The Sheku Bayoh Public Inquiry

Witness Statement

Prof Mary Sheppard

Taken by [redacted]

Via MS Teams

on Wednesday 18 January 2023

Witness Details

1. My Name is Mary Sheppard. My contact details are known to the inquiry.

2. I am a Professor of Cardiac Pathology at St George's, University of London. I am head of the cardiac pathology unit that undertakes examination of the heart in cases of sudden death in the UK.

Professional Background and Qualifications

3. I completed my basic medical degree and decided to specialise in Histopathology, getting the fellowship of the Royal College of Pathologists in

Signature of witness ..... [redacted] .....

1986. I pursued an academic career specialising in lung and heart disease at the Brompton Hospital. I also did post graduate studies with a Fogarthy postgraduate Fellowship to NIH Washington DC and Armed Forces Institute of Pathology, Washington DC in 1996.

4. I moved completely into cardiac pathology in 2006 with funding from a family based UK charity called CRY. Overall, I have 30 years' experience in cardiac pathology. I am the only person in the country completely specialising in cardiac pathology. I have been asked if I am a forensic pathologist. No, I am not. I am purely cardiac. I specialise in both adult and child heart disease.

5. Since my appointment at St George's Hospital, I have undertaken extensive research and teaching with an emphasis on sudden cardiac death. I have been appointed as national trainer for cardiac pathology for forensic and general pathology trainees in UK. I have published 460 peer reviewed papers, 71 case reports, 24 book chapters, 36 reviews/letters/articles, 9 international guidelines/government reports. In addition, I have co-edited one book, co-authored four books including cardiac pathology and imaging, pulmonary pathology and cardiac valve disease and has completed the third edition of my book on Cardiovascular Pathology published in 2022.

6. I am the Cardiovascular Pathology representative on the specialist advisory committee of Royal College of Pathologists. I am a member of the Editorial Board and subeditor (cardiac pathology) for Histopathology and Editorial board member of Cardiovascular Pathology. I am President of the Pathology Section of the Royal Society of Medicine. I am also an advisor to governmental bodies in relation to cardiovascular pathology.

Signature of witness …… [redacted] ……

7. I also co-wrote the guidelines on investigation of sudden cardiac death published by the Royal College of Pathologists with Kim Suvarna from Sheffield, and they were recently updated in 2022.

8. I am also on the National Steering Group developing a national database on sudden cardiac death. We are initiating a national programme for screening of families who have suffered a cardiac death because most of the causes are genetic and this started in 2022 and will continue into 2024.

Explanation of Heart Slides

9. I have been asked if I recognise the document "Blocking diagram of the heart" (WIT-00002). Yes, I recognise that diagram - this is a diagram where the heart histology slides are labelled according to where they have been taken from the heart. The pathologist has given them specific labels for the area. Then I can identify the area from the letters on the slide when I look at the slides down the microscope.

10. This is usual practice for how slides are marked. If you've got an autopsy where you have had a sudden cardiac death, this is the routine slides I would advise taking from the heart in all cases, according to the Royal College of Pathologists guidelines as well.

11. I have been asked what is usual practice in a case of sudden death: whether slides of the heart are prepared and sent to me for examination or whether I am sent the whole heart for examination. It is appropriate to do either. The pathologist can prepare the slides themselves at the autopsy and take the appropriate blocks, or they can keep the heart, fix it and send it on to an expert like me. They had the choice to do either.

12. I have been asked if I feel disadvantaged in any way by not having the opportunity to examine the whole heart. No, not with the sampling that was done in this case.

Signature of witness ..... [redacted] .....

Simplified explanation of the heart structure and function

13. I have been asked if I could explain in layman's terms how the heart works. The heart is the organ that helps supply blood and oxygen to all parts of the body. It is divided by a partition (or septum) into two halves. The halves are, in turn, divided into four chambers. The Atria are the two upper chambers of the heart and the ventricles are the two muscular lower chambers of the heart. The ventricles are the two big muscles that pump the blood into the lungs from the right ventricle and from the left ventricle into the rest of the body. The ventricles are the most important chambers.

