SHERIFFDOM OF GLASGOW AND STRATHKELVIN AT GLASGOW

[2017] FAI 4

B952/16

DETERMINATION

BY

SHERIFF LINDA M RUXTON

UNDER THE FATAL ACCIDENTS AND SUDDEN DEATHS INQUIRIES (SCOTLAND) ACT 1976

into the death of

RICHARD CLOW

GLASGOW, 21 December 2016. In terms of section 6 of the Fatal Accidents and Sudden Deaths Inquiry (Scotland) Act 1976, the Sheriff having considered the evidence, the productions, the joint minute and submissions thereon, FINDS and DETERMINES:

(1) In terms of section 6(1)(a) of the Act, that Richard Clow, date of birth 14 August 1964 formerly of 27 McColl Place, Alexandria, West Dunbartonshire G83 0HS, residing latterly at Her Majesty's Prison, 190 Crosshill Road, Low Moss, Bishopbriggs died within cell 23, D Hall there during the early hours of 3 April 2015. Life was pronounced extinct at 0736 hours on that date.

(2) In terms of section 6(1)(b) of the Act, the cause of death was:

Part I (a) ischaemic heart disease

Part II buprenorphine intoxication; sleep apnoea syndrome.

(3) In terms of section 6(1)(c) of the Act, there were no reasonable precautions whereby the death might have been avoided.

(4) In terms of section 6(1)(d) of the Act, there were no defects in any system of working which contributed to the death.

(5) In terms of section 6(1)(e) of the Act, there were no other facts relevant to the circumstances of the death in respect of which any determination falls to be made.

NOTE

Introduction

[1] This is a Fatal Accident Inquiry in terms of section 1(1)(a)(ii) of the Act as Mr Clow was in custody at the time of his death.

[2] The Inquiry commenced on 23 August 2016 after which two further days of evidence were led on 18 and 31 October 2016. The Crown, in the public interest, was represented by Ms Carrie Macfarlane, Procurator Fiscal Depute. Ms Sarah Phillips, Solicitor, appeared on behalf of the Scottish Prison Service.

[3] The following witnesses gave evidence:

1 Mrs Carol Clow, wife of the late Richard Clow and next of kin.
2 Dr Francis Gerald Dunn, Consultant Cardiologist, Stobhill Hospital, Glasgow.
3 Miss Suzanne Wilson, Head of Operations, HM Prison, Low Moss.
4 Dr Michael Parsons, Consultant Forensic Pathologist.
5 David McGregor, Detective Sergeant, Police Scotland.
6 Dr Grace Campbell, General Practitioner, Low Moss Prison.
7 Dr Julie McAdam, Consultant Forensic Pathologist, University of Glasgow.
8 Dr Andrew Flapan, Consultant Cardiologist, Royal Infirmary, Edinburgh.

Uncontroversial factual evidence was presented by way of a joint minute of agreement.

[4] In terms of the relevant legislation, a fatal accident inquiry must be held if the death has occurred while the deceased person is in lawful custody. Although the holding of the Inquiry was mandatory because Mr Clow was a serving prisoner when he died, the circumstances of his death did not give rise to any matters of serious public concern. However, his family had expressed concerns about the certified cause of death and about apparent delays in medical treatment he had received in connection with recently diagnosed heart disease. Accordingly, in the course of the Inquiry, two matters in particular were explored: (1) the cause of death, with particular reference to the inclusion of buprenorphine intoxication as a primary cause of death; and (2) whether there had been any undue delay in diagnosis and treatment of Mr Clow's cardiac problems. The matter of illicit drugs in circulation in Low Moss prison was also briefly explored.

Background and Medical History

[5] Richard Clow was born on 14 August 1964 and was 50 years old when he died. He had been married to his wife, Carol, for 30 years and they had three children.

[6] On 9 September 2013, he was convicted of two contraventions of the Misuse of Drugs Act 1971, in respect of which he received a sentence of imprisonment for

four years. Accordingly, at the time of his death he was a prisoner in lawful custody in Her Majesty's Prison, Low Moss, Bishopbriggs.

[7] According to his wife and as documented in his medical records, Mr Clow had a long-standing history of addiction problems involving both drugs and alcohol. He was addicted to diamorphine (heroin) and diazepam (valium) and, at the time of his admission to Low Moss, had been consuming alcohol at a rate of one bottle of vodka per day. While Mr Clow was in the community, his general practitioner prescribed buprenorphine (the proprietary drug Suboxone) in order to assist him in tackling his drug addiction. He had been prescribed this drug since 23 May 2011 until his reception into Low Moss on 9 September 2013. He was prescribed 8 milligrammes per day. According to Mrs Clow, the medication was working well.

