing blood pressure. Against that background, the deceased's blood pressure could have been raised simply by walking up the steps to enter A&E at Belford Hospital.

[85] In Mr Nichol's view, the deceased would have been kept in hospital on admission because of his high risk. The core aims would have been to seek to avoid such stressors, and indeed anything involving an increase in heart work rate, particularly in the form of exertion. Mr Nichol's view was that, had the deceased been kept in hospital, and received appropriate treatment, and kept at rest, then, on the balance of probabilities, the chances of him suffering a cardiac arrest on 6 January 2011 would have been reduced, but not excluded entirely.

[86] Under reference to the document "Acute Coronary Syndrome", taken from the Patient.co.uk website, there were two types of non-ST-elevation ACS. The first, unstable angina, was recognised where no infarction had occurred, and troponin levels were normal. The second, non-ST-elevation MI (NSTEMI) was recognised where there was a rise in troponin levels.

[87] In cross-examination, Mr Nichol was referred to his report at paragraph 2 "HISTORY - from medical records". On admission to Belford Hospital on 6 January 2011, the ECG carried out had been normal, as was a chest X-ray. Blood samples had been taken for troponin analysis (paragraph 2.5). Troponin is a protein present in the cells of heart muscle. When the muscle is damaged the protein is released and can be measured in blood samples. The resulting values are indicators of the level of damage to the heart muscle.

[88] The reference at paragraph 2.7 to left axis deviation shown on ECG recordings, also found to be present on previous ECG recordings, was a reference to comparison with an ECG recording taken in 2009.

[89] The deceased's troponin level was found to be within the normal range (paragraph 2.8). When asked if that was a reasonable basis for the assumption that no infarction had taken place, Mr Nichol explained that a single measured value of troponin was of limited assistance in that context. The analysis required to be repeated. It was not safe practice to assume heart muscle was intact on the basis of only one measure. While the recorded single value did not indicate myocardial infarction, on the basis of that measure alone, it was not possible to rule out unstable angina.

[90] At paragraph 2.11, Mr Nichol had noted that subsequently, on that date, the deceased had suffered a cardiac arrest at home, had been defibrillated six times, but that ultimately his heart had become asystolic, and he had been pronounced dead shortly after 4.45pm.

[91] When considering what might have happened if the deceased had been first admitted to hospital on Friday 31 December 2010, given his history as at that time, including two prior attendances with his GP, Mr Nichol confirmed that a diagnosis of ACS would have been considered, would have been treated as a presumptive diagnosis, and the deceased would have been prescribed beta blockers, and the case assessed by a senior physician. ACS presented a high risk. Where there was perceived inadequate blood flow to heart muscle, the aim was to seek to prevent progression to heart attack or potentially fatal cardiac dysrhythmia.

[92] He agreed that, in general terms, a heart attack was caused by a clot or plaque causing the heart to be deprived of oxygen. Arrhythmia was a different, but related, causation. It might result from a heart attack or from a reduction in blood flow.

[93] In response to the suggestion that the cause of death had been arrhythmic sudden death in the absence of ACS, Mr Nichol disagreed that ACS had not been present. In his

view it had indeed been present, and the deceased had died because of inadequate blood flow to his heart. Arrhythmia could be triggered simply by myocardial substrate, and in the case of the deceased, at *post mortem*, previous scarring in the heart muscle had been found together with hypertrophy, and a narrowed coronary artery. He accepted, that in order for arrhythmia to be induced, something was required to trigger it.

[94] In that regard, where there was a risk of a clot, in the context of a working or presumptive diagnosis of ACS, the standard approach would be to prescribe medication to reduce the attendant risk. Medication would involve the prescription of aspirin and heparin. Beta blockers would have no effect on clotting.

[95] Where there was not a clot present, aspirin and heparin would have no effect. Beta blockers, on the other hand could assist. They impact on the receptors of heart muscle, to reduce heart rate and contractility, thus reducing work rate and reducing the level of oxygen required.

[96] Under reference to paragraph 5.10 of his report, had the deceased been referred to A&E on Wednesday 29 December 2010 then, in circumstances in which a second troponin test proved to be normal, in conjunction with a normal ECG, it was probable that the deceased would have been discharged home for an ETT to be carried out later in January 2011, having been given appropriate safety net advice.

[97] Under reference to paragraph 5.11 of his report, however, where he had noted the evidence of Mrs Brown, to the effect that, on 30 and 31 December 2010, the deceased had been suffering chest pain occurring on minimal exertion, resulting in a return to his GP, the question of the degree of necessary urgency required to be considered, in the context of the fact that 31 December 2010 was a Friday and the next four days were public holidays. An ETT was a procedure which would not be conducted outwith normal working hours. On

that basis, the deceased would have been kept in hospital, because of his history and the imminent holiday period, until at least Wednesday 5 January 2011 which would have been the earliest date for an ETT to be carried out. Another alternative would have been simply to proceed directly to transfer to an appropriate hospital for angiography. The timing of angiography would be some days after input by an A&E consultant.

[98] If the resultant angiogram had indicated a cardiac problem, then the most likely consequence would have been that the deceased would be referred to the nearest cardiac centre. Meantime, he would have received full anti-anginal therapy. An identified problem would most likely be treated by dilating the area of restricted blood flow, and implanting a stent appropriately, all of which could be done simultaneously during angiography.

[99] Although Mr Nichol indicated that he would defer to a cardiologist as to the cause of death, he was clearly of the view that beta blockers would have served to reduce the risk of abnormal heart rhythm.

[100] He disagreed with Mr Johnson's view that if the deceased had attended hospital on Friday 31 December 2010, been found to have normal troponin levels, and a normal ECG, had been prescribed aspirin, beta blockers, heparin, and had been given safety net advice, then, rather than being admitted, if he was asymptomatic, the hospital would have discharged him home to rest. The basis of Mr Nichol's disagreement was that the deceased's symptoms of experiencing pain at rest and on minimal exertion had been subject to rapid progression. If, on the other hand, the deceased at that time, had been in a functioning state such as to be able to walk without chest pain, then in these circumstances, discharge home might have been a possible consideration.

[101] At A&E, it had been noted that the deceased was suffering pain at rest on Tuesday 4 January 2011. It was already known that the deceased had suffered pain on minimal

exertion, in failing to reach his garden gate. These were circumstances of minimal exertion. In the context of the evidence of Mrs Brown that, after Hogmanay, the deceased, although having to stop regularly, was able to walk a quarter of a mile, Mr Nichol's view was that in the context of covering 440 yards, but having to stop a number of times because of pain, such activity should be properly characterised as minimal exertion.

[102] In re-examination, he confirmed that stenosis of the coronary artery, resulting in a reduced blood supply to the heart, would cause pain and could trigger arrhythmia, leading to death. The onset of arrhythmia required a trigger, which could be a clot or something else. Where there was no clot, the prescription of aspirin would make no difference, but the prescription of beta blockers would be effective. Bed rest in hospital would be indicated regardless of any trigger.

[103] Discharge from hospital would be appropriate only where the patient was able to exercise and remain pain free. Given the known extent of the deceased's stenosis, the question of whether appropriate drug therapy would have made him sufficiently pain free to allow his discharge, could have been answered only after a trial period to assess his functioning.

[104] He confirmed that the prescription of aspirin, including in general practice, for cases of ACS was standard practice for some years prior to 2010.

[105] (2) Mr Graham Johnson, consultant in emergency medicine at St James's Hospital, Leeds, was interponed to give expert evidence on behalf of the defenders, under reference to his report dated 22 September 2016.

[106] He agreed with Mr Nichol that, on the assumption that the deceased had attended hospital some time after 4.30pm on Friday 31 December 2010, presenting with pain in his chest on minimal exertion, which resolved with rest, then, at A&E, a presumptive diagnosis

of ACS would have been appropriate. He agreed that the appropriate treatment would have been further investigation in the form of an ECG, and the taking of troponin levels, the prescription of aspirin or other anti-platelet therapy, and heparin as an anti-coagulant agent. Beta blockers would not have been appropriate where COPD was a factor. Pain relief would have been prescribed as appropriate.

[107] With such a presenting history, suggestive of ACS, the appropriate treatment plan would have involved referral to a cardiologist or, in the case of Belford Hospital where no cardiologist was available, to the appropriate senior physician on call. An appropriate plan would have been to admit the deceased, not necessarily for bed rest but to exclude exertion, with ongoing review of medication. The intention would be to confirm coronary heart disease by means of an ETT, and thereafter to determine its severity. Where there was an abnormality, the intention would be to define by angiogram, in a cardiology department, the position of any blockage in the coronary artery. In circumstances where there was no local cardiology department and, because of the time of year, a resultant delay in carrying out an ECG, emphasis would be placed on assessing the appropriate necessary medication, prior to confirmation of the diagnosis. In that regard, beta blockers would be expected to reduce chest pain in a period of 24-48 hours. The anticipated effect of aspirin would be on a similar timescale.

[108] In a normal week, the deceased once in hospital, would have been retained as an in-patient until an ETT had been carried out. In the particular circumstances of his case, where the weekend was imminent and where in addition, because of the time of year, public holidays would intervene, where an ECG could not be carried out before the elapse of say, two to three days, then it was likely that he would have been sent home. That would have been subject to an assessment of how he was responding to medication. He would have

been discharged home only on the basis that as a result of the medication, his suffering had been reduced to an acceptable level.

[109] The incidence of ACS was relatively common. It occurred in two to four per cent of patients subject to investigation for possible cardiac chest pain.

[110] Although there were three main types of ACS, all presenting with different risks, the deceased's presentation was either non-STEMI ACS, or unstable angina. Which diagnosis was appropriate was dependent on troponin results. In the case of both there was a recognised variable potential to progress to myocardial infarction and death. The risk was broadly one of five per cent over 14 days.