14. There are four main blood vessels that take blood into and out of the heart. The aorta is the largest artery in the body. It carries oxygenated blood away from the left ventricle to the body. The vena cava is the largest vein in the body. It carries deoxygenated blood from the body back to the heart. The pulmonary artery carries deoxygenated blood away from the right ventricle to the lungs. Lastly, the pulmonary vein returns oxygenated blood from the lungs to the heart.

15. The heart is situated within the chest cavity and surrounded by a fluid-filled sac called the pericardium (essentially, the surface that's overlying the heart). The heart generates electrical impulses that cause the heart to contract, pumping blood throughout the body. The cardiac conduction system plays an important role in causing the heart to contract. It is comprised of the SA node (Sinoatrial node), the AV node (Atrioventricular node), the bundle of His (also known as the Atrioventricular bundle) and the Purkinje fibres.

16. In my report, I make reference to the first of these 3 components. I will explain their functions in very basic terms. The SA Node is a section of nodal tissue that sets the rate of contraction for the heart. The AV node is a section of nodal tissue that delays and relays cardiac impulses. The bundle of His is

Signature of witness ..... [redacted] .....

a continuation of the specialised tissue of the AV node, and serves to transmit the electrical impulse from the AV node to the Purkinje fibres of the ventricles.

Annotated Medical Report

17. I have been referred to my report in this case [redacted]. At page four of the report I speak about "microscopic examinations", and I have been asked if this paragraph is my description of my own examination of the individual histology slides of the heart. Yes, it is.

18. I looked at all the sections of both the right and the left side of the heart, as was mapped out by the pathologist. I am seeking to look for and eliminate common causes of cardiac sudden death. I say in this section that there is no fibrosis. Fibrosis is scarring. Scars replacing the myocytes. There may also be inflammation with inflammatory cells in the heart muscle. Inflammation is called myocarditis and can cause somebody to die suddenly. Arrhythmogenic right ventricular cardiomyopathy is a particular type of damage to the heart, which occurs in the right or left ventricle and can be a cause for sudden death. I look, particularly, at the right or left ventricle for this and did not find it. I also look for myocardial infarction. That means death of the muscle due to the coronary artery being blocked. Infarction results in the cutting off of the blood supply to the heart. Again, I could see no evidence of infarction, which is due to coronary artery disease.

19. The overlying pericardium was normal. There were some scattered lymphocytes (a type of white blood cell) which is a normal feature. Both ventricles showed hypercontracted myocytes. A myocyte is a particular length, and when they contract, they become shorter. When you have a cardiac arrest, all your myocytes contract and seize up. They go into spasm, and they are hypercontracted. This is classic of cardiac arrest, particularly cardiac arrest under stress. When I say cardiac arrest under stress, I am referring to stress brought on the body by physical exercise or exertion. Since Mr Bayoh was being restrained by the police that would be in-keeping with the

scenario of stress. I saw no evidence of pathology like myocyte disarray or hypertrophy to indicate hypertrophic cardiomyopathy.

20. I have explained that the ventricles are the most important chambers. The atria don't really cause you to die suddenly. It's the ventricles that are the main killers. If they have damage or a cardiomyopathy, then they could cause a sudden cardiac death., The pathologist has sampled the heart at midventricular level. He has cut across both ventricles and sampled both ventricles.

21. I have been asked what lymphocytes are. These are chronic inflammatory cells. They act in your immunological system, combating infection, and are quite common throughout the body. Small little clusters of them are there to guard the heart from infection, and so they're a normal finding in everybody's heart.

22. I have been referred to the line in the report *"There are also white cells: leukocytes within dilated capillaries."* Leukocytes are inflammatory cells as well, with label of white cells which are usually the lymphocytes. Again, they guard against infection but often, when you're in cardiac arrest, the white cells accumulate within the dilated capillaries. It is nonspecific evidence of cardiac arrest, and is not indicating any infection or specific pathology. The white cells accumulation is very typical also when somebody is resuscitated.

