The Sheku Bayoh Public Inquiry

Witness Statement

Dr Steven Karch

Taken by [redacted]

Via MS Teams

on 26 January and 20 February 2023

Witness details

1. My name is Steven Karch. My contact details are known to the Inquiry.

2. I am a cardiac pathologist and toxicologist. I have been in practice for more than 50 years.

Professional Background and Qualifications

3. My qualifications are MD, FAAFP (Fellow of the American Academy of Family Physicians), FFFLM (Fellow of the Faculty of Forensic and Legal Medicine), FCSFS (Fellow of the Chartered Society of Forensic Sciences).

4. I attended undergraduate school and earned a Bachelors degree in philosophy from Brown University in Providence, Rhode Island. I did postgraduate studies in Biochemistry and Human Anatomy at Stanford University for a year although I didn't complete the year. I left Stanford to go to Tulane University Medical School

in New Orleans, where I received a Medical Doctorate. This is our standard degree for medicine in the United States.

5. I did a Fellowship under Professor Henry Urich in neuropathology in 1971 at the Royal London. I left London after a bit more than a year and returned to the United States. I then did additional training in Neurology at Stanford University. However, I didn't earn any qualifications from that study. I had to leave because of health issues.

6. Then I did general practice for 2-3 years. This role is the same as that of a General Practitioner (GP) in the UK. I found the role boring, which is why I left.

7. I did an unofficial Fellowship in Cardiac Pathology at Stanford University from 1983 to 1989. Stanford University hired my London mentor, Professor Urich, as the visiting professor in the Neuropathology Department at Stanford. He had been my professor in London, and he introduced me to a Cardiac Pathologist, Professor Margaret Billingham. I told her what my research interests were, and she offered me the opportunity to come work for her. It wasn't an official Fellowship. A fellowship is a generally supported portion of graduate studies. I did not receive any pay for my work, financial support or any qualifications for the work undertaken. I did these studies purely for my own learning. I did these studies in cardiac pathology one day per week.

8. It was research based on practice. I worked in the laboratory that supported the transplant programme at Stanford, which was the first transplant programme in the United States. It was also the first cardiac pathology laboratory in the world. I worked for Professor Billingham from about 1983 to 1989 as an assistant, my interest being resuscitation and the science of resuscitation. Many of the hearts I examined during this period were the hearts of transplant patients. A messenger would come from the airport everyday with heart slides prepared in Jordan or Singapore for example. We would read the slides and send back telegrams or letters saying what we found. Stanford had a very active Cardiology Department and it was not uncommon to do biopsies on living patients of their hearts and

read microscopic slides of the samples so obtained. During my time with Professor Billingham, I examined hearts both microscopically and macroscopically. I would go to the operating room and would stand next to a window where someone would hand me the diseased heart that had just been removed from the transplant patient. After I had gotten the heart, I would take it back to the laboratory and do the initial analysis and then would clear it with Professor Billingham. This period at Stanford University was my only cardiac pathology training.

9. Thereafter I held a few different positions. From 1989 to 1990 I was Director of Emergency Services at the University Medical Center in Las Vegas. I was responsible for educational activities and witnessing or assisting at autopsy of individuals who died in the trauma center. From 1990-1992 I was Director of Trauma Center Development, also at the University Medical Center, Las Vegas, Nevada. Then from 1993-1994, I was Director of Trauma and Quality/Outcomes Research, again at the University Medical Center, Las Vegas.

10. In 1995 I moved to the Medical Examiner's Office in San Francisco, which is similar to the Coroner's Office in England. I worked as an Assistant Medical Examiner in Cardiac Pathology on a consultancy basis for about 10 years – I worked here one day per week. I was not paid for this role. I was not in need of income and I was doing it for my interest in the science. Another day or two per week, I would write papers on cases or book chapters.

11. My role was to examine hearts, I was not responsible for total autopsies. Generally, here I examined prepared slides of heart histology and interpreted some. Once or twice a year, I might be called into the autopsy room and help them take the heart out if there was some gross abnormality. I would do any case the Chief Medical Examiner asked, but my interest was drug cardiotoxicity. San Francisco had, and still has, many drug deaths and I became interested in the subject of drug deaths and how the interaction between the drug and the heart caused death, deranged behaviour and other peripheral manifestations of drug abuse.