[111] In re-examination, Mr Johnson accepted that although that risk was statistically relatively small, it was nevertheless significant, since it involved the risk of death. It was not a risk to be taken lightly.

[112] He accepted that where no COPD was present, beta blockers were an appropriate medication. That would be the case even where the patient, in the absence of COPD was "wheezy". In any event, even if there were contra-indicators, alternatives to beta blockers were available. He agreed that the alternatives of rate limiting calcium channel blockers, long-acting nitrates or nicorandil, were all reasonable alternatives.

[113] On the basis of the *post mortem* results, Dr Johnson's view was that had the deceased undergone an ETT, its results were unlikely to be normal or, expressed differently, were likely to be abnormal, and would have triggered referral for angiography.

(iii) Cardiology

[114] (1) Professor Stephen Brecker, Chief of Cardiology at St George's University Hospitals NHS Foundation Trust and St George's, University of London, and Consultant

Cardiologist at St George's Hospital, London, gave expert evidence on behalf of the pursuers, under reference to his report, dated 24 April 2016.

[115] Both he and Professor Channer, the expert witness on cardiology for the defenders, at a joint meeting held in London on 23 November 2017, had agreed, in terms of their joint report dated 28 November 2017, that the deceased's death was caused by changes in the rhythm of his heart, leading to ventricular fibrillation, possibly having involved preceding patterns of ventricular tachycardia or arrhythmia. The likely cause of the ventricular fibrillation was ischaemia, caused by reduced blood supply to the heart muscle, brought about by stenosis. All of that had been the subject of agreement between Professor Brecker and Professor Channer.

[116] They did disagree, however, on the issue of the diagnosis which should have been made in advance of death. Although they agreed that the deceased had been suffering from ACS, in the form of unstable angina, they disagreed on the timing of the commencement of the condition.

[117] Under reference to the document "Acute Coronary Syndrome", located on the Patient.co.uk website, Professor Brecker confirmed that there was a spectrum of clinical presentation of coronary disease, which included asymptomatic conditions, stable angina, or acute coronary syndrome (ACS). Of the two principal classes of ACS the one which was pertinent to the deceased's case was non-ST-elevation ACS (NSTE-ACS), which was further divided into (a) non-ST-elevation MI (NSTEMI), characterised by elevated troponin levels, and (b) unstable angina, characterised by relatively normal troponin levels.

Professor Brecker and Professor Channer had agreed that, by Wednesday 5 January 2011, the deceased had been suffering from unstable angina.

[118] In the context of the attendances made by the deceased on Dr Smith, three scenarios were put to Professor Brecker:

1. The deceased was referred by his GP for same day assessment to Belford Hospital on Friday 31 December 2010:

In such a situation the standard treatment would have included assessment in A&E, including if required, contact with the intake medical team and a cardiologist if one was present. A diagnosis of ACS should have been made. In a situation where unstable angina was diagnosed at A&E, but no cardiologist was present, then a referral would have been made to the senior physician, for confirmation of the diagnosis as between NSTEMI and unstable angina. The deceased would have been admitted unless, on appropriate medication, he would have been able to exercise without pain. In the case of the deceased, that would have been unlikely in circumstances where, on the basis of the *post mortem* results, he was at that time suffering from severe main stem coronary artery stenosis. That would have made it unlikely that he could exercise even only to the extent of minimal exertion, without pain. On referral to a senior physician, the deceased would have been admitted, intravenous access obtained, and anti-platelet and anti-thrombotic drugs administered, together with aspirin, or an appropriate alternative, to put in place dual anti-platelet therapy. It would have been standard practice to prescribe beta blockers in order to reduce his cardiac workload. He would also have been prescribed vasodilators, to increase blood supply, by widening the coronary artery and reducing demand on the heart. In this context, nitrates and calcium channel blockers would have been prescribed in addition to beta blockers. If the deceased had been continuing to experience pain at rest, intravenous nitrates would have been

administered. In addition, he would have been given injections of anti-thrombotic drugs, such as heparin or similar, and would have been started on other drugs with longer term effects, such as statins to reduce cholesterol levels, and vasodilators such as ACE inhibitors. All of that would have been standard practice. Had the deceased received such treatment on Friday 31 December 2010, it is likely that significant difference would have been made to the supply to and demand on his heart, and that his level of ischaemia would have been reduced.

2. The deceased was admitted to Belford Hospital on Saturday 1, or Sunday 2 January 2011, having previously received, and acted upon, safety-netting advice given on Friday 31 December 2010:

In this situation, where the deceased had presented himself to hospital, there would be no difference from the first scenario. He would have received the same treatment, with the same effect. The likely outcome would have been the same, resulting in a reduction in ischaemia over the course of the treatment. A further factor to be taken into account was that, following admission to hospital, the deceased would have had several days of treatment involving a combination of therapy and rest in hospital, as a consequence of which, in Professor Brecker's opinion, the likelihood of ventricular fibrillation, would, on the balance of probabilities been removed. The combination of all of these measures would have removed the ischaemic provocation, which resulted in ventricular fibrillation, or would have significantly relieved it. In that context, where stenosis was present, another factor was generally required to trigger arrhythmia and cardiac arrest. It was not always possible to identify what was that triggering factor. Ventricular fibrillation was not always predictable. It was predictable in patients known to be at risk, but the provocative event triggering it

might not be discernible. In the deceased's case there had been sufficient substrate, in the form of stenosis, to trigger ischaemia and sudden death.

3. The deceased was admitted to Belford Hospital, following referral by his GP on Wednesday 5 January 2011, with a diagnosis of unstable angina:

In that scenario, the treatment would again have been the same as that in respect of the first scenario. The difference however between the third scenario and the first two scenarios related to the likely outcome. In the first and second scenarios, the outcomes were dependent upon the presence of an appropriate therapeutic level of drugs in the deceased's blood stream, and the availability of sufficient time for these medications to take effect. On the basis that such an appropriate period of time was available, these drugs would have reduced his level of ischaemia. As far as the third scenario was concerned, where there was a more limited period of time available, until the critical date of 6 January 2011, it was not possible to say that the risk of ischaemia could have been avoided. The risk of ischaemia would have been reduced, but it would not be possible to conclude that in a limited timescale the medications administered would have achieved their maximum effect. The drugs would have had some effect, which would have been more than negligible. The effect of the medication would have been not insignificant, but it was not possible to say its effect would be more than 50 per cent likely to have achieved its desired aim.

[119] In a situation where the deceased's condition had been deteriorating over several days, these several days would have been available for the appropriate drugs to take full effect. Therefore, had he been admitted on Friday 31 December 2010 or Saturday 1, or Sunday 2 January 2011, ventricular fibrillation would probably have been avoided. That was to be contrasted with the scenario in which he had been admitted on 5 January 2011, in

the context of which all that could be said was that there would have been a continuing reduction in his level of ischaemia. It was also significant that, in relation to the first and second scenarios, at the time when medication would have been commenced the deceased would have been in better physical condition, and would have responded better to medication than would have been the case by Wednesday 5 January 2011, by which time he would have deteriorated and would have been experiencing more frequent and prolonged pain.

[120] Professor Brecker fundamentally disagreed with Professor Channer's view that the deceased had been suffering from stable angina on Wednesday 29 December 2010, progressing to unstable angina on Wednesday 5 January 2011. In so far as the deceased's condition on Wednesday 29 December 2010 was concerned, it was not possible to characterise it as stable where there had been no known period of stability. Such a diagnosis could only be made in circumstances whereby it was known that the condition had not changed over time. It was not possible to characterise a condition as stable in a period of only a few days. A better description would have been to characterise the condition as "recent onset angina". But notwithstanding that, however, where it was plain that pain was brought on by minimal exertion, the correct diagnosis was unstable angina.

[121] Professor Brecker did not accept a progression which began with stable angina, developing into unstable angina, and resulting in death within a matter of days. In the context of ACS, a better and more logical analysis was that the angina being suffered by the deceased had been unstable from its commencement. It was relevant to note that patients with stable angina could remain stable for years. The fact that the deceased had previously been fit and healthy was a further significant factor. Given his prior state of health, it was clear that, in relation to his heart function, something had changed.

[122] Professor Brecker adhered to the terms of his report, dated 24 April 2016. Nothing had come to his attention, since then, which had altered his view as there expressed.

[123] He accepted the view expressed by Professor Pounder, consultant pathologist, to the effect that the deceased's fatal cardiac event had not had a thrombotic cause, but rather was the result of vasoconstriction, in combination with a provocational trigger, as being very plausible. He also accepted that exposure to cold could have constituted the provocational trigger. It was recognised that cases of stable angina in which pain was brought on by exertion, specifically in cold weather, were common. Further, had the deceased been admitted to hospital at an earlier stage, he would not have been exposed to cold, the provocation to vasoconstriction would have been removed and, as a result, in all probability, there would have been no ventricular fibrillation. He confirmed however that although he regarded the suggestion by Professor Pounder as a plausible explanation, it did not alter his own opinion.

[124] In cross-examination, Professor Brecker was referred to the A&E notes relating to the deceased's attendance at Belford Hospital on Thursday 6 January 2011. The noted history indicated that there had been one week of chest pain induced by exercise, following an episode of flu-like symptoms. Pain had been much worse "last night", when at rest. Professor Brecker accepted that the terms of the history indicated a change in symptoms, from what had previously been the case. He attached importance to what had been noted, as the described symptoms were significant and plainly worse than had been the case. Where there was pain at rest, it would be appropriate to consider the possibility of myocardial infarction.

[125] He did not consider that the described symptoms were such as to raise the immediate possibility of coronary vaso-spasm. That was on the basis that pain at rest was a

classic symptom of ACS which would prompt consideration rather of coronary artery disease. In relation to that, on the suggestion that there was a dispute between him and Professor Channer in relation to whether the deceased's condition had been ACS, Professor Brecker indicated that not to be his understanding. Professor Channer had told him that his view was that the deceased had been suffering from ACS on Wednesday 5 January 2011.