23. I have been asked to explain the line white cells are within dilated capillaries with blood vessels with surrounding interstitial oedema. Fluid leaks out from the capillaries as a terminal event as they become leaky, leading to oedema. This is a nonspecific finding with cardiac arrest and is not indicative of infection in my opinion.

24. Continuing on page 4, I have been referred to the line where I state "*These changes are typical of resuscitation. Sections of the left ventricle show no*

Signature of witness ..... DocuSigned by: [redacted]

evidence of significant myocyte disarray to indicate hypertrophic cardiomyopathy." I have been asked to explain what hypertrophic cardiomyopathy is. It is a genetic condition that causes sudden death and is one of the most common causes of sudden death in young people. It's an innate abnormality of the heart muscle leading to thickening of the muscle and it causes a cardiac arrest, often with no warning or symptoms. You may not be aware you have the condition, and its genetic. Again, I'm ruling out this as a potential cause of sudden cardiac death based upon examining the histological slides in this case.

25. I have been asked to explain what myocyte disarray is. That's where the muscle cells of the heart are disorganised. Myocytes usually work together in parallel with each other. They work together side by side like soldiers in an army, all marching together in step. With disarray, they go interfering with one another, and they cross over one another, and they prevent each other from working properly. That explains the cardiac arrest.

26. I have been referred to the line *"there's myocyte disarray in the right ventricle."* I have been asked to explain this. Myocyte disarray is normal in the right ventricle. Disarray can occur normally in parts of the heart when you look down the microscope as in the right ventricle. Myocyte disarray is seen in the right ventricle and anterior/posterior walls where the right and left chambers intersect with each other, but this is where it's found normally. Pathologists can misinterpret findings in the normal heart because they're not familiar with the microscopic findings in the normal heart. So, this myocyte disorganised disarray is quite it's normal in the right side of the heart and in these anteroseptal/posteroseptal walls. I accept these findings as normal. They are not evidence of pathological myocyte disarray and hypertrophic cardiomyopathy in these sections examined.

27. I have been referred to the line *"there are no atrophic myocytes or fibrosis to indicate dilated cardiomyopathy".* That is the third type of cardiomyopathy:

Signature of witness ..... DocuSigned by: [redacted]

arrhythmogenic is the first; hypertrophic in the left ventricle; and then dilated cardiomyopathy, where the left ventricle in the heart dilates. This is a cause of sudden death and the person may also not know they have it as in the other types as well. I found no histological evidence of dilated cardiomyopathy in the sections examined.

28. I have been asked to explain what I meant when I wrote *"There are no giant cells or granulomas noted."* You can have entities called giant cell myocarditis or sarcoidosis which cause sudden cardiac death. The finer detail of these conditions are irrelevant here; I looked for evidence of these entities which can cause sudden death. Granulomas indicate sarcoid which are a particular inflammatory collection of cells in sarcoidosis. It involves the lungs and the heart and maybe other organs in the body. It's a condition of unknown cause and it can affect the heart, causing these inflammatory foci called granulomas. I found no evidence of these in the heart.

29. I have been referred to the line *"There is no evidence of acute or chronic infarction."* Infarction is when the heart dies or parts of the heart die due the cutting off the blood supply in the coronary arteries usually.

30. I comment that the Epicardial vessels are normal. These are the vessels on the surface of the heart in the epicardial layer. That's where the main vessels surround the heart and embed it and lock the heart in. From these vessels, all the other vessels go in to supply the muscle, and they're very, very important because the muscle of the heart is so demanding. The smaller vessels going into the muscle wall are also normal.

31. I have been asked to explain vasculitis or emboli. Vasculitis is inflammation of the blood vessels that can cause them to block up. There was no inflammation. Emboli is where the blood vessels are blocked by material labelled as embolus, a clot or foreign material that blocks the vessel, which

Signature of witness ..... [redacted]

could cause infarction. I saw no evidence of any emboli, be it a blood clot or thrombus or foreign material which you can get with IV, drug abusers, etc.