[8] This prescription was continued on admission to prison. However, in October 2013, it was suspected that Mr Clow had been concealing his Suboxone medication and, in accordance with strict prison protocol, his prescription was immediately discontinued. He declined the offer of methadone as an alternative medication but was prescribed dihydrocodeine to assist with his detoxification. Never at any time after the discontinuation of his prescription did he refer himself to the addiction team within the prison.

[9] Although Richard Clow was no longer prescribed Suboxone, on 16 September 2014 a routine prison drug test produced a positive result for the presence of buprenorphine. It was clear that he had been accessing an illicit source of Suboxone

within the prison although it was not known how regularly he accessed such a source or the amounts that he was taking.

[10] Mr Clow had suffered from high blood pressure (hypertension) for a number of years. His general practitioner had prescribed medication (Ramipril and amlodipine) in an attempt to lower and control it. This medication was continued in prison and his blood pressure was regularly monitored. In addition, he suffered from sleep apnoea syndrome – a condition whereby during sleep the muscles relax, so much so that air cannot be drawn into the lungs and normal breathing is interrupted. Mr Clow was also a heavy smoker. Beyond his hypertension, it was not known that he had any significant heart disease while he was in the community.

[11] However, while he was serving his sentence, Mr Clow was diagnosed with serious heart disease. In October 2014, he had complained of blurred vision. It was suspected that he might have suffered a mini-stroke and a fast-track referral was made to the relevant clinic at Stobhill Hospital. In the course of investigations at the stroke clinic, a routine electrocardiogram (ECG) was seen to be abnormal. Mr Clow was transferred into the care of the cardiology department. Further investigation revealed that he suffered from a number of heart conditions, each one serious and potentially fatal.

[12] Essentially his cardiac problems were two-fold: severe coronary artery disease and severe aortic valve disease. In terms of coronary disease, investigations revealed that, although he was unaware of this, Mr Clow had previously had a myocardial infarction (heart attack). Associated scarring of the heart muscle was noted. There was

severe atheromatous narrowing of the left anterior descending coronary artery; at least moderate atheromatous narrowing of the right coronary artery; and mild narrowing of the circumflex coronary artery. In addition, he suffered from cardiomegaly (a grossly dilated and enlarged heart) indicating that it had been working hard to maintain his circulation for some time. He had left ventricular hypertrophy. Again this showed that his heart was having to work harder to maintain adequate oxygenation which had, in turn, led to a thickening and enlargement of the heart muscle (hypertrophy). Thicker muscles require a greater blood supply which in turn increases the amount of work which the heart has to do to maintain circulation thus leading to a vicious circle of dysfunction.

[13] In addition to his coronary artery disease, Mr Clow suffered from severe aortic valve disease. The diseased aortic valve (not noted at autopsy but confirmed by hospital tests) was a congenital condition which had been present from birth but which had become progressively worse to the point that the valve was seriously compromised. The aortic valve is the main valve which joins the heart to the aorta, the main blood vessel in the body. Thus the valve performs an important role in the circulation.

[14] Both aspects of heart disease had compromised Mr Clow's circulation significantly. Both conditions predisposed him to disturbance of the heart rhythm, known as cardiac arrhythmia. In such circumstances, sudden death is the most feared consequence.

Circumstances of death

[15] Richard Clow died in his cell (Cell 24, Clyde Hall, level 3, D Section) during the night between lock-up at 2030 hours on 2 April and his body being found early the following morning. At about 0700 hours on 3 April 2015 Carol-Ann Graham, Prison Officer, was carrying out a numbers check in Clyde Hall, Level 3, D Section. When she opened the door to Cell 24, she saw Richard Clow lying face down on the floor and noted a small pool of blood around his head. A "code blue" call for urgent assistance was made and a full emergency response followed within minutes. On being turned onto his back, it could be seen that Mr Clow had been lying with his left arm across his body against his chest, perhaps suggestive of him having clutched his chest when he collapsed. It was clear that Mr Clow had been dead for some time and that any effort at cardio-pulmonary resuscitation would have been futile. His life was formally pronounced extinct at 0736 hours by ambulance paramedics although death clearly had occurred several hours earlier during the night.

[16] A search of Mr Clow's cell by prison officers revealed a plastic wrap containing 2¼ white tablets underneath a pillow. These tablets were later analysed and found to be Suboxone.

[17] The packaging of the tablets was sent for forensic analysis and was swabbed for DNA. The swab was analysed and found to contain a mixed DNA profile with one major contributor and at least two minor contributors. The major contributor profile was the DNA of Richard Clow. The remaining traces of DNA were unsuitable for comparison purposes.