[126] Pure coronary spasm was a rare condition in the absence of coronary disease. Against that, however, cardiac pain at rest was a recognised classic symptom of ACS. Although spasm was an alternative possible cause of cardiac chest pain at rest, Professor Brecker doubted that, clinically, a differentiation could properly be made between cardiac pain at rest in the absence of ACS, and unstable angina.

[127] Under reference to Professor Channer's report, dated 22 February 2016, Professor Brecker considered that it was possible that Professor Channer may have been using the term "ACS" to describe only troponin positive syndromes, and was removing unstable angina from that class of conditions. Be that as it may, unstable angina, involving normal troponin levels and chest pain was generally considered to be part of the recognised category of ACS.

[128] Professor Brecker did not accept that his opinion, on the possibility of the deceased surviving with appropriate treatment, was predicated on the cause of death having been caused by a blood clot. Rather, his view was that death had been primarily caused by ischaemia in conjunction with other factors. It was significant that no clot had been found *post mortem*. Notwithstanding that, it was possible that there had been microscopic clots which had contributed to death, but Professor Brecker's opinion did not depend on that having been the case. In his view the primary cause was ischaemia.

[129] Under reference to the joint report by Professor Brecker and Professor Channer dated 28 November 2017, at Question 4, Professor Brecker had referred to an article "The Pathophysiology of Acute Coronary Syndromes", by Davies, in which it was stated:

> "A significant proportion, but not all, of samples from unstable angina contain thrombus, while most samples from stable angina, but not all, do not contain thrombus. The absence of thrombus in unstable angina is in part related to the time delay between acute symptoms and atherectomy."

Whereas it appeared that Professor Channer was excluding unstable angina on the basis that no thrombus was present, Professor Brecker had referred to the article, as support for his opinion that the deceased had been suffering from unstable angina, on the basis that it indicated recognition, following research, that in ACS, presenting as unstable angina, thrombus need not necessarily be present. On that basis, Professor Brecker's opinion was not dependent on the presence of thrombus.

[130] Against that background, at paragraph 6 of the joint report, Professor Channer had stated that, on Wednesday 5 January 2011, the deceased had reported pain when at rest, which would be compatible with the diagnosis of unstable angina. Further, at Question 8, Professor Channer had stated, in relation to the attendance at A&E on 6 January 2011:

> "Troponin was negative so the final diagnosis of 6 January 2011 was *troponin negative unstable angina*. An elevated troponin level would be expected in patients suffering from an ACS."

On the basis of these two contradictory positions, Professor Brecker characterised Professor Channer's approach as being inconsistent. Professor Channer's approach appeared to be that unstable angina should be excluded where there is no thrombus, but also that, notwithstanding the absence of thrombus, the deceased was suffering from unstable angina.

[131] Under reference to an article relied upon by Davies, as support for the statement in his article, Professor Brecker accepted that the evidence justifying Davies' conclusions had been based on conditions in living patients. It was stated in the article relied upon by Davies that:

> "There are a number of possible explanations for the absence of thrombus in atherectomy samples taken from unstable angina patients. One explanation is that pure coronary spasm may have been responsible for the angina."

Another explanation was that, over the passage of time, the body was able to break down a thrombus. Professor Brecker accepted that such a situation was different from that where there was *post mortem* examination following recent death, in the course of which it would be routine to identify any blood clots. Although Professor Brecker accepted that the statement in the Davies article was based on research involving living patients, he maintained that there could be many explanations why no thrombus might be present in cases of angina. Clearly the question of time-interval absorption by the body could not apply where death occurred, but other explanations such as pure coronary spasm or a clot missed in the course of *post mortem* examination, were other possible explanations.

[132] Professor Brecker accepted that in a situation where no clot had been present, prescription of aspirin would have had no effect. In such circumstances, he also accepted that blood thinning products would have had no effect on the deceased. He maintained, however, that even where there was no blood clot, the prescription of beta blockers would have had some immediate anti-arrhythmic effect but, in particular, would have served to reduce cardiac demand and workload.

[133] He accepted that it was possible for ventricular fibrillation to occur without a provocational trigger. In the circumstances of the deceased's case, in the absence of a clot, a provocational trigger could have been constituted by what appeared to be a clear substrate

for ischaemia, that being 70 per cent stenosis of the left main stem coronary artery in conjunction with pain on minor exertion. Such facts would tend to suggest a conclusion of resultant ischaemia. In the circumstances of sudden cardiac death, it was logical to link ischaemia as the causative factor for ventricular fibrillation. Where there was 70 per cent stenosis of the left main stem coronary artery, significantly reducing supply to the left ventricle, it would not be reasonable to suggest that stenosis and the resulting ventricular fibrillation were not related. In general, left main stem disease would be regarded as a red flag warning to any cardiologist.

[134] In considering whether beta blockers would have impacted on the effect of stenosis, Professor Brecker took into account the high level probability that ischaemia had triggered the ventricular fibrillation. Where ischaemia was brought about by stenosis, whether or not in circumstances involving a blood clot or spasm, beta blockers would serve to reduce the level of ischaemia by reducing the cardiac workload. In his view, the administration of beta blockers would have served to reduce the overall level of ischaemia to significant effect.

[135] Professor Brecker accepted Professor Channer's view that beta blockers would have no effect on spasm as a causative factor for ventricular fibrillation. It was significant, however, that the administration of nitrates can relieve spasm.

[136] Professor Brecker differed from Professor Channer in his stated view that the only mechanism for cardiac chest pain at rest, absent ACS and unstable angina, was spasm. In Professor Brecker's view that was but one mechanism. Relevant texts all indicated that ACS could be defined by coronary pain at rest. Where there was cardiac chest pain without coronary disease, the appropriate descriptor was "vasospastic angina". In the case of the deceased, however, it was known that he had coronary disease.

[137] It was not possible to offer an opinion on the isolated effect of beta blockers on patients with ACS because all of such patients would necessarily be simultaneously prescribed all the other necessary medications to treat their condition. It remained Professor Brecker's view, however, that the medication which the deceased would have been prescribed in hospital, as treatment for ACS, together with the fact of simply being in hospital, would, on the basis of his clinical experience and on the balance of probabilities, have prevented ischaemia and therefore ventricular fibrillation. He accepted that, given the limitation on assessing the effect of beta blockers in isolation, it was at least theoretically possible that such patients would survive in any event, even if beta blockers had not been prescribed. He thought that more likely, though, where a presentation different to that of the deceased was the case. Here, the deceased had the classical substrate for ischaemia and sudden death, in the form of left main stem coronary artery stenosis. That comprised the condition clinically most associated with sudden death, and indeed was known colloquially in the profession as "the widow maker".

[138] In so far as the timing of the effect of medication was concerned, Professor Brecker considered that the relevant period was probably one of about three days. Thus, if the deceased had been admitted to hospital on Friday 31 December 2010, death on Thursday 6 January 2011 would have been avoided. Where the effect of medication had been over the period of only one day, it was not possible to say that the medication would have made a 50 per cent difference. Although, after administration, drugs such as aspirin, beta blockers and nitrates would have had a material effect on the level of ischaemia within hours, on the balance of probabilities about three days would have been required as the minimum period to achieve alteration of the eventual outcome.

[139] On the hypothesis that the deceased was suffering from ACS on Wednesday 29 December 2010, the differences between Professor Brecker and Professor Channer, as to the probable cause of ACS in his case, were set out in their respective answers to Question 7 in their joint report dated 28 November 2017.

[140] Professor Brecker accepted that it would not have been appropriate to administer intravenous nitrates in hospital where the patient was pain free at rest. If a patient presented with a history of pain at rest, intravenous administration of nitrates would be appropriate on admission, but subject to discontinuation when the patient became pain free.

[141] In re-examination, Professor Brecker noted that Professor Channer had stated in his report, dated 22 February 2016, at paragraph 6.1, that the deceased had died following cardiac arrest after a short history of unstable angina. Unstable angina was a form of ACS.

[142] In their joint report, in answer to Question 8, Professor Channer had stated that the deceased's history at Friday 31 December 2010, of exercise related chest pain, was compatible with a diagnosis of angina, and that his reported chest pain at rest on 5 January 2011 was compatible with a diagnosis of unstable angina. Professor Brecker agreed that recognition of unstable angina was a clinical diagnosis rather than a pathological one.

[143] Although Professor Channer held the view that the deceased's *post mortem* examination excluded ACS, he had also stated that the deceased was suffering from unstable angina. Professor Brecker's response was that although, at times, Professor Channer appeared to seek to exclude unstable angina from the general class of ACS , equally his position seemed to be that since there was no ACS, there was no unstable angina.

[144] Professor Brecker confirmed that his opinion was not dependent on the existence of a thrombus. Rather, his view was that the deceased had been suffering from ACS and that

appropriate treatment would have prevented ischaemia and therefor ventricular fibrillation. In his answer to Question 4 of the report of his joint meeting with Professor Channer, Professor Brecker had stated that, although in ST-segment elevation myocardial infarction (STEMI) one would expect to find thrombus, in unstable angina thrombus may or may not be present. In that respect, the absence of thrombus was not conclusive. It was also significant that thrombus was something discoverable only *post mortem*. It was not possible to know of the existence of thrombus in life.

[145] It was significant that in his experience, with appropriate treatment, the majority of patients presenting with left main stenosis and ventricular fibrillation survive.

[146] (2) Professor K S Channer, Honorary Professor of Cardiovascular Medicine, Sheffield Hallam University, Consultant Physician and Cardiologist, gave expert evidence for the defenders under reference to his report, dated 22 February 2016, and the report of the joint meeting between him and Professor Brecker, held on 23 November 2017.