32. I have been asked what replacement and interstitial fibrosis are. That means scarring in the heart. There are no scars in the sections examined. You can sometimes get fibrosis from unknown causes in the heart or due to drug use or toxics or chemotherapy, but I found no evidence of scarring in the right or the left ventricle.

33. I have been referred to the line *"There is no evidence of abnormal infiltrate such as amyloid"* and asked to explain what it means. Amyloid is a particular dense material that is deposited in the heart in some people, mainly elderly, and it's made up of fibrils due to inflammation, or due to tumours, or it may be an idiopathic unknown cause. Amyloid is a well known abnormal material that gets into the heart and stops the heart functioning i.e. causes a cardiac arrest. There is no amyloid in these sections examined.

34. I have made reference to the atria. The left and right atrium are the upper chambers of the heart. I got some histology of the atrium. The upper chamber was included, and I found nothing wrong with the upper chamber - the atrium.

35. I have been referred to the line *"it shows normal morphology with no significant inflammation or fibrosis. Sections of the conduction system show the SA node and sections"*. The SA node is the pacemaker in the upper chamber. It's in the right atrium. The pathologist dissected out that area for me to look at in these sections. It looked normal. I have also been asked about what I meant in my sections and when I say, "not in 'W' or 'X'." Yes. These were further sections taken of the conduction tissue, but sometimes you don't see it because it's an irregular shaped object. As a result, some of the sections will show it and others won't.

Signature of witness ..... [redacted]

36. I have been referred to the line where I speak about *"sections 'J, I, K, L, M, N, O and P' do not show the AV node, bundle of His, or dividing branches. I would advise doing deeper sections of the blocks 'J' to 'P' in order to see the components of the AV node."* I have been asked what would be the benefit of looking at this in this way. The AV node is highly unlikely to yield the cause of death. The blocks of tissue that were taken do not show it. That's quite common because it's invisible to the naked eye. The pathologist may find it difficult to block it out. He just takes a blind block of tissue in the area where he or she thinks the AV node is, and it's quite a complex area of the heart, so you may not see it because the blocking misses it. It's quite a difficult dissection to do. I am not surprised that it's not there, and it wouldn't make any difference to me, generally in coming to a diagnosis of the cause of death. I have never had the AV node cause death, except for one unusual tumour called an AV nodal tumour, and that would be highly unlikely in this man. I'm not worried about missing it here as it is so rare.

37. I have been asked what doing "deeper sections" would entail. The deeper sections may show up the node. If the pathologist or the technician in the laboratory goes deeper into the blocks, it may show the components of the AV node. As I explain, it is a difficult dissection to do. It wouldn't yield an important cause of death or change my opinion.

38. I am shown my summary section which states *"This young man has a heart which is within expected weight of the patient (in males heart is 0.43-0.51% of total body weight). The heart is morphologically normal. There is no evidence of damage in the right or the left ventricle which would indicate the use of cocaine. He has no evidence of an inherited cardiomyopathy which may be responsible for his sudden death. He has no evidence of myocarditis or any abnormal infiltrate to explain his sudden death. He does not have evidence of coronary artery disease or myocardial infarction to explain his sudden death."* I understand that Mr Bayoh was very muscular man and worked out at the gym, that he'd been taking steroids and so he had quite a

Signature of witness ..... DocuSigned by: [redacted]

lot of upper body bulk, although his heart size was not increased. With steroid abuse, the heart can increase in size. There can be increase in heart weight – what we call cardiac hypertrophy – but Mr Bayoh had no evidence of this. Myocarditis is inflammation of the heart. Inflammatory cells that I've talked about. These are different types of inflammatory cells. Myocardial infarction is known as a heart attack with damaged muscle due to lack of blood supply.