Post-mortem findings

[18] On 16 April 2015 at the Southern General Hospital, Glasgow a post mortem examination was carried out by Dr Michael Parsons and Dr Julie McAdam, both consultant forensic pathologists at the University of Glasgow. The heart disease that had been identified in the course of the various hospital investigations was confirmed at autopsy. Mr Clow had serious and extensive heart disease described under the umbrella term of ischaemic heart disease. This would have made him liable to cardiac arrhythmia and sudden death at any time.

Toxicology

[19] Post-mortem samples of blood and urine revealed the presence of buprenorphine. The level was recorded less than 5ng/L. This level was within the range of a therapeutic dose for someone on a daily dose of 8mg of Suboxone. Levels below 5ng/L are difficult to detect and as a matter of practice, the lowest level recorded is <5ng/L. Therefore, it follows that the actual level in Mr Clow's sample might well have been lower than five.

Buprenorphine

[20] Buprenorphine is a powerful opioid pain-killing drug that is used in opiate replacement therapy. It acts as a partial agonist and works by blocking the opiate receptors. It can be prescribed in doses of 4, 8, or 24mg tablets. Buprenorphine is a potent and long-lasting drug. As with all opiates, it has a number of side effects.

Among these is its ability to cause respiratory depression, particularly in the new, naive user. Severe respiratory depression can induce coma and death. Like all opiates, buprenorphine can also cause blood pressure to drop. Those who are in regular receipt of the drug quickly build up a tolerance to its potentially dangerous adverse effects. However, that tolerance may be lost or eroded within a few days where the drug is stopped or is used intermittently.

Certified cause of death

[21] Having regard to the potential effects of buprenorphine on someone, like Mr Clow, whose tolerance to the drug was unknown but who was suffering from severe heart disease, Dr Parsons and Dr McAdam considered that both elements should be reflected in the principal cause of death. The death certificate was issued recording the cause of death as

Ia Buprenorphine intoxication and ischaemic heart disease.

They included buprenorphine as a principal cause of death although both acknowledged that the extent to which buprenorphine was involved, if at all, was actually unknown.

Mechanism of death

[22] Mr Clow died as a result of a fatal cardiac arrhythmia to which he was highly susceptible. Dr Flapan, the independent expert cardiologist, explained that heart arrhythmias are very uncommon. For such an event to occur, it is believed that

three elements must be in place and coincide. First, a substrate is required – in other words there has to be some underlying heart disease. Secondly, a precipitating event is required. This can be as simple as the heart having an extra beat (something which many experience without even noticing that it has occurred but in situations of disease and compromise may have a significant effect). Thirdly, and just as important, that precipitating event must affect and alter the environment in which the heart lies. Anything which impacts on the physiology of the body (for example, a reduced oxygen supply) alters the environment in which the heart is functioning. If all three elements are present, then the chances of an arrhythmia are dramatically increased. Given the extent of his heart disease, Mr Clow was very vulnerable to cardiac arrhythmia and sudden death.

Potential role of opiate medication in Mr Clow's death

[23] Both Dr Parsons and Dr McAdam acknowledged that it was impossible to determine the precise role, if any, that buprenorphine had played in Mr Clow's death. It was not possible to get any clear picture of his usage and therefore hard to predict his tolerance level. Dr Parsons felt that as he had not been prescribed the drug for several months, it could reasonably be assumed that he had lost his tolerance or that his tolerance would have been reduced to a degree, depending on his usage. Dr McAdam shared that view: Mr Clow would have had a period of enforced abstinence from the drug which in turn would lead to a loss of tolerance, thereby creating a real danger.

However, she agreed that it was impossible to predict how his tolerance had been affected – everyone was different.

[24] Both pathologists agreed that Mr Clow's heart disease was such that he could have suffered sudden death at any time. Dr McAdam described him as "teetering on the brink": any insult to an organ of the body, while not itself the cause of death, might have pushed him over the edge. In their joint report, both pathologists agreed that, on balance, it appeared that the most important factor in Mr Clow's death was his heart disease and that natural disease was likely to have been the major factor in his death. There were no pathological features to suggest that the mode of death was one of respiratory depression. The findings at autopsy were more consistent with sudden death than a progressive induced coma. Both felt that a sudden collapse was most likely. That said, Dr McAdam was troubled by the fact that there was no circumstantial evidence to suggest that he had collapsed suddenly. Had someone witnessed him clutching his chest before collapsing, then she would have had no difficulty in certifying death as the result of ischaemic heart disease. But in the absence of any such evidence, there were two different mechanisms of death – he could have died as a result of heart disease or drug intoxication – and she could not distinguish between the two mechanisms of death or choose one cause of death over the other. Dr McAdam did not consider the circumstances in which Mr Clow had been found to have been helpful and believed that to extrapolate any finding from that would involve speculation.