[147] He was asked to consider a set of circumstances in which, at some time between late afternoon on Friday 31 December 2010 and midnight on Sunday 2 January 2011, the deceased had been admitted to hospital with a diagnosis of ACS. He accepted that in such circumstances, standard treatment would have been as described by Professor Brecker, including the administration of beta blockers. On that basis, where the risk was of myocardial infarction, caused by a blood clot, beta blockers would have been given early in order to reduce the risk of death by 10-15 per cent, but the benefit was not acute and risk reduction occurred only after 15 days. If the risk had been ventricular fibrillation, then there was evidence that beta blockers did not significantly reduce risk until the elapse of six days from the occurrence of myocardial infarction. Reference was made to the MIAMI study, reported in the European Heart Journal (1985). Where cardiac arrhythmia had occurred, in

the absence of myocardial infarction, there was no evidence that beta blockers would reduce the risk of sudden death, except in cases of a severely damaged heart.

[148] In cross-examination, Professor Channer stated that he had retired from clinical practice in the NHS in April 2012, and that since 2014 he had been engaged only in medico-legal matters. He had not been involved in the clinical treatment of NHS patients for over six years. He was instructed in medico-legal matters principally on behalf of defenders. Over a recent period of 12 months, less than 20 per cent of his reports had been instructed on behalf of claimants. He accepted that he had a reputation for being supportive of doctors, and that he sat on the Council of the Medical Defence Union. He accepted that a style report, produced by him, and dating from 2012, but similar to the report prepared by him for the present case, was available on the internet, but that he had not published any corresponding style report appropriate for a claimant's case.

[149] He accepted that unstable angina was a form of ACS, or at least, was one of the manifestations of ACS. Under reference to the paper "Acute Coronary Syndrome" available on the Patient.co.uk website, a paper he had not previously seen, he accepted that unstable angina was characterised by normal troponin levels.

[150] In his view, ACS involved acute pathology, most commonly in the form of a ruptured plaque in the coronary artery, with associated thrombus. ACS could be caused by unstable angina, involving pain at rest, but could also be caused by coronary artery disease in association with thrombus. There were two types of unstable angina. In terms of the NICE Guideline 2010, as set out in the report of the joint meeting between him and Professor Brecker:

> "The term 'acute coronary syndrome' encompasses a range of conditions from unstable angina to ST-segment elevation myocardial infarction (STEMI), arising from thrombus formation on an atheromatous plaque."

ACS was therefore a coronary event involving thrombus formation on plaque rupture. He did not agree that ACS was a clinical diagnosis rather than a pathological one. When referred to the joint report, where the NICE Guidelines, at paragraph 1.2.1.3, were quoted to the effect that, of the symptoms which may indicate ACS, one set was:

> "New onset chest pain, or abrupt deterioration in previously stable angina, with recurrent chest pain occurring frequently and with little or no exertion, and with episodes often lasting longer than 15 minutes.",

he agreed that, on that basis, unstable angina was a clinical diagnosis based on exhibited symptoms.

[151] Where, as in the present case, at *post mortem* examination there had been no evidence of thrombus in the coronary artery, there was no pathology for the most common cause of unstable angina. Nevertheless, it was possible that the deceased could have been suffering from unstable angina by Wednesday 5 January 2011, without such pathology. Where that was the case then, prior to that date, the deceased would have been suffering from recent onset angina. His understanding was that the deceased's unstable angina, involving pain at rest, had developed on 5 January 2011, and that before that his angina had been brought on by exercise. With hindsight, he considered that when the deceased attended his GP, complaining of chest pain on exercise, on Wednesday 29 and Friday 31 December 2010, he had been suffering from angina. He characterised pain on minimal exertion as angina, rather than unstable angina. Where "crescendo" angina was experienced, the condition fell to be characterised as unstable angina.

[152] At paragraph 2.6 of his report, Professor Channer had noted on that, on Friday 31 December 2010, the deceased had reported chest pain which was sharp and retrosternal, lasting five minutes, precipitated by walking. He had assumed that what was being

described was normal walking. In contrast, what Dr Smith had noted at the consultation was "chest pain, as last Wednesday - went for a walk yesterday - sharp retrosternal pain after 5 mins". He accepted that what had been recorded was pain after five minutes of walking, rather than pain lasting five minutes. When asked whether he would define walking for 5 minutes as minimal exertion, his response was that any answer would depend on the definition of "minimal". He accepted that unstable angina was defined by pain at rest, or pain of a crescendo pattern, which was getting worse. His preference was to describe the condition of experiencing pain after minimal exertion as new onset exercise related angina. He accepted that a pattern of changing pain on walking was crescendo in nature. Where the pattern of pain changed on exercise, and became crescendo in nature, that fell within the pattern of unstable angina.

[153] Under reference to his report at paragraph 3.6 "Unstable angina or acute coronary syndrome", he had characterised ACS as being caused by a sudden reduction in coronary blood flow because the narrowed artery had been intermittently blocked by a thrombus or clot attached to the atheromatous deposit or plaque, resulting in the heart muscle being acutely starved of blood.

[154] At paragraph 3.7, under reference to the European Society of Cardiology ("ESC") ACS guidelines 2007, he had stated that in most cases of unstable angina, the chest pain occurs at rest, and that, by definition, the pain must last for more than 20 minutes for the diagnosis of unstable angina to be accepted. When referred to the ESC ACS guidelines 2007, at page three, he accepted that the reference to persistent pain lasting more than 20 minutes was in relation to ST-elevation ACS which was not the condition from which the deceased had been suffering. At page six, paragraph 4.1, of the ECS ACS guidelines 2007, the clinical presentation of NSTE-ACS was described as including

prolonged (more than 20 minutes) anginal pain at rest, but also three other separate sets of exhibited symptoms. His explanation for not including the other three presentations at paragraph 3.7 was that the most common presentation involved chest pain at rest for more than 20 minutes. He characterised the other presentations as being not common. He accepted that in considering unstable angina, he would not exclude the other presentational patterns. He accepted that the deceased had been suffering from severe angina. He accepted that, since that was one definition of unstable angina, the deceased had been suffering from unstable angina.

[155] At paragraph 3.14 of his report, he had stated that in about two thirds of cases of sudden coronary death, the death had occurred as a consequence of an acute coronary syndrome, having occurred in the early stage of a thrombotic occlusion of the artery. He accepted that only in the last sentence of the paragraph had he stated that, overall, sudden death is the first and fatal manifestation of coronary artery disease in about one third of cases. He confirmed that such cases would involve no evidence of thrombus, and that death most probably resulted from malignant arrhythmia.

[156] At paragraph 4.19 of his report, in relation to the question of whether Dr Bawa had been negligent in discharging the deceased from hospital on Thursday 6 January 2011, while he considered that a diagnosis of unstable angina would have been reasonable, his opinion, nevertheless, was that she had not breached her duty of care by discharging him. That was contrary to the opinions of Dr Johnson and Mr Nichol, both accident and emergency consultants, and contrary to an admission of liability, made earlier in the case, by Dr Bawa's employers. Against that, Professor Channer maintained that he had reached his own opinion on the matter.

[157] He agreed that the case of the deceased involved a longstanding condition comprising stenosis of the left main stem coronary artery, left ventricular hypertrophy, and scarring from previous endocardial changes, in an active man who had previously lived asymptomatically, impacted by a trigger which had caused arrhythmia, leading to death, in the absence of thrombus. He agreed that in those circumstances, an appropriate trigger was necessary for death to result. He agreed, in particular, that the condition had progressed to ischaemia and that something would have been required to trigger arrhythmia. He accepted that although thrombus was one possible trigger, it was not the only cause, and that one third of comparable cases had other pathology. He accepted that the four precipitating factors identified by Professor Pounder, being exercise, significant emotion, exposure to cold, and ingestion of a large meal, were factors which could promote the occurrence of unstable angina.

[158] On the hypothesis that the deceased had been admitted to hospital with unstable angina, notwithstanding a normal ECG result and negative troponin, the standard treatment to be expected would have involved the administration of aspirin, anti-platelet medication, heparin statins, beta blockers and nitrates. He accepted that beta blockers have an anti-ischaemic effect, the maximum effect of which occurs within two to three days of administration. Calcium channel blockers would not have been administered unless there was a contra-indicator for beta blockers. Beta blockers reduced demand on the heart, correcting any supply/demand imbalance likely to cause ischaemia. Pain relief would have reduced the risk of arrhythmia where the patient was experiencing continuous pain. Pain was associated with sympathetic activation that caused vasoconstriction and increased the workload of the heart (ESC-AMI guidelines 2012, at paragraph 3.2). Accordingly, the administration of pain relief would reduce the risk of ischaemia. He accepted that a

combination of all of the above treatments would have had the effect of reducing ischaemia which in turn would have reduced the risk of arrhythmia.

[159] Whether any failure to provide such treatment in the case of the deceased had contributed to the development of ischaemia was dependent on how long he might have been in hospital. Where an ETT or angiogram had disclosed a significant problem, he would have been referred for surgery and would most likely have survived. His experience was that, of patients presenting with stressors similar to those impacting on the deceased, who received such treatment, the majority would survive. Given the obvious prohibition on an unethical trial, there was no evidence to indicate the proportion of such patients who would survive without such treatment.

[160] In re-examination, on the hypothesis that the deceased had received such treatment on the basis of a diagnosis of ACS, in circumstances where in fact he was suffering from a different condition, Professor Channer's view was that in circumstances where the standard treatment for ACS was administered, all of the drugs prescribed to treat thrombus, that is to say aspirin, heparin and anti-platelet therapies, would have had no effect. That would leave anti-ischaemic drugs such as beta blockers, and nitrates which were vasodilatory. If the deceased had presented at hospital on Friday 31 December 2010, Professor Channer anticipated that if he was experiencing severe exercise related pain, which came on early but without pain at rest, then the clinicians would be looking for ACS confirmation by means of ECG and troponin testing. If these tests had produced abnormal results he would have been admitted. His presentation would have suggested cardiac pain but the employment of these risk stratification tools would have been necessary for a fuller understanding of his condition.