12. When Dr Stephens, Chief Medical Examiner, and my mentor died in 2005, I devoted myself to writing and teaching Cardiac toxicology for various organizations, including a yearly course on sudden death and poisoning for pathologists at the United States Army's Institute of Pathology. I also began a busy career as an independent consultant in cardiac pathology and toxicology.

13. Overall, I have 20-30 years' experience working in Cardiac Pathology. This experience comes from the 7 years of working at Stanford under Professor Billingham one day per week, 10 years of working one day a week as an assistant medical examiner and the remainder from working in private consulting practice. I have been asked how many hearts I have examined over the course of my career. It's difficult to say. Perhaps more than 1000.

14. I have board certification for family practice and for quality assurance physicians. Board certification is not mandatory in the United States, but it is generally considered to be a mark of distinction in your chosen specialty. U.S. law specifies that an individual qualifies as an expert by "virtue of knowledge, training, or experience." I am asked why I have Board certifications for these areas of family practice and quality assurance but not cardiac pathology or forensic pathology. There is now a Board of Cardiac Pathology, but there was no certifying Board until 1985 and insufficient members even to support a peer-reviewed journal online until 2022-2023. In relation to forensic pathology, it seemed to me it would be irrelevant. There are a lot of skills that I wasn't going to use. Generally, board certification was of little interest to me. I wasn't studying cardiac pathology and neuropathology with the goal academic advancement.

15. In terms of my toxicology experience, I have no formal toxicology qualifications or training as a clinical toxicologist. That is because I only practice post-mortem toxicology (and have published extensively in that area, most recently last December). I had taken biochemistry at Stanford as a graduate student for a year before leaving to start medical school. When I was working as assistant medical examiner, the head Medical Examiner taught me the toxicology

techniques (at the time he trained, toxicology proficiency was required for certification as a pathologist in the United States). He only taught me how to use the analytic equipment. He never gave me formal lectures, but he did explain cases as we processed them for court.

16. My position could be described as that of an apprentice, and I learned how to operate the equipment and prepare the materials for analysis. Occasionally I did the analysis, but there was no reason to, as we had about five or six analysts by the time I left.

17. When in private practice I undertook medicolegal reports in many cases.

18. I was retained by the U.K. Home Office to work alongside Dr John Rutherford in the Harold Shipman case. I testified at trial and helped with the investigation. I was instructed by the Crown to supply expertise in the toxicology of the Shipman related deaths. My role was examining the effects of drugs on the heart, ascertaining the viability of drugs surviving in the dead bodies, and suggesting alternative possible sources and explanations that might be used by the defence to explain the presence of morphine.

19. I was practicing until 2019 when I retired. I still have a handful of cases that need to be resolved or ongoing involvement in historic cases. I am not taking new cases. I have written more than a dozen books – some used internationally. I continue to write. My most recent book was published in December 2022 (Karch's Drug Abuse Handbook, Taylor and Francis Press, London).

Role of a Medical Examiner

20. I have been asked to explain the role of a medical examiner. A medical examiner is employed by the municipality so effectively by the local government for the city of San Francisco. In San Francisco, and American cities that have adopted the Chief Medical Examiner system, The Examiner is responsible for the medicolegal investigation and the certification of the cause of death and the manner of death

for only those deaths under its jurisdiction i.e. sudden, unexpected, or violent deaths. In the US, a forensic pathologist and general pathologist both undertake autopsies. Forensic pathologists have additional responsibilities in terms of attending crime scenes, examining blood stains, exhumation of bodies, estimating weapon use. General pathologists are normally limited to finding the causes of fatal diseases i.e carrying out the autopsy and examining the histology. In most cases, at least in San Francisco, investigation of the scene is performed by one of the examiners hired by the Chief strictly for the purpose of examining the scene and obtaining an accurate history.