39. I have been referred to the next part of the summary section which says *"Sections of the conduction tissue show the SA node, which is normal, but the sections of the AV nodal area do not show the major components of the AV node, including the AV node itself, bundle of His and divided branches. This is a very well sampled heart with all the major components being demonstrated apart from the AV node."* I have been asked what the AV Node and the bundle of His is quite a complex structure. It's invisible, and quite often it cannot be identified when the pathologist takes the area. It takes quite a lot of work to be able to dissect out the AV node, so I'm not surprised that I cannot see it because it's a tiny, tiny little structure. I have been asked if I would expect to see a cause of death for sudden cardiac death arising from these areas. No, very rare, extremely rare. One needs a history of heart block, where your heart impulse is blocked. Mr Bayoh has no clinical evidence of that or of cardiac arrhythmias. So it's highly unlikely that there is an abnormality in the AV conduction tissue to explain his death. I've only found a conduction disease cause of death in 0.01 per cent of my 7000 cases and, in those cases, the patient has a history of arrhythmias or of heart block.

40. I have been referred to page 5 of my report where I say, *"While the drugs may have an effect on the heart, there is no evidence, pathologically, of any damage to the heart due to drugs."* I have been asked what sort of effects I would expect to see when the drugs Mr Bayoh had taken where MDMA and Alpha-PVP and if I were to see effects on the heart what would that be. Normally I'm dealing with individuals who have taken cocaine showing infarction or fibrosis. With MDMA and Alpha-PVP, I don't know. Normally,

Signature of witness ..... [redacted]

drugs cause infarction or fibrosis, or acute damage like myocarditis. I found none of these changes in this case. There was no evidence from a pathology point of view indicating drug use. It may be that MDMA and Alpha-PVP may cause arrhythmias and sudden death, but I found nothing pathologically, so I cannot comment.

41. I have been referred to the line, *"while the CS and PAVA may have an effect on the heart, there's no evidence, pathologically, of any damage."* I've not come across any evidence of pepper spray or given a case where it's damaged the heart. In Mr Bayoh's case, this was a normal heart so there's no specific finding that I can point to and suggest it may have been caused by CS spray or PAVA spray. Certainly, I can confirm that there was no sign pathologically of any damage to the heart caused by CS or PAVA spray.

42. I have been asked to explain how restraint is linked to sudden cardiac death. The pathologist fins at autopsy that the heart's is normal, and everything else is normal. This is SADS, Sudden Adult Death or Sudden Arrhythmic Death. Where restraint has been involved, proving the restraint or how long the individual was restrained for and how Aare important forensic considerations. All I can say as a cardiac pathologist and not a forensic pathologist – is that the heart was normal.

43. I have been asked how I was able to establish a link between restraint and sudden cardiac death. It is the circumstances of the cases. In the majority of cases, where the heart has been sent to me for examining and the individual died during or following restraint, I find the heart is normal. So we are presuming that they died due to cardiac arrhythmia combined often with a sublethal level of drugs on board and sublethal level of alcohol, and also severe stress of being pursued by the police or restrained by the police.

44. I have been asked in terms of my expertise if I am able to comment on how restraint might affect the heart in terms of causing arrhythmia. Restraint can

Signature of witness ..... [redacted]

affect breathing. This can cause hypoxia. Obviously an expert on restraint who can provide a forensic opinion, will be better placed to comment than me. Various factors as relevant including the length of time the person is restrained, how it's applied and its effects on the ribs and on the chest and if the person is obese or not. If the restraint stops them breathing properly, the person can get hypoxia and hypoxia affects the heart, causing it to deprive it of oxygen in the bloodstream and causing it to arrest. There are multiple theories are out there about restraint, I defer to my forensic colleagues to talk about the hypoxic theory.

45. I am asked about the effects that MDMA and Alpha-PVP have on the heart. This is really a question for a toxicologist. I am aware that they can cause arrhythmias in the heart and have the potential for sudden death. However, as a cardiac pathologist, I cannot see pathological evidence of an arrythmia. The major one I see is cocaine in which one can see damage in the myocardium with fibrosis or infarction or coronary artery thrombosis. As I have explained, there is no evidence of damage in these cardiac that you would expect to see with a cocaine user.