[161] Where there was no diagnosis, and he might have been suffering chest pain for some reason other than ASC, then, subject to a medical test, he would have been treated on a putative diagnosis of angina, but not kept in hospital if his ECG and troponin levels were normal. Such results would have indicated a low risk.

[162] In the absence of a blood clot, a possible pathological mechanism which could have resulted in the necessary provocation to induce arrhythmia could have been the blocking of the critical artery by spasm, which could have occurred spontaneously at the site of narrowing of the artery. Such a spasm would lead to ischaemia, which itself would lead to ventricular fibrillation. It was possible that such a spasm could be caused by exposure to cold, especially on the face, but it could also be caused by sympathetic aggravation brought about by the experience of, for example, anger or excitement.

The pathological perspective

[163] Professor Derek Pounder, Professor Emeritus of Forensic Medicine, at Dundee University (retired) gave expert evidence for the defenders, under reference to his report dated 10 March 2018. Under reference to the report of the *post mortem* examination carried out on the body of the deceased, Professor Pounder confirmed that, of the two authors, he knew Dr Mark Ashton, consultant pathologist, personally, and had no reason to doubt his skill as a forensic pathologist. He confirmed that no evidence of occlusive thrombus was found *post mortem*. The relevant findings in relation to the heart were as follows:

> "The heart was enlarged with mild left ventricular hypertrophy and it weighed 475g. The left main stem coronary artery showed marked atherosclerosis with approximately 70 per cent narrowing and there was also significant narrowing of the proximal anterior descending branch. The left circumflex branch and the right coronary arteries showed mild atherosclerosis. There was no evidence of an occlusive thrombus. The cardiac valves were unremarkable."

He agreed in general with the opinions expressed in the report of the *post mortem* examination.

[164] Under reference to his own report, he confirmed that the assessment of the cause and mechanism of death required consideration of both the clinical history and the *post mortem* findings, and that the core of the clinical history in the present case was exercise induced chest pain for a period of about one week, culminating in an episode of chest pain leading to a cardiac arrest due to ventricular fibrillation. *Post mortem*, there had been evidence of hypertensive heart disease, with increased muscle mass of the main pumping chamber of the heart (left ventricular hypertrophy), resulting from the increased work required to maintain a higher than normal blood pressure. The heart weight of 475g was above the maximum expected and would have resulted in an increased demand for blood flow to the heart muscle (myocardium), and an increased risk of ischaemia (caused by an imbalance between myocardial oxygen demand and supply, resulting in myocardial hypoxia which, in turn, produced electrical instability). The extent of the increased heart muscle mass was not sufficient in itself to constitute a likely cause of ischaemia and electrical instability. In general, a heart weight of 500g or more would be required to firmly entertain that possibility. Notwithstanding that, the left ventricular hypertrophy found *post mortem* in the present case would have exacerbated the risk of ischaemia arising from atherosclerotic coronary artery disease.

[165] He described the standard method of dissection of the heart, and examination of the coronary artery, by slicing at 3mm intervals in order to allow identification of disease in the artery wall or in the *lumen*. The colour of the interior of the artery, being off-white, provided a contrast to the red colour of any clot which might be present. While he accepted that there was always the possibility of human error, in his view, failure to identify a clot which was

present would amount to incompetence on the part of the examining pathologist. On death following a cardiac event, a pathologist would always concentrate on looking for a clot. Clot formation was a potential cause, although never concluded to be *the* cause, unless all other possible factors were excluded. On the other hand, where a clot was found, it would usually be the cause of death.

[166] Where no clot was found *post mortem*, the possible mechanisms of death involving heart disease, particularly in the case of sudden deaths, were coronary artery disease, atherosclerosis, or, generally, chronic longstanding disease without any acute manifestation, such as severe narrowing of the coronary artery, or enlargement of the heart as in the present case. In such circumstances, where such conditions led to arrhythmia, the likely cause would be some dynamic factor imposed on the longstanding abnormality.

[167] Ventricular fibrillation was caused by electrical instability which resulted in disruption of the regularity of the heartbeat as it moved through the heart muscle.

[168] Ischaemia was caused by the imbalance between demand from the heart muscle and the supply of blood flow from the coronary artery. Such an imbalance could occur where increased muscle mass increased demand, in the form of increased rate of heartbeat, in circumstances where, as the result of stenosis in the coronary artery, or the existence of a clot occluding the artery to any extent, there was a reduced blood supply to the heart. In the present case, increased heart size would have resulted in increased demand, which, coupled with the noted stenosis of the coronary artery, which would have reduced blood flow, would have resulted in an electrical imbalance within the heart. Heart muscle would have become ischaemic, through lack of oxygen, resulting in instability which would have affected muscle contraction and led to arrhythmia.

[169] Under reference to his report, he confirmed that where, as here, the *lumen* of the coronary artery was reduced by approximately 70 per cent, it was to be noted that such a figure was at the lower end of the range of narrowing likely, in itself, to account for ischaemia. Having said that, however, the presence of left ventricular hypertrophy increased the likelihood of ischaemia developing. As he put it, these circumstances were certainly enough to account for ischaemia. At a later stage in his evidence, he described these facts as being borderline for the risk of the development of ischaemia, or sufficient to account for ischaemia. His final position was that where these circumstances were present, and the patient was already suffering from angina, then the risk of sudden death by ischaemia was significantly increased.

[170] It was significant that focal scarring was found on the inner surface of the muscular wall of the left ventricle (sub-endocardial fibrosis). The development of such scarring was the result of ischaemia due to reduced blood supply, possibly exacerbated by a rapid heart rate. The presence of the scarring, a form of necrosis, was an indicator of a previous myocardial infarction. Its presence was also significant in that it increased the likelihood of the future development of cardiac arrhythmia, even in the absence of acute ischaemia.

[171] In the absence of a blood clot, the combination of these factors, comprising the presence of focal severe coronary atherosclerosis in the left main coronary artery, left ventricular sub-endocardial scarring from previous myocardial infarction, and left ventricular hypertrophy, were sufficient anatomical findings to account for myocardial ischaemia and an ischaemic sudden death consequence upon an arrhythmia, all as suggested by the given history of collapse in the case of the deceased.

[172] On the other hand, it was significant that, at *post mortem* examination, there was an absence of evidence of fresh myocardial infarction which was in keeping with the known

normal ECG result and the negative troponin levels recorded in hospital some hours before death. No thrombus was found within the major coronary arteries and the atherosclerotic plaques within the coronary arteries showed no surface features which would have suggested the formation of a previous thrombus subsequently dissipated. These findings suggested that death had been caused by an electrical imbalance leading to dysfunction of the left ventricle, due to factors other than the presence of a blood clot.

[173] ACS was an umbrella term which embraced a spectrum of atherosclerotic coronary artery disease (ACAD) including unstable angina, myocardial infarction, and ischaemic sudden death, but not including stable angina. As used currently in clinical practice, the term included unstable angina and myocardial infarction. It was not a term used by pathologists who, instead, would make anatomical diagnoses of ACAD, coronary thrombosis or myocardial infarction as explanations for ischaemic sudden death. There was a distinction to be drawn between the role of the pathologist in making an assessment of the cause of death, as against clinical diagnosis of a condition in life. Unstable angina was a clinical diagnosis and not a pathological diagnosis; it is a clinical syndrome, and not a specific disease. It is angina in a crescendo pattern induced by limited physical activity or at rest. To distinguish it from stable angina required a clinical interpretation of the patient's historical account of their angina, and was therefore a subjective clinical symptom. It had been noted authoritatively that:

> "the distinction between new angina, worsening angina and unstable angina is notoriously difficult and based on a clinical assessment and a careful and full clinical history."

[174] Again under reference to his own report, in circumstances where, following *post mortem* examination, there was no basis for the conclusion that a transient thrombus had arisen from an atherosclerotic plaque, that being the most usual pathological basis of

unstable angina, there were four other possible causes of unstable angina, only one of which was referable to the present case. That cause was one of dynamic constriction of the relevant vessel (vasoconstriction) which could not be visualised *post mortem*. In the present case the time interval between the exhibited symptoms and death had been very short. No thrombus had been identified and no active smooth muscle proliferation, which would have indicated prior thrombus formation, had been noted as being present on any atherosclerotic plaque (a "culprit plaque"). On the other hand, the fact that no clot had been found at *post mortem* examination was not surprising. Research indicated that no clot was found in 20-30 per cent of comparable cases.

[175] In the absence of a "culprit plaque" or a coronary artery thrombus or fresh myocardial infarction, there was no evidence to suggest that in this case, the pathway to death had been by thrombotic occlusion or partial occlusion of the coronary artery. Where that was the case, death could arise as a result of the concurrence of fixed or dynamic factors, the latter being impossible to assess during an autopsy, although sometimes to be inferred from a history of physical exertion or high levels of emotion. Vasoconstriction was a dynamic phenomenon in the living which could not be seen after death. Similarly, stress-induced reduction in the activity of the autonomic innervation of the heart (reduced parasympathetic vagal tone) could facilitate the development of ventricular arrhythmia which could not be assessed *post mortem*. A rapid heart rate increased susceptibility to sub-endocardial ischaemia and arrhythmia. An acute rise in blood pressure as a result of exposure to cold could also increase myocardial oxygen demand and increase the risk of ischaemia and arrhythmia.

[176] In considering why it was that the deceased died on 6 January 2011 rather than at an earlier date, it was possible only to view the matter in hindsight by informed speculation.