[25] Thus it was their clear view that they could not exclude the potential role of drug intoxication. It was difficult to disregard its presence as a potential cause of death or a

contributing factor. According to Dr Parsons, buprenorphine could have been a significant factor in Mr Clow's death: he had a drug on board which had the potential to aggravate his natural disease. Although Mr Clow could easily have died from heart disease at any time, buprenorphine had the potential to have precipitated death. Accordingly, both ischaemic heart disease and buprenorphine intoxication were included in the death certificate as potential mechanisms under Part I. He remained of the opinion that the two aspects were equally consistent as a cause of death. However, he acknowledged that it was ultimately a matter of opinion and that some doctors might have put the buprenorphine under Part II on the death certificate.

[26] Dr McAdam agreed that it was a matter of opinion whether it was included as the main cause of death under Part I or as a contributory factor under Part II of the death certificate. Her firm view was that buprenorphine remained a significant factor in his death.

[27] Dr Grace Campbell was surprised to learn of the certified cause of death. She is the Lead Clinician for Greater Glasgow and Clyde Prison Health Care. In her professional career she has specialised in addiction medicine and, in particular, in relation to drug and alcohol addiction. She has a great deal of experience in addiction matters both as a practitioner and on the policy side. In her capacity as general practitioner in Low Moss Prison, Mr Clow was her patient and she had both continued and discontinued his Suboxone subscription in Low Moss. She explained that there were very few deaths associated with buprenorphine only. It was regarded as one of the safer opiate substitutes. Indeed, had Mr Clow come to see her with on-going addiction

issues, there was every possibility that she would have put him back on Suboxone, notwithstanding his cardiac problems. The medication would have been given under supervision. In her view, prescribed buprenorphine would have been a safer option than illicit use.

[28] While Dr Flapan was not an expert in toxicology, his opinion about the likely effects of opiates on heart disease was something well within his area of skill. He explained that opiates had to be used with caution with patients who have heart disease. There were several potential mechanisms by which opiate drugs might affect the heart.

[29] Firstly, cellular bio-chemical mechanisms affect the electrical behaviour of the heart. All opiate drugs will have an effect. Some drugs have more of an effect than others. For example, it is well documented that methadone affects the electrical behaviour of the heart by prolonging the PQ interval. While buprenorphine has very little effect, it cannot be said to have no effect.

[30] The second mechanism involves an alteration of the environment in which the heart is functioning. All opiate drugs can cause respiratory depression. If this occurs and the amount of oxygen is decreased, this in turn reduces the amount of oxygen delivered to the heart. Buprenorphine is said to produce less respiratory depression than, for example, methadone. However, it is important to note that this depends on a person's tolerance to the drug and susceptibility to its effects at the time. If the drug is well tolerated, then it can be taken in a large dose without affecting the blood pressure or the occurrence of respiratory depression. Tolerance depends on exposure and where

the drug is taken intermittently tolerance may be much lower. Buprenorphine is not without risk but is less risky than other opiates such as methadone or morphine.

[31] All opiate drugs can lower the blood pressure and therefore there was the potential for a drop in Mr Clow's blood pressure. The delivery of blood to the heart muscle depends on the blood pressure and the level of delivery of oxygen to the heart can vary according to the pressure. Such a variation would create an environment whereby there is a potential to reduce the amount of oxygenated blood going to the heart muscle.

[32] Therefore, buprenorphine can pose very significant risks to someone with serious heart disease, depending on the degree of tolerance to the drug. Accordingly, Dr Flapan would have a level concern were a patient with serious heart disease to be prescribed buprenorphine in the community. He would not prescribe the drug outwith a hospital setting where the effects could be closely monitored. Equally, he would be concerned about the risks associated with intermittent, illicit use.

[33] Any respiratory depression that alters the level of oxygen in the blood could be very, very significant in someone, like Mr Clow, who was very vulnerable. Dr Flapan considered the recorded condition of sleep apnoea syndrome to be important to note because, if it occurs very frequently, sleep apnoea can contribute to high blood pressure and heart arrhythmias. This could be significant as it might have been the trigger for Mr Clow's fatal arrhythmia.

[34] Had he been certifying the death, Dr Flapan would probably have put buprenorphine under Part II as a contributing factor, not under Part I. Heart disease –

the substrate – was clearly the cause of death. The role of the buprenorphine was uncertain. Had Mr Clow not had underlying heart disease, the significance of buprenorphine would have been much reduced. It would, therefore, have been reasonable to have certified heart disease as the main cause of death in Dr Flapan's opinion.