46. I have been asked what effect an arrhythmia has on the heart. Depending on the arrhythmia, if the heart beats too fast or too slow, then it will fail to bring blood via the circulation up to the brain and you will go unconscious because of lack of blood supply to the brain and you will die within five minutes if you're not resuscitated. The heart has to contract 70 beats a minute to give you a blood supply to all your organs. If it contracts too rapidly, then the blood won't get through, and also if it's too slow. That's how it causes the cardiac arrest.

47. I have been asked what toxicological opinion was necessary in this case. Obviously, there are drugs involved , so a toxicologist's opinion is required to discuss the effects on various organs of these drugs: brain, kidneys, heart, etc. In the heart they cause arrhythmias, but I can't provide a full account of

Signature of witness ..... DocuSigned by: [redacted]

that because I'm not a toxicologist. As a pathologist, I found no evidence of any drug related damages.

48. I have been asked what conclusions I drew from the absence of any damage to the heart. That this man has died suddenly, under restraint, with drugs on board. He is a classic sudden death during restraint. However, whether the restraint caused it, has to be proven by the circumstances, and is a matter for a forensic pathologist. To me, he died of a terminal cardiac arrhythmia, because everybody finally dies of a cardiac arrhythmia, and your heart simply stops/arrests. However, the cause of that cardiac arrest is the key question and may be multifactorial. One of the matters for consideration is what place the restraint element had, in terms of my involvement, I can't prove anything. My role is to eliminate every other cause of sudden death from the cardiac point of view.

49. I have been asked that if restraint was a factor in Mr Bayoh's death, whether I would expect to see any pathological evidence in the heart if restraint was a factor in the cause of death. No, I would not expect to see that pathologically. I'm afraid, unfortunately, there is no way a cardiac pathologist can say someone died due to restraint. It is a diagnosis of circumstances and eliminating other causes.

50. I have been referred to page 5 of my report which says *"The combination of a, b, c [i.e the drugs, the CS and PAVA spray and restraint] in combination can be linked to sudden cardiac death and I have published recently on this in the literature. The sudden cardiac death causes are usually multifactorial, and no one cause alone is responsible for the death. There is no evidence pathologically of any damage to the heart."* I have been asked if I can explain how the combination of restraint, drugs and the CS spray and PAVA are linked to sudden cardiac death. It's a perfect storm. You're highly stressed, you're on drugs that can affect the heart, there is restraint, the CS Spray and PAVA may also cause an increased stress. I have published an article on

sudden cardiac death. This article is "Sudden cardiac death with stress and restraint: the association with sudden adult death syndrome, cardiomyopathy and coronary artery disease" (WIT-00024). The study highlights sudden cardiac death during psychological stress and restraint. These deaths were mostly in young males where the sudden death occurred in the absence of structural heart disease. But it's a combination, the perfect storm of the whole lot coming together, and the big issue is obviously seeing which factor predominates.

51. I have been asked what conclusions I draw from the absence of any damage to the heart when you have these three things (Restraint, drugs and spray) in combination. All I can say, is that his heart stopped but there's no underlying explanation in the heart to explain the stoppage of his heart, the cardiac arrest. So you have to take into account then his body mass – which is not an issue here; he's not obese – the fact of his drugs on board in the blood combined with the restraint, causing the possibility of hypoxia; lack of oxygen to the heart, causing the cardiac arrest. Beyond this, it is a matter for the opinion of a forensic pathologist and for the determination by the chair to the Inquiry.

52. In terms of what effect the CS Spray or PAVA would have on the heart, being sprayed with PAVA or CS may cause a release of adrenaline due to the stress, but then getting evidence of that in this individual case, it's impossible, However, showing evidence of that or evidence that CS spray or PAVA spray caused the death is impossible, from a cardiac pathology point of view.