Circumstances immediately prior to his collapse could have increased demand on his heart. The experience of being in hospital could sometimes be stressful, resulting in increased blood pressure and increased heart rate. Physical activity would also increase demand on the heart. Exposure to cold would have increased demand, by a rise in blood pressure without a commensurate fall in heart rate. In short, it was not possible to say definitively how the necessary factors combined at 6 January 2011. In theory, the deceased could have died at any time whilst engaging in the ordinary activities of life such as walking, running for a bus or, if he was sensitive to cold, by walking outside in winter weather.

[177] In response to the suggestion that had the deceased been an in-patient in hospital between 31 December 2010 and 2 January 2011 then, being in a warm environment with the benefit of bed rest, he would have avoided any exposure to cold which could therefore be eliminated as a possible cause of death, Professor Pounder qualified any answer as being one involving retrospective speculation. Even if the deceased had been sensitive to the cold, and the weather had been particularly bad, it was not possible to say definitively what would have happened. Even the experience of severe emotion could be enough to cause someone suffering from angina to collapse. The condition from which the deceased was suffering was the most common cause of sudden natural death. Although there was a possibility that some part of the hospital environment might have triggered a fatal cardiac event, it was to be noted that, in hospital, there was, of course, the potential for immediate resuscitation.

[178] In cross-examination, he accepted that the deceased had been suffering from a longstanding background condition which, as a result of a provocative trigger factor, had progressed to arrhythmia. He also agreed that it was not surprising that the deceased had been asymptomatic until only one week before his death. Even accounting for the

deceased's condition, comprising focal severe coronary atherosclerosis in the left main stem coronary artery, left ventricular sub-endocardial scarring from previous myocardial infarction, and left ventricular hypertrophy, it had not been inevitable that he would die of these conditions. Appropriate treatment would undoubtedly have reduced the risk of that.

[179] On the basis that unstable angina was a clinical diagnosis rather than a pathological diagnosis, in circumstances where there could be no obvious pathological basis for the presence of unstable angina, it was not possible to rule out unstable angina on the basis of pathological findings. The identification of unstable angina was a matter for a clinician, on the basis of the history given by a patient and the clinician's interpretation of the information provided.

[180] Of the five possible causes of unstable angina, vasoconstriction of the coronary artery, because of its dynamic nature, could not be confirmed at *post mortem* examination, and therefore could not be excluded as a cause of unstable angina leading to death on the basis of pathological findings. A comparable example was that of the limitations of *post mortem* examination in a case of fatal epilepsy. In Professor Pounder's view, on the basis that unstable angina had been the correct diagnosis, then vasoconstriction was the only possible explanation for death.

[181] Under reference to the A&E notes of Belford Hospital, in relation to the deceased's attendance on 6 January 2011, he accepted that the deceased's increased blood pressure at 10.45am (170/80), could have been caused by the experience of attending hospital and having negotiated the steps to the entrance of A&E, which in turn could have triggered arrhythmia.

[182] The possibility that exposure to cold could cause an acute rise in blood pressure, increasing myocardial oxygen demand and the risk of ischaemia and arrhythmia, was

supported by the relevant epidemiological research. There was no doubt that there was evidence to support that theory.

[183] It was commonly accepted that there were four principal precipitating causes for angina in susceptible patients. Such patients were warned against the risks involved in exercise, experiencing heightened emotion, exposure to cold, and the consumption of large meals.

[184] He confirmed that acceptance of a clinical diagnosis of unstable angina was not dependent on confirmation by pathological findings. Although discovery of thrombus would generally be expected as the most common cause of death in unstable angina, in 20 per cent of the referable cases there were other explanations.

[185] Although it was possible, in the generality, that very small or tiny clots could be formed, causing unstable angina, subsequently resolving so as not to be present at *post mortem* examination, in the present case there were none of the features in the atherosclerotic plaques which would be expected if that had occurred. If a thrombus had not dissolved, or had otherwise been assimilated by the body, but had become detached and moved downstream through the coronary artery, then the resulting occlusion, or partial occlusion would have resulted in a myocardial infarction. Viewed in the round, at *post mortem* examination, there had been no evidence of thrombus, and no evidence that there had ever been a thrombus. On that basis, his view was that any suggestion that there had been a thrombus was not well founded.

[186] In hindsight, the symptoms experienced by the deceased in the week before his death could be explained by the extent of the narrowing of the coronary artery and the enlargement of his heart which, together, were sufficient for the development of ischaemia,

particularly where there were added factors such as additional strain on the heart through exercise by, for example, moving up and down stairs.

Discussion

[187] Senior counsel for both parties adopted their respective written notes of argument, the content of which, together with the submissions made at the bar, is reflected in what follows.

[188] I accepted the witnesses to facts, namely, Dr Smith, Mrs Brown, and Mr Campbell, as being credible and reliable in their evidence. In my assessment, Dr Smith presented as a frank and open witness who, very responsibly, was doing her best to assist the court.

[189] In relation to the proper assessment of expert evidence, I was referred to *Kennedy* v *Cordia* 2016 SC (UKSC) 59. Applying the principles to be derived from that case, I found each of the expert witnesses, albeit to varying extents, and with the exceptions of Dr Gaskell and Professor Channer, to be appropriately qualified and experienced witnesses who gave authoritative evidence in an appropriately measured, detached and professional manner. In general, their views were coherent and clearly expressed.

[190] In contrast, I found both Dr Gaskell and Professor Channer to be unsatisfactory witnesses. Since Dr Gaskell's opinion was based solely on the content of the notes recorded by Dr Smith, and took no account of the totality of the deceased's symptoms as described, in particular, by Mrs Brown, it was in effect predicated on a basis which was contrary to the evidence which I accept, namely, that, by Friday 31 December 2010, the deceased's pain was occurring frequently and with little, minimal or no exertion. There were significant inconsistencies in his evidence. Although he made positive references to the SIGN 96 and NICE Guidelines in his report, his oral evidence, in relation to the appropriate use of GTN

spray and beta blockers was inconsistent with their terms. Contrary to the guidance of NICE 2010, which he accepted was relevant to practice in Scotland, he considered GTN spray to be appropriately used as a diagnostic tool. Notwithstanding the advice set out in SIGN 96 "*Management of Stable Angina*", to the effect that beta blockers should be used as first line therapy in cases of stable angina, he approved of Dr Smith's reasoning in not prescribing beta blockers and was not critical of her failure to prescribe available and appropriate alternatives. Somewhat surprisingly, given the strong views expressed by almost all the other experts on the importance of the matter, he expressly stated that he did not regard the provision of worsening advice to be a necessary requirement of ordinary competence. Contrary to other evidence which I accept, in particular from Mr Nichol, he did not accept that in the context under consideration, the prescription of aspirin was standard practice in 2010. I assessed him, as a witness, to be overly defensive of Dr Smith's actions, and indeed not obviously impartial. To the extent that his opinion differed from those of the other experts, I attached little weight to his views.

[191] Professor Channer had retired from NHS clinical practice in 2012, and, since 2014 had been engaged solely in medico-legal matters, instructed principally by defenders. He accepted that he had a reputation as being supportive of doctors. Although, in his report, he found that in the case of the deceased, death had been caused after a short history of unstable angina, and accepted in cross-examination, as was agreed between the parties, that unstable angina was a form of ACS which could be characterised by normal troponin levels, at other points in his evidence he appeared to consider ACS only to include troponin positive syndromes and to remove unstable angina from that class. In my assessment, the differentiation which he sought to draw in the context of the issue in this case, amounted to little more than a distinction without a difference, and, viewed more critically, was

unhelpful and unnecessarily obfuscatory. He was selective in his use of medical literature; in one instance he sought to characterise unstable angina by reference to one definition, in circumstances where, of the three other cited definitions, one more closely fitted the facts of the case. He was not familiar with the Patient.co.uk website. Although he recognised that the deceased, on Wednesday 5 January 2011, had been suffering from unstable angina involving pain at rest, his assertion that, prior to that day, the deceased was suffering merely from angina brought on by exercise was inconsistent with the evidence of Mrs Brown. In his report, he had noted that, at Friday 31 December 2010, the deceased had been suffering from chest pain lasting five minutes, precipitated by walking, whereas in fact what Dr Smith had noted was that the deceased was suffering chest pain after five minutes of walking. His evidence was internally inconsistent. He appeared reluctant to characterise the deceased's pain as having been brought on by minimal exertion, expressing the view that whether or not that had been the case depended on how minimal exertion was to be defined. Although he characterised chest pain brought on by minimal exercise as angina, rather than as unstable angina, on the basis that unstable angina was manifested by a crescendo presentation, he accepted that unstable angina can be defined not just by pain in crescendo pattern, but also by pain at rest which was getting worse, consistent with the deceased's condition. He accepted that changing pain on walking amounted to a crescendo pattern, and that crescendo pain on exercise was indicative of unstable angina. On the evidence, by Friday 31 December 2010, the deceased was exhibiting such a presentation. In my assessment of him as a witness, I found Professor Channer to be insufficiently neutral, independent and objective. As with Dr Gaskell, to the extent that his opinion differed from those of the other experts, I attached little weight to his views. By comparison, I had no such

reservations about the evidence of Professor Brecker, whose evidence, in relation to the issues on which they were each asked to express opinions, I prefer.

[192] My findings as to the agreed issues for determination by the court are set out in what follows. Unless the contrary is indicated, the evidence to which I have referred is evidence which I accept.

(i) What symptoms did the deceased suffer from between 29 December 2010 and 6 January 2011?