[35] Having regard to the evidence, the procurator fiscal depute invited me to make a finding under section 6(1)(b) that the cause of death was Ia Ischaemic heart disease noting potential contributory factors of buprenorphine intoxication and sleep apnoea under Part II. She submitted that given the uncertain evidence about the role of buprenorphine, buprenorphine intoxication did not sit easily in Part I on the death certificate. What was absolutely clear from the evidence was that severe heart disease could, on its own, have accounted for sudden death at any time. Ms Phillips agreed.

Conclusion

[36] It was clear that Mr Clow could have died at any time and his death from cardiac causes would not have been unexpected. Something happened to trigger the fatal arrhythmia. It would not have needed much to upset the highly sensitive environment in which his heart functioned. Undoubtedly, buprenorphine could have caused a drop in blood pressure or some degree of respiratory depression which set in train the fatal event. However, given Mr Clow's vulnerability and susceptibility to cardiac arrhythmia, anything that upset the delicate balance might have caused it: even something as simple as a missed heartbeat.

[37] Accordingly, I was not persuaded by the pathologists' evidence as to the significance of buprenorphine in Mr Clow's death, at least insofar as including it as a principal cause of death. While I was entirely satisfied as to the risks associated with buprenorphine and its potential effect in triggering an arrhythmia, the fact remained that the pathologists could not say if, or to what extent, it played a part in Mr Clow's death. As Dr McAdam said, the problem is we *just do not know*. Given that position, I have difficulty in seeing how that uncertain state of knowledge could translate into a positive finding that buprenorphine intoxication was a principal cause of death. On the evidence before me, the potential action of buprenorphine was one of several possible mechanisms whereby a fatal cardiac arrhythmia occurred. However, the role played by buprenorphine in triggering Mr Clow's cardiac arrhythmia was unknown. Its effects, *if any,* could not be quantified.

[38] Having regard to the evidence before me, in particular that of Dr Flapan, I am satisfied that Mr Clow suffered a sudden cardiac death. I found the circumstances in which Mr Clow was found in his cell to be indicative of a sudden collapse. There were a number of other factors, such as sleep apnoea or even the random miss of a heart beat that would have been enough to have tipped Mr Clow into arrhythmia, as well as the buprenorphine. Coronary artery disease and aortic valve disease alone put him at high risk of arrhythmia and sudden death.

Revised cause of death

[39] For that reason, I have determined that the appropriate certification should read ischaemic heart disease as the primary cause of death with buprenorphine intoxication and sleep apnoea being noted under Part II as conditions present that might have contributed to death.

Illicit buprenorphine (suboxone) in prison

[40] In its proprietary form, Suboxone (which contains the active ingredients of buprenorphine and naloxone), has a high currency value in prisons: in monetary terms it can apparently fetch up to £20 to £40 per tablet. Illicitly obtained tablets are usually crushed and snorted in 2-3mg doses. Dr Grace Campbell explained that Suboxone is undoubtedly the most sought-after drug in the custodial setting. Accordingly, there is an active market for Suboxone in prisons generally. This is not a local phenomenon but is a problem world-wide, despite diligent efforts to control its introduction and circulation within prisons. Dr Campbell explained that the drug is given daily under supervision and nursing staff adopt various strategies to ensure that the drug is properly ingested and not saved or concealed to be sold later. These strategies reduce the risk of the drug being diverted into the illicit market within the prison. The illicit availability of Suboxone places the prisoner to whom it is prescribed at risk as well as other prisoners. The circulation of drugs within prisons is clearly an on-going problem that is difficult to eradicate.

[41] Ms Suzanne Wilson, Head of Operations at Low Moss, explained that unauthorised access to drugs, both illicit and prescribed, remains a challenge within the

prison setting. The prisons authorities are constantly looking at different technologies and procedures to reduce, if not eliminate, the flow of contraband into prison establishments. General measures include search, x-ray and supervision and the use of specially trained search dogs. Staff remain vigilant in the visiting areas and CCTV technology is in use. Other, more advanced and sensitive techniques are also used. In spite of increasingly complex technology and search procedures, contraband, including drugs, continues to enter the establishment and circulate there.

[42] Highly sought-after drugs such as Suboxone are therefore dispensed in such a way as to reduce the chance of secretion and onward circulation. They are dispensed under supervision on a daily basis. Each person is first given a drink of water to moisten the mouth. The drug is then dispensed and further water given. The prisoner remains under the supervision of a prison officer and an oral search is undertaken to ensure that the drug has been consumed. After 10 minutes, the prisoners are allowed to return to their halls. Even with such strict supervision, it has not proved possible to eliminate the problem of recycling drugs totally and it remains possible for a prisoner to maintain a supply by illicit means.