53. I have been referred to the report where I state, again at page 5, *"The deceased had no cardiac abnormality identified at his death; however, this does not rule out sudden cardiac death due to an electrical abnormality, the cardiac channelopathies".* A channelopathy is an electrical channel that is present in individual myocytes. For the myocardium to work together to do the heartbeat, they all have to communicate, and they communicate through

these channels. If these channels have abnormalities, then they stop communicating and they start going all over the place. It's called a channelopathy, pathology of the channels. It's invisible to the pathologists and the heart is normal. I had no evidence of that in this case. To find evidence, you'd have to go back to previous ECGs of the person or of his family. DNA testing can be carried out but may not yield a result in most cases.

54. I have been asked what sort of DNA testing will show up a channelopathy. A DNA test would be positive only in about 20 per cent of cases. You may get negative results in 70 per cent, so it doesn't eliminate channelopathies, a DNA test can be useful if you find it positive, but if it's negative, it's not useful.

55. I have been asked if I would expect to see anything on the deceased's medical records that might suggest that he had had an ECG. Yes. that he had evidence of collapses in the past, fainting attacks, that would point towards the possibility that he had sudden cardiac blackouts; syncope; collapses; chest pain. These from the history would be useful. However, if there was no episodes of that nature, it does not allow you to exclude a channelopathy as 70 per cent of people who have a channelopathy are asymptomatic prior to death.

56. I have been asked how common or rare a channelopathy is. We don't really know the incidence of channelopathies. It's the most prominent cause of sudden death in my database. I cannot tell what percentage of people with channelopathies you would find in the population. You'd have to get a cardiologist to do that for you who has an interest in these inherited cardiac conditions.

Signature of witness ..... [redacted]

57. I have had sight of Dr Soilleux's report (COPFS-00031) and have been referred to paragraph 59 where she states *"It's unlikely that the CS and the PAVA sprays had a significant effect. However, the deceased had taken MDMA and Alpha-PVP – drugs that increase heart rate and increase the risk of a rhythm abnormality. Stressing a heart in this setting, for example by struggling against restraint, would very significantly increase the risk of a rhythm abnormality developing, which may well be what happened in this case."* I agree with what Dr Soilleux states here. Yes, it can happen. The drugs with stress can cause an arrhythmia. On the other hand, the alternative scenario suggested it is the restraint with hypoxia. Dr Soilleux goes on to state *"However, an alternative scenario is that there was a degree of positional or mechanical asphyxia (paragraph 64), meaning that less oxygen was able to get into the blood, while the oxygen requirements were probably increasing due to the struggling against restraint and possibly the effects of stimulant drugs (MDMA and alpha-PVP). This could have led to cerebral ischaemia (insufficient oxygen reaching the brain) and thus loss of consciousness. It could possibly also have led to myocardial ischaemia (insufficient oxygen reaching the heart), which may have predisposed to subsequent rhythm abnormalities and heart pump failure."* Either scenario can be put forward. There is nothing here that I disagree with. However, there is nothing that I saw in the heart pathologically that allows me to express an opinion as to which scenario is more likely. Again, it is for a forensic pathologist to provide an opinion on that.

58. I have been asked if I agree that the struggle against restraint additionally causes an increased risk of arrhythmia abnormality. I am sure it does, yes. While it is entirely possible, I am unable to prove it pathologically but it is likely if they're fighting against the restraint that the persons' adrenaline levels are going to be very high.

Report of Dr Karch

Signature of witness ……[redacted]

59. I have been referred to Dr Steven Karch's report (PIRC-02526(a)). On page 1 he states that he saw histological abnormalities and concluded that the Mr Bayoh suffered from *"pre-existing heart disease that could have been fatal in its own right."* I am told that he's reviewed the same slides that I have reviewed. As I have already stated, I am quite clear that Mr Bayoh did not have pathological heart disease.