[193] On the agreed facts and the evidence which I accept, the deceased, having previously been a healthy, fit and active man, who participated regularly in golf, skiing, walking and cycling had the following symptoms at the relevant dates:

> *Wednesday 29 December 2010*: He experienced the first episode of chest pain when moving up and down stairs in his home. The pain eased after five minutes rest.
>
> *Thursday 30 December 2010*: He experienced chest pain when walking on the flat, over the short distance from his front door to the garden gate, the pain resolving on rest. Dr Smith noted the incident as indicating more chest pain. She accepted that such pain experienced after five minutes walking was equivalent to pain on minimal exertion. Viewed objectively, since he was now experiencing pain on the flat, rather than when moving up and down stairs, his condition had worsened. Mrs Brown described the deceased, on that day, as not doing very much, not being his usual active self, and that, whenever he moved, he experienced pain. Each time he rose from a seated position, the pain returned and, on sitting again, the pain resolved.

Friday 31 December 2010: The deceased experienced chest pain when getting dressed. The pain was retrosternal and localised. His wife described the situation as being that any movement on his part appeared to bring on pain.

Saturday 1 January 2011: Mrs Brown described the deceased as suffering pain whenever not sitting down. Walking to Mr Campbell's home, he continued to experience pain as before.

Wednesday 5 January 2011: The deceased was experiencing continuing chest pain and chest pain at rest including at night when lying down.

Thursday 6 January 2011: The pain experienced by the deceased, at rest, had been much worse (10/10) during the previous night. He had by that point been suffering chest pain for one week.

(ii) What symptoms did the deceased report to Dr Smith on 29 and 31 December 2010?

[194] On the basis of her recorded notes, Dr Smith noted the deceased as having reported as follows:

Wednesday 29 December 2010: The deceased had suffered chest pain after going up and downstairs, which had eased after sitting for five minutes. He had not been feeling sick or sweaty.

Friday 31 December 2010: The deceased had suffered sharp retrosternal chest pain after walking for five minutes. He had to slow right down. He did not feel sick or sweaty or unwell.

(iii) What condition or conditions was the deceased suffering from between 29 December 2010 and 6 January 2011?

[195] As disclosed at post-mortem, the deceased had been suffering from focal severe coronary atherosclerosis in the left main stem coronary artery, left ventricular sub-endocardial scarring from previous myocardial infarction, and left ventricular hypertrophy.

Wednesday 29 December 2010: On the basis of the evidence of Professor Brecker: new onset unstable angina.

Friday 31 December 2010: I accept the evidence, in particular, of Professor Brecker that, at this date, the deceased was suffering from ACS. Support for his opinion is provided by the evidence of Professor Wall which was to the effect that chest pain on minimal exercise should be taken as indicating ACS, until proven otherwise, and by Dr Nichol and Dr Johnson who agreed that pain on minimal exercise or at rest, relieved by rest, ought to have given rise to a presumptive diagnosis of ACS. Professor Brecker's evidence was that the deceased should have been diagnosed as suffering from ACS, in the form of unstable angina, since 29 December 2010. I accept his analysis that, on the known facts, the concept of a progression from stable angina on 29 December 2010, progressing to unstable angina subsequently, was not appropriate.

Wednesday 5 January 2011: Both Professor Brecker and Professor Channer agreed that, certainly by that date, the deceased was suffering from ACS in the form of unstable angina.

Thursday 6 January 2011: Death following a history of unstable angina.

(iv) Whether Dr Smith failed to act properly in seeking to obtain a medical history which would have resulted in the deceased disclosing that he had pain in the chest on minimal exertion? Whether Dr Smith breached her duty of care to the deceased on 31 December 2010 by failing to diagnose ACS?

[196] Both the SIGN 96 and the NICE Guidelines indicate that at the stage of assessment and diagnosis, in relation to patients with suspected angina, there is a clear requirement for a detailed clinical assessment and a detailed history. Mr Nichol's evidence was that, particularly in relation to patients presenting with chest pain, the taking of a proper history was the cornerstone of diagnosis, and that, as set out in his report, such a history should include the noting of details of the type of pain, whether it is new or a progressive symptom, its duration, its distribution and radiation, its alleviating and aggravating factors and all other associated features. The histories noted by Dr Smith on Wednesday 29 and Friday 31 December 2010, given the possibility of a life-threatening condition, were inadequate in these respects. In that context, given the imminent holiday period, and the high risk associated with a serious, unstable and potentially fatal condition, it was incumbent on Dr Smith to adopt a proactive approach in taking the deceased's history, which should have involved appropriately refined and penetrating questioning, and a recognition of the limitations, in that context, of a reliance on the passive recording of his reported symptoms. Given the known facts, comprising, in particular, the detail of Mrs Brown's evidence in relation to the deceased's symptoms over the period to Friday 31 December 2010, Dr Smith clearly failed to do that. Her notes do not record that he was suffering pain on minimal exertion, or that his condition had worsened. In relation to the taking of the deceased's history, she failed to act properly.

[197] Dr Smith's evidence was that although she did not consider ACS at the time, despite the recent onset of the deceased's pain, she accepted that, having been previously fit and active, he had presented to her with pain which had become worse over the previous two days and which arose on minimal exertion. These were symptoms in a previously active and fit man, indicating recent onset chest pain and therefore indicative of a potentially serious problem. Professor Wall's evidence was that in a previously fit man, where there was no proper basis for any muscular cause, a description of chest pain on exercise should have raised the question of possible angina. History was critical in the diagnosis of angina. The incidence in Scotland of angina was 6.7 per cent, or 1/15, within the relevant age group. I accept the evidence of Professor Wall that, by Friday 31 December 2010, chest pain experienced on minimal exercise was indicative of ACS, until proven otherwise, and should have required immediate referral to hospital; the evidence of Mr Nichol, that a presumptive diagnosis of ACS should have been made; and that of Professor Brecker, that, had the deceased been admitted to hospital on Friday 31 December 2010, a diagnosis of ACS would have been made. On the basis of that evidence, I find that, by failing, on 31 December 2010, to diagnose the condition of ACS, Dr Smith breached her duty of care to the deceased.

(v) If it was reasonable not to diagnose ACS, what treatment should Dr Smith have provided to the deceased?

[198] Dr Smith's evidence was that on Wednesday 29 December 2010 she had considered one possibility to be a cardiac problem and, in particular, had considered a possible diagnosis of stable angina. She had not ruled out angina. By 31 December 2010, she had considered angina to be more likely. On Friday 31 December 2010, she prescribed GTN spray, as a diagnostic tool, contrary to the NICE Guidelines, and made an urgent referral for

ETT. In that regard, she accepted that, except in relation to her prescription of GTN spray, she had not followed the SIGN 96 Guidelines in relation to the drug intervention recommended for first line therapy in the management of stable angina. Professor Wall's evidence was that, in cases of stable angina, the appropriate first line intervention was the prescription of GTN spray, low dose aspirin, beta blockers, statins, and, given the very great risks posed by angina, the provision of appropriate warning advice. Had it been reasonable for Dr Smith not to diagnose ACS, but rather stable angina, that is the course of treatment she should have provided. Her evidence was that she had not prescribed beta blockers because the deceased had been "wheezy", had already been prescribed salbutamol, and because, in cases of asthma, beta blockers were contra-indicated. In any event, she did not consider the possibility of prescribing any of the available alternatives to beta blockers. Professor Wall's evidence was that where a patient was intolerant of beta blockers, no reasonably competent GP would have failed to provide an alternative drug. The effect of beta blockers or the available alternatives would have been to reduce the level of cardiac ischaemia, by reducing the heart's workload, and thereby reducing the risk of ventricular fibrillation. Both Professor Brecker and Professor Channer expressed the view that the maximum effect of such drugs would be achieved within about three days. Although there was reference in the evidence to a longer relevant period, in particular in relation to the findings of the MIAMI international trial, reported in the European Heart Journal (1985), I have not placed weight on these findings as they related to patients, who, unlike the deceased, were suffering from acute myocardial infarction.

[199] It was accepted that, on Friday 31 December 2010, Dr Smith ought to have provided the deceased with worsening advice, but failed to do so. In the course of the evidence, what that worsening advice ought to have been was expressed in a variety of different ways.

Dr Smith indicated that it would have been appropriate that the deceased be told to call an ambulance or go to hospital if his symptoms deteriorated, and Dr Gaskell's view was that the advice should have been to seek an earlier review if they worsened. It was suggested on behalf of the defenders that by Friday 31 December 2010, the deceased's symptoms had reached a plateau and did not in fact become worse prior to Wednesday 5 January 2011, but, rather, during that period maintained a constant level. However, Professor Wall, Mr Nichol and Professor Johnson all spoke of the very great risks associated with angina, and taken together with the context of the imminent holiday period, I prefer the evidence of the witnesses other than Dr Smith and Dr Gaskell, to the effect that the appropriate worsening advice should have been more extensive. Professor Wall's view was that the deceased should have been advised to call a 999 ambulance in the event of further chest pain on minimal exertion or at rest. Mr Johnson, in his report, referred to advice specifically to ring 999 if chest pain returned and did not respond to GTN spray and persisted more than 20 minutes or came on frequently or with no exertion. Mr Nichol, in his report referred to advice in relation to pain at rest, on minimal exertion, lasting for 15 minutes, or being unresponsive to GTN spray. Given the deceased's condition at Friday 31 December 2010, and on the basis that a full history had been taken, the tenor of that evidence is, in effect, that the appropriate advice should have been to call an ambulance if there was no improvement, in the sense that his symptoms continued as they currently were. On that basis, in the context of the high risk symptomatology associated with angina, the concept of symptoms having reached a plateau has no application in relation to the issue of causation following failure to give such advice.

(vi) What was the cause of the deceased's cardiac arrest on 6 January 2011?