[43] Police investigations following the discovery of the Suboxone tablets in Mr Clow's cell under his pillow did not reveal how the drug had come to be in his possession. However, a fellow inmate told one of the prison officers that Mr Clow had been accessing the drug on a regular basis.

[44] I was satisfied that robust systems are in place in Low Moss prison to prevent the introduction of illicit drugs into the establishment, insofar as it is possible to do so.

Procedures are constantly kept under review and new, increasingly sophisticated practices introduced in an effort to reduce the incidence of contraband, including illicit drugs, into the establishment. This is an on-going problem world-wide. The re-cycling of prescribed medication among prisoners – particularly Suboxone – is likewise a continuing problem within prisons generally. I was satisfied that the strict dispensing procedures together with the strict discontinuation policy for those caught concealing the drugs are likewise robust measures which will go some way towards reducing the problem. Neither the procurator fiscal depute nor Ms Phillips on behalf of the prison service invited me to make any findings under section 6(1)(c) or (d) in this connection. I am satisfied that none should be made.

Hospital treatment

[45] While he was in prison, Mr Clow had complained of having visual disturbances and feeling unwell. It was suspected that he might have suffered a mild stroke and an urgent referral was made to the minor stoke clinic at Stobhill Hospital on 31 October 2014. As part of his examination, a routine ECG (electrocardiogram) was carried out to assess his heart function. The technician who carried out the ECG noticed an abnormality in the test and spoke to Dr Dunn, consultant cardiologist, who was nearby holding a cardiology clinic. Dr Dunn looked at the ECG and saw that Mr Clow had had a heart attack in the past, although it was not clear when it had occurred. Because of this, and having regard to Mr Clow's relatively young age, Dr Dunn decided to speak to him. He carried out a limited physical examination during which he heard a murmur in

Mr Clow's heart. He asked the technician if, as a favour, he would carry out an echogram that afternoon. This would routinely have taken several weeks to arrange.

[46] The technician obliged and the ultrasound test disclosed a number of abnormalities. It confirmed that one of the heart valves was clearly abnormal and had a significant narrowing described as moderate to severe. It demonstrated that part of the heart muscle was not working as well as it should and was indicative of a previous myocardial infarction. It also showed evidence of hypertrophy – the thickening of the heart muscle. Having reviewed the test results, Dr Dunn spoke to Mr Clow and explained the situation to him. He also telephoned the GP at Low Moss, discussed the medication with him and informed him that Mr Clow would be referred for further investigations.

[47] Although two separate but significant cardiac issues had been identified which required further investigation, Dr Dunn was comfortable at that time that Mr Clow did not require emergency admission to hospital. He had none of the symptoms suggestive of heart failure that would have precipitated such an admission – fluid on the lungs, breathlessness, chest pain at rest, syncope (fainting). Cigarette-smoking and high blood pressure were additional areas of concern given the extent of his heart disease. Dr Dunn referred him back to the cardiology clinic as soon as an appointment was available.

[48] In January, Dr Dunn still had Mr Clow in his mind. He had not come to any of his clinics. In the meantime, Mrs Clow had become concerned that her husband had heard nothing further about an appointment. She was becoming increasingly anxious and frustrated. Indeed, she was so concerned that she contacted the Health Board and

even raised the matter with her local MP. She then got in touch with Dr Dunn's secretary and spoke to Dr Dunn himself to express her concern that her husband had not been seen. Dr Dunn, on further investigation, discovered that Mr Clow actually had an appointment for 21 January with another consultant. He asked Mr Clow to cancel that appointment and added him to his own already over-booked clinic on 9 January 2015. He was anxious to see Mr Clow who was a relatively young man who required prompt investigation albeit he did not present as an emergency. It was the contact from Mrs Clow that, as much as anything, had prompted Dr Dunn to see why Mr Clow had not come back to his clinic.

[49] There are National Health Service standards and targets. The target for an out-patient appointment from time of referral is 18 weeks. The 21 January appointment was still within than timescale. However, Dr Dunn was keen to see Mr Clow and brought his appointment forward.