60. At page 4 of his report, he states *"My microscopic examination disclosed remodeling changes throughout the heart. These changes included the presence of enlarged heart cells with abnormal cell nuclei and abnormal fibrous tissue located between the heart cells and around blood vessels."* What he's describing to me in the second sentence is normal. He just doesn't know the normal heart histology microscopically. In terms of the remodelling changes he refers to, I believe these "remodelling" changes are a normal feature of the adult heart. I've seen 7,000 hearts in my lifetime with microscopy in all of them. He is entitled to his opinion, but what he is describing in my opinion, is seen in the normal adult human heart and the fibrous tissue described is not actually fibrous tissue but is the normal structural collagen network which support the myocytes and blood vessels. The enlarged heart cells are also a normal feature of adult human hearts in which there is a wide variation in shape and size of the adult human heart myocytes.

61. This section at the end of page 4 continues *"The blood vessels themselves were abnormally narrowed and many of the vessels were surrounded by abnormal fibrous tissue that would act to diminish blood flow to the rest of the heart, favoring arrhythmia and sudden death."* I can understand what Dr Karch is describing in the blood vessels in the histology. However, he is over interpreted the normal variation in blood vessel lumen and wall thickness within the normal heart. These variation in blood vessels size and shape is a normal feature in the human heart.

Signature of witness ..... DocuSigned by: [redacted]

62. It is important to understand, that I've seen lots of normal hearts, which other pathologists can interpret as being abnormal. I suspect that this is why Dr Karch can say, “I see remodelling. I see this, I see that, and I think it's all due to drugs.” Well, he'll find it very difficult to prove it's due to drugs in discussion with other pathologists. I see contraction band necrosis in one in five hearts that come to me every day and they're not on drugs and they' have been resuscitated and we have published on this. Pathologists may not be familiar with the pathological changes of resuscitation. This is what I lecture to pathologists all the time: do not over interpret what you're looking at.

63. I am informed that Dr Karch is an American cardiac pathologist. Dr Karch is claiming that there's remodelling and there's fibrosis and there's damage in the heart and that it's due to drugs. I would go into a court of law and say, “No, I would disagree with you.” When I looked at this heart, it was normal and displayed resuscitation affects. What Dr Karch is describing are normal features in a normal heart that he interprets as being pathological. What I would say because the size of myocyte fibres, the nuclei can vary dramatically between normal individuals.

64. Dr Karch describes uneven staining pattern of the myocardium, fragmentation and waviness of fibers, perivascular connective tissue growth, intramuscular fibrosis and scarring, disintegration of cardiomyocytes, nuclear disintegration, loss of cross-striations and thickening of blood vessel wall. This is all describing normal findings or changes due to resuscitation. It does not prove drugs misuse. Dr Karch is over interpreting what to me, are normal changes or resuscitation changes. He states the blood vessels are thickened. They are not; they are within a normal spectrum. The blood vessels within the heart vary considerably and can look thickened.

65. I can produce multiple sections of hearts with thickened blood vessels within the wall and they're normal. Microvascular disease, causing sudden death, is very controversial, and not established pathologically.

Signature of witness ..... [redacted]

66. I am an acknowledged national and international expert in cardiac pathology. I see 12 hearts per week, and I see histology of every one of those. I'm aware that the original pathologists found the heart normal. I'm advised that Dr Nat Cary and Dr Jack Crane also examined the heart histology and found it was normal.

67. In relation to question e *"the significance of the effect of the deceased being handcuffed to the front and leg restraints applied and whether such a restraint could have contributed to any positional asphyxiation, given the fact the restraints continued to be applied following deceased becoming unresponsive and subsequently during and follow resuscitation attempts."* I would defer to a forensic pathologist in relation to this question.

68. In relation to question g, *"The fracture to the deceased rib as outlined In page 9 and 15 of the post mortem report and the most likely mechanism as to how this Injury was sustained",* I did not to answer this question in my report as it is outside my expertise.

69. I believe the facts stated in this witness statement are true. I understand that this statement may form part of the evidence before the Inquiry and be published on the Inquiry's website.

March 20, 2023 | 3:13 PM GMT

Date.....

Signature of witness .....