[200] Although there was reference to the fact that the most common cause of death in cases of coronary artery disease involves the presence of thrombus, I am satisfied that death did not occur in that way in the case of the deceased. At *post mortem* examination, no blood clot was found to be present, and, in the absence of evidence of any active smooth muscle proliferation which might indicate prior thrombus formation on atherosclerotic plaque, there is no basis from which to conclude that there ever was thrombus present. The issue of any thrombus having been dissipated or assimilated by the body does not arise given the timescale of the deceased's sudden death. Since the matter was raised, if only incidentally, in the course of the proof, for completeness, I would add that, in the context of no clot having been found at *post mortem* examination, I find there to be no persuasive basis from which it could be inferred that either of the two pathologists concerned was negligent in that regard. I find, rather, in circumstances in which the deceased had been suffering from longstanding focal severe coronary atherosclerosis in the left main stem coronary artery, left ventricular sub-endocardial scarring from previous myocardial infarction, and left ventricular hypertrophy, death was caused by ventricular fibrillation which, in turn, was caused by ischaemia brought about by reduced blood flow to the heart muscle, resulting from stenosis of the left main stem coronary artery. That conclusion was common ground between Professor Brecker and Professor Channer. The evidence of both of these witnesses, taken with that of Professor Pounder, was to the effect that the ischaemic nature of the heart muscle, however it was triggered, was the principal precipitating factor, in that it was ischaemia which caused the ventricular fibrillation leading to sudden death. In Professor Brecker's view, the primary cause of the ventricular fibrillation was ischaemia. He considered that there had been sufficient substrate, in the form of the extent of the noted

stenosis, coupled with pain on minimal exertion, to trigger ischaemia and thereafter sudden death, absent another provocational trigger. In Professor Pounder's view, increased heart size coupled with stenosis of the coronary artery would have reduced blood flow to the heart, rendering the heart muscle ischaemic, and leading to cardiac arrhythmia. In circumstances in which the stenosis present was reducing the *lumen* of the coronary artery by 70 per cent, and was coupled with the noted left ventricular hypertrophy, these factors together, in his opinion, were sufficient to account for ischaemia of the heart muscle. Where the deceased had, in addition, been suffering from angina, the risk of sudden death brought about by ischaemia was significantly increased.

(vii) What difference, if any, would treatment or advice offered by Dr Smith on either 31 December 2010 or 5 January 2011 have made to the outcome?

[201] If, on Friday 31 December 2010, Dr Smith had prescribed beta blockers or an appropriate alternative, the deceased would have had the benefit of their anti-ischaemic effect. On the basis that their maximum effect would have been in place within about three days, the likelihood of a fatal cardiac event, the principal cause of which was ischaemia, would have been significantly reduced, and, on the evidence of Professor Brecker, reduced to the extent that it would have been prevented.

[202] If on Friday 31 December 2010, Dr Smith had immediately referred the deceased to hospital, then, under reference to my findings in relation to ***Issue (viii)***, below, with the benefit of hospital treatment, death on 6 January 2011 would have been avoided.

[203] If, on Friday 31 December 2010, Dr Smith had provided appropriate worsening advice, the deceased, on her evidence and that of Mrs Brown, would have followed it, and

would have been admitted to hospital, following the events of New Year's Day as described by Mrs Brown, or, at the latest, following their visit on foot to Mr Campbell.

[204] If, on Wednesday 5 January 2011, Dr Smith had prescribed beta blockers or an appropriate alternative, the deceased would have had the benefit of their anti-ischaemic effect, but to a lesser extent. On the evidence of Professor Brecker, even if the deceased had been admitted to hospital on that date, it would not be possible to say that the risk posed by ischaemia could have been avoided. It could have been reduced, but it could not be said that the medication would have reached its maximum effect. The effect would have been more than negligible, and not insignificant, but it could not be said that more than 50 per cent of its desired effect would have been achieved prior to the time of death.

(viii) On a hypothesised attendance at Belford Hospital between 31 December, after attending Dr Smith, and the end of 2 January 2011, what difference, if any, would treatment have made to the outcome?

[205] I accept the evidence of Mr Nichol, largely agreed by Mr Johnson, that if the deceased had attended hospital on Friday 31 December 2010 , a diagnosis of ACS would have been made, prompting the prescription of aspirin, heparin, and GTN spray, together with morphine if the deceased was in pain. Thereafter the deceased would have been transferred to an acute medical unit within the hospital. On the basis that the presentation would have indicated that blood flow to the heart muscle was critically impaired, with the risk of progression to acute cardiac arrest due to abnormal heart rhythm, the deceased would have been kept in hospital. Blood tests would have been carried out, beta blockers, or where there was a contra-indication, appropriate alternatives, and statins prescribed, high blood pressure treated, and an ETT carried out. In broad terms, Professor Channer

agreed that such a course of treatment would have been put in place. Professor Brecker's evidence was that the deceased would have been admitted, intravenous access obtained, and anti-platelet and anti-thrombotic drugs administered, together with aspirin, or an appropriate alternative, to put in place dual anti-platelet therapy. It would have been standard practice to prescribe beta blockers in order to reduce his cardiac workload. He would also have been prescribed vasodilators, to increase blood supply, by widening the coronary artery and reducing demand on the heart. In the case of the deceased, nitrates and calcium channel blockers would have been prescribed in addition to beta blockers. If the deceased had been continuing to experience pain at rest, intravenous nitrates would have been administered. In addition, he would have been given injections of anti-thrombotic drugs, such as heparin or similar, and would have been started on other drugs with longer term effects, such as statins to reduce cholesterol levels, and vasodilators such as ACE inhibitors. All of that would have been standard practice. The deceased would have been kept in hospital on admission because of his high risk. The core aims would have been to seek to avoid stressors, and indeed anything involving an increase in heart work rate, particularly in the form of exertion. Mr Nichol's view was that, had the deceased been kept in hospital, and received appropriate treatment, and kept at rest, then, on the balance of probabilities, the chances of him suffering a cardiac arrest on Thursday 6 January 2011 would have been reduced, but not excluded entirely. Professor Brecker's evidence was that even in the absence of thrombus, the prescription of beta blockers would have had immediate anti-arrhythmic effect but, in particular, would have served to reduce cardiac demand and workload. In so far as the timing of the effect of medication was concerned, he considered that the relevant period was probably one of about three days. In cross-examination, Professor Channer agreed with that. He accepted that beta blockers

have an anti-ischaemic effect, the maximum effect of which occurs within two to three days of administration. Calcium channel blockers would not have been administered unless there was a contra-indicator for beta blockers. Beta blockers reduced demand on the heart, correcting any supply/demand imbalance likely to cause ischaemia. On the same analysis, Professor Brecker's evidence, which I accept, was that if the deceased had been admitted to hospital on Friday 31 December, the medication which he would have been prescribed, as treatment for ACS, together with the fact of simply being in hospital, on the basis of his clinical experience, and as I find on the balance of probabilities, would have prevented ischaemia and therefore ventricular fibrillation. Death on Thursday 6 January 2011 would have been avoided. Although it was suggested by Mr Johnson that the deceased might have been discharged home, that was contradicted by both Mr Nichol and Professor Brecker. On the basis that the deceased would not have been admitted, or would subsequently have been discharged only if, on appropriate medication, he would have been able to exercise without significant pain, Professor Brecker's view was that given the *post mortem* findings indicating severe main stem coronary artery stenosis, discharge home would have been unlikely.

[206] Both Mr Nichol and Professor Brecker were of the opinion that had the deceased attended hospital later, for these purposes up to the end of Sunday 2 January 2011, the treatment he would have received would have been the same. In Professor Brecker's view, the effect, and likely outcome would have been the same in that death would have been avoided.

(ix) What difference, if any, would the non-drug treatment associated with being in hospital on 6 January have made to the outcome?

[207] On the evidence of Mr Nichol, the deceased when in hospital would have had the benefit of being kept in bed rest, and would thus have been protected from any exposure to the consequences of physical exertion which might have provoked cardiac arrest.

[208] On the evidence of Professor Pounder, the possibility that exposure to cold could cause an acute rise in blood pressure, increasing myocardial oxygen demand and the risk of ischaemia and arrhythmia, was supported by relevant epidemiological research. If in hospital on Thursday 6 January 2011, the deceased would not have been exposed to the low temperature out of doors and would have been protected from that risk.

Conclusion

[209] On the basis of the relevant case law to which I was referred, namely *Hunter* v *Hanley* 1955 SC 200, *Bolitho* v *City and Hackney Health Authority* [1998] AC 232, *Honisz* v *Lothian Health Board* [2006] CSOH 24, and *Amanda McGuinn* v *Lewisham and Greenwich NHS Trust* 2017 EWHC 88 (QB), and my findings in relation to the agreed issues, I find that Dr Smith was professionally negligent and breached the duties of care which she owed to the deceased, in that on Friday 31 December 2010, she (i) failed to diagnose unstable angina, (ii) failed to refer him immediately to hospital, (iii) failed to provide appropriate worsening advice, and (iv) failed to prescribe the medication appropriate for a diagnosis of stable angina. Having considered the further authorities to which I was referred, namely *Wright* v *Cambridge Medical Practice* [2013] QB 312; *Hughes-Holland* v *BPE Solicitors* [2017] 2 WLR 1029; and *South Australia Asset Management Corp.* v *York Montague Ltd* [1997] AC 191, and applying the principles to be derived from them to the facts of this case, I am satisfied, given my

findings in relation to the agreed issues, that it is proved that these failures were materially causative of death, in that their consequences were that the deceased was denied the appropriate treatment which, on the balance of probabilities, would have reduced his level of cardiac ischaemia to the extent that ventricular arrhythmia would have been prevented. I find, in addition, that in any event, if the fatal cardiac event on Thursday 6 January 2011 was caused by exposure to cold, as a provocational trigger, her failures were responsible for him being denied the protection from such exposure which admission to hospital would have provided.

Decision

[210] For these reasons, I find in favour of the pursuers. I sustain the pursuers' first and second pleas-in-law, repel the defenders' third, fourth and sixth pleas-in-law, and award to the pursuers the sums agreed. I reserve, meantime, all questions of interest from the date of proof, and expenses. The case will be put out By Order to allow these matters to be addressed.