[50] Dr Dunn saw Mr Clow at his clinic on 9 January and had a long discussion with him about the various abnormalities in his heart. Dr Dunn felt that Mr Clow should have further detailed investigations which were not available at Stobhill Hospital. He referred him for coronary angiography. Although Dr Dunn would have been referring Mr Clow for further investigations in respect of his coronary artery disease, it was the aortic valve stenosis that was the major prompt for referral. Dr Dunn undertook to try to get these done as quickly as possible and, that day, telephoned the Golden Jubilee Hospital. He spoke to one of the consultants there, emphasising how keen he was to have these investigations carried out as soon as reasonably possible. Although the usual

waiting time for such an appointment for coronary angiography would have been about two months, Mr Clow was accommodated and given an early appointment for 26 January. Dr Dunn, in the meantime, issued Mr Clow with a handwritten prescription for simvastatin to lower cholesterol and wrote to Mr Clow's GP.

[51] Responsibility for Mr Clow's care then passed to the Golden Jubilee Hospital. Dr Rocchiccioli, Consultant Cardiologist, saw Mr Clow. It was decided that before any decision was taken as to the surgery, a CT angiogram was required. From a conversation Mrs Clow had with her husband, she understood that this procedure should happen quite quickly. However, nothing further happened between the end of January and April. Mrs Clow, understandably, was becoming more anxious and increasingly frustrated. She made several telephone calls to Dr Dunn's secretary, wrote to Dr Dunn and telephoned the Golden Jubilee Hospital.

[52] In response to her letter, and on making further inquiry, Dr Dunn discovered that the delay was due to a misunderstanding as to who was to order the tests. On checking Mr Clow's notes, he saw a letter from Dr Rocchiccioli advising him that he wished Mr Clow to have a CT of the aorta before proceeding to surgery (this is a non-invasive test designed to give the surgeon more detailed information about the aorta). Such a test was not a standard one and was not one that would normally have been done at Stobhill. It was not clear from the correspondence where the test was to be done and who had the responsibility to arrange it. Dr Dunn had not seen the letter before.

[53] On realising that there had been a misunderstanding and that some delay had been incurred, Dr Dunn immediately telephoned Dr Rocchiccioli. The latter made

arrangements for the test to be carried out at the Golden Jubilee Hospital on 2 April and, to save time, he arranged for Mr Clow to be seen by the surgeon on the same day rather than requiring him to wait for another appointment. His appointment on 2 April was within the Health Board guidelines for cardiac surgery. A CT appointment should be within six weeks. The appointment with the surgeon to decide a plan should be within 3-4 weeks after that. Once a decision has been taken to proceed with surgery, the operation should be scheduled within 12 weeks.

[54] Dr Flapan was instructed by the Procurator Fiscal to review Mr Clow's care, with particular reference to the timescales involved. He produced a report and gave evidence to the effect that he found nothing wanting in that connection. He found that the recording of the history and symptoms was very thorough and carefully done. All investigations were appropriate and undertaken with proper recognition of urgency. He considered the period from late October to early January to be "a pretty good referral time" and one which had required particular effort and active attention to detail on Dr Dunn's part.

[55] Dr Flapan discussed the need for a CT scan of the aorta. While not all cardiologists would consider such a test necessary and would be content to proceed on the basis of the echocardiogram which did not suggest significant aortic root dilation, it very much depended on where and when the cardiologist in question trained. Moreover, where there is congenital aortic valve disease, there is a well-recognised association with enlargement and dilation of the aorta. A referral for CT of the aorta to exclude significant aortopathy was therefore a very sensible thing to do although it

would have introduced some delay simply because it required to be organised. The question was whether the aorta itself would require treatment. If so, then that would have changed the magnitude of the surgery required making the whole intervention more complex. This, too, would have had implications for the consent procedures. In the event, the urgent steps taken by Dr Rocchiccioli to ensure that the test and the surgical consultation were scheduled for the same day meant that no actual delay occurred.

[56] Dr Flapan concluded that Mr Clow had received appropriate care at all times. His investigations had progressed timeously and had been thorough and processed with a degree of urgency. He would have received his surgery well within the six month absolute guarantee. At no time were there any signs or symptoms that Mr Clow should have been admitted as an emergency. It was appropriate that the investigations were progressed on an elective basis. The consultation with Dr Quinn (a locum consultant surgeon) on 2 April was thorough. Dr Quinn had made a thorough assessment and again there were no symptoms that would have precipitated emergency rather than planned surgery.

[57] The procurator fiscal invited me to conclude that there was nothing more that could reasonably have been done in terms of Mr Clow's treatment in accordance with the independent review by Dr Flapan. I was asked to make no findings.

Conclusion

[58] Dr Dunn impressed me as a highly dedicated and caring physician. On several occasions, he went out of his way to facilitate Mr Clow's care and treatment. Initially, Dr Dunn came out of his clinic, and gave advice about a patient at a separate clinic. It did not stop there. Dr Dunn not only took time out of his own clinic to talk to Mr Clow, but arranged (asking a favour of a colleague) to have an ultrasound test done there and then. That favour in itself saved several weeks, if not months, of waiting time. Again, that same day, after the ultrasound results were available, Dr Dunn spoke at some length to Mr Clow taking time to explain the situation. Dr Dunn arranged an appointment for Mr Clow to come to one of his clinics. At that stage, Mr Clow's treatment could not, in my view, have been better.