21. In terms of my role as an assistant medical examiner, I have been asked if this involved undertaking the role of a forensic pathologist. If autopsy is required, it may be performed by either a forensic or clinical pathologist working for the chief. My role was not as a forensic pathologist as I had no interest in anything but the heart.

Expertise in Restraint

22. I have been asked whether I have expertise in restraint in terms of qualification or medical experience. Regarding the role of restraint in some cases of sudden death, I have actually seen two living cases of excited delirium (which involves restraint), examined multiple hearts from excited delirium cases and, in the last decade, published more than 10 peer reviewed papers on the subject. I also have had extensive experience in pulmonary function testing. The last paper I wrote about ED (“Prolonging the prone postulate) was published in 2020. I have also written book chapters on the effects of pulmonary restriction in nearly 1000 fire-fighters who were undergoing their annual fitness for duty testing. I feel comfortable discussing the restraint aspects of this case. I also have experience in the examination of the hearts of drug users who die of excited delirium, the possibility of which sounds very likely this case. I am not a forensic pathologist. but I did receive some training in general pathology as part of my neuropathology training at the Royal London. I do not have expertise in levels of force or application of force.

Histological Examination

23. I have been referred to the first report[1] I produced, as instructed by the Police Investigations & Review Commissioner (PIRC).

24. I was asked to consider four questions by the PIRC, which are outlined in the first paragraph of the first page of my report. I confirmed that to answer those questions, I required to perform my own histological examination of the heart.

25. At the second paragraph on page 1 of my report, I state: *"My review disclosed histological abnormalities that were apparently overlooked at the original autopsy."* In the following paragraph on page 2, I continue: *"The histological changes discussed are not illustrated, but can be if it is so desired (the changes were particularly obvious in slides designated AG, AH, AA and Z in the original autopsy report)."*

26. I remember that I was in London to lecture when I was contacted about the case. San Francisco was virtually the cocaine capital of the world at the time and I was writing about it more than anyone else. I don't recall whether I received a call from the Police Investigations Review Commissioner or whether I called them. They were aware that I was in London and DSI William Little arranged to bring the slides to London and made arrangements with Guys and St Thomas' Hospital where I could examine the slides. I did so on 25 August 2015.

27. Someone, and I have no idea who, examined the heart appropriately in the same way I had, in conformance with academic standard academic protocols. In the United States, examination of the heart is much more superficial than in the UK.

[1] PIRC-02526(a)

In this case there were so many slides that I asked DSI Little to write down what I was seeing. After we were done, DSI Little took the slides away. I was not aware until I was contact by this Inquiry that the slides were created by the original pathologists and subsequently examined by Professor Sheppard and several other pathologists.

28. I've been asked if I would be able to illustrate the histological changes I saw in the slides and referred to in my report. I probably could find some illustrative examples from another case. I can see in my head the changes that I mentioned. I think several of them stood out to me as being pronounced. But finding either node would be very difficult given the number of times the heart had been examined and sampled. To a certain extent all adults develop what we call myocardial fibrosis, sometimes other changes also appear with aging. The use of stimulant drugs is a known accepted cause for myocardial fibrosis which is important to this discussion for the simple reason that that interstitial fibrosis can lead to arrythmia and sudden death.

29. I received a diagram of a section of heart showing how they had numbered the slides. Those were the numbers I used in reference in my report. I have been shown a blocking diagram of the heart which I am advised was prepared by Dr Kerryanne Shearer[2]. Initially, I did not think that this was the diagram I received. However, having had an opportunity to compare this diagram with the version I was given, I can now see that they are the same document.