[59] Dr Dunn had been satisfied that Mr Clow did not require to be admitted to hospital. Although he had a number of serious heart conditions, there were no symptoms that would have flagged up immediate dangers. Dr Flapan agreed with this. There was evidence of conditions that required further investigations and treatment but no "red flag" symptoms to cause immediate concern. Although serious, the pattern of Mr Clow coronary disease was considered to be stable. It was therefore entirely appropriate to carry out investigations on an elective, planned basis.

[60] Because of his relatively young age and the potential for corrective surgery, Dr Dunn took an interest in Mr Clow and had him in mind. As he was wondering why Mr Clow had not been to his outpatients clinic, he heard from Mrs Clow. He discovered that in fact Mr Clow had an appointment at the cardiology clinic on 21 January, but with another cardiologist. Dr Dunn responded to Mrs Clow's concerns and ensured that

Mr Clow was seen at his clinic, moving his appointment forward and accommodating him at an already over-booked clinic. That, again, was an impressive and prompt response by Dr Dunn who could easily have let the later appointment stand as both were within the National Health Service target waiting times. He was clearly taking a close interest in Mr Clow's progress.

[61] Having seen Dr Dunn, Mr Clow was referred for further investigation and treatment to the Golden Jubilee Hospital and to Dr Rocchioccoli. Again, Dr Dunn took a particular interest in his patient and telephoned the Golden Jubilee cardiology department to speak to one of the cardiologists there, stressing the need for Mr Clow to be seen urgently. In response, an urgent appointment was given and he saw Dr Rocchiccioli some 12 days later on 26 January for an angiogram procedure. This was considerably earlier than the normal two month target waiting period.

[62] It was at this stage that some delay occurred because of a misunderstanding as to where and by whom a further test would be carried out. Mr Clow, by this time, was a candidate for surgery – most likely a coronary artery by-pass graft operation and a surgical valve repair. Although Dr Rocchiccioli considered that a CT scan of the aorta should be done to exclude significant aortopathy, he did not delay in referring Mr Clow to the cardiac surgeons to consider aortic valve replacement and coronary artery bypass graft. An appointment was made for 2 April. Again, it was not until Mrs Clow's concerns were voiced and acted upon by Dr Dunn that it was realised that no-one had made arrangements for the CT. The terms of the letter (which had not gone to Dr Dunn but had been received by one of the other cardiologists at Stobhill Hospital) were

slightly ambiguous. While it may have appeared that the test would be instructed by Stobhill, such tests were of a specialist nature and were not routinely carried out at that hospital. They required to be performed at the Golden Jubilee. Once Mrs Clow had raised her concerns, Dr Dunn again intervened and contacted Dr Rocchiccioli, highlighting the delay that had occurred. In response, an arrangement was made whereby Mr Clow was given an appointment for the CT aorta on the same day as he was due to see the cardiac surgeon. Thus, any potential delay caused by the misunderstanding was avoided.

[63] Mr Clow was seen by Dr Quinn on 2 April to discuss surgery. Sadly, he died during the night following that consultation. It was clearly a matter of great regret on Dr Dunn's part that Mr Clow died whilst awaiting surgery – surgery which might well have been successful. These sentiments were echoed by Dr Flapan. They recognised that the fact that Mr Clow, had he lived, would have had his corrective surgery within the six month target period would provide no comfort to his wife and family.

[64] The target waiting periods established by the Scottish Government were met in Mr Clow's case. I consider that there were no shortcomings in the care that was afforded to Mr Clow. On the contrary, he received a very high standard of care, from Dr Dunn in particular. I am equally satisfied that at no time were there signs which would have precipitated Mr Clow's emergency admission throughout the period of investigation. Sadly, the harsh reality is that some patients die while awaiting surgery.

Concluding remarks

[65] I am grateful to the procurator fiscal depute, Ms Macfarlane, for her efficiency and focus in presenting the evidence and for carrying out further investigations and calling additional witnesses to address matters which arose during the course of the Inquiry.

[66] It remains for me to express my condolences to Mrs Clow and her family. Mrs Clow had persistently advocated on her husband's behalf and prompt referrals at various stages were very much as a result of her interventions. She and other family members listened with dignity and fortitude to the evidence led before the Inquiry. I hope that their concerns have now been put to rest.