Final Post Mortem Report

30. I have been showed the final post-mortem report[3], dated 18 June 2015. This was the double-doctor post-mortem by Dr Kerryanne Shearer and Dr Ralph BouHaidar carried out on 4 May 2015. I can see that at page 9, the report records: *"the pericardial sac was normal. The heart (430g) was of normal size and configuration. The coronary arteries, myocardium and cardiac valves were normal. The aorta was normal."* Also, I am directed to page 13, under the

[2] WIT-00002
[3] PIRC-01445

heading of histology, the report records *"Heart - The heart was mapped and the sinoatrial (SA) node and atrioventricular (AV) node sampled. SA node - No obvious significant abnormality. AV node - No obvious significant abnormality. Ventricular mapping - There is no evidence of significant abnormality in the sections examined."*

31. Initially, I did not think that this was the Final Post mortem report that I received. However, having had an opportunity to compare the document with the version I was given, I can now see that they are the same document. The blocking diagram I have been shown is not what I would consider mapping. There is a difference in the terminology that is used in the UK versus the United States. In America, 'mapped' means tracing electrical currents in a living heart. In the Final Post mortem report the pathologists did not mention preparing anything like comprehensive slides preparation.

32. I did not examine the AV node specifically because it is too diffuse. It is a small structure located in the triangle of Koch triangle near the coronary sinus on the interatrial septum. Specifically, it is located at the junction of the crista terminalis in the upper wall of the right atrium and the opening of the superior vena cava. In a right-dominant heart, the atrioventricular node is supplied by the right coronary artery.

Changes Observed The Heart

33. I have been referred to page 2 onwards of my report where I discuss the drug, Alpha-PVP. I'm specifically referred to the sentence where I describe the changes it is known to produce in the heart:
"uneven staining pattern of the myocardium, fragmentation and waviness of fibres, perivascular connective tissue growth, intramuscular fibrosis and scarring, disintegration of cardiomyocytes, nuclear disintegration, loss of cross-striations and thickening of blood vessel."

34. I am asked to explain what I can see when I refer to *'uneven staining pattern of the myocardium'*. If you were looking at a piece of wallpaper with a pattern on it, you would notice all the colours were bright and shiny at the edges, but at the centre the colours were faded out. Simply that some parts are stained more darkly than others.

35. I am asked to explain what I mean when I say *'fragmentation and waviness of fibres'*. Usually the muscle fibres in the heart are oriented. They may look disoriented to a lay person, but in fact they are oriented. There were areas where the fibres seemed to be in a state of disarray, which would not be seen in a normal heart. In addition, the fibres are normally long and continuous, not broken into small fragments.

36. I have been asked to explain what I mean when I say 'perivascular connective tissue growth'. Fifteen percent of the cells in the heart are called fibroblasts which produce connective tissue. When there is damage to the myocardium, fibroblasts morph into cells called myofibroblasts. The later secrete different molecules including Type I and Type 3 collagen. The later can be deposited inside or outside ("perivascular") some vessels causing the obstruction if located inside the blood vessel.

37. *'Intramuscular fibrosis and scarring'*: Collagen forms and maintains the structure of the heart. It can be found inside and outside of blood vessels. Its molecular structure is the same inside or outside of the muscle cell, although type 3 is the main component of cartilage. No type of collagen conducts electrical signals – this may lead to aberrant signal conduction, a known cause of sudden unexpected death.

38. I am asked to explain what I mean when I say *'disintegration of the cardiomyocytes'*. There were some cells that were obviously in the process of dying. Cardiomyocytes are the muscle cells. Cellular death in generally is referred to as necrosis. When destruction of the cell is a consequence of processes within the cell, the result is cause autophagy, and it follow a specific

time sequence. When necrosis is the cause of death the destruction occurs at the same rate and involves all the same parts of the cell at the same time.

39. ‘Nuclear disintegration’ is where the nuclei are recognisably deformed. This is usually a bad sign. It’s often a sign of malignancy, but I don’t think that’s an issue here. It’s just a sign of dying. The cause for nuclear disintegration could be anything that kills a cell but, ultimately is a result of break-down of laminin and other proteins that give the nucleus it’s normal oval configuration during life.

40. I have been asked to explain what I saw when I referred to ‘loss of cross-striations and thickening of blood vessels’ and how that differed from a healthy heart. As far as loss of cross-striations, there were some areas where I couldn’t see cross striations. Normally you can see the individual muscle bundles, but not in those areas. Thickening of the blood vessel wall means just that. It is also a finding in most deaths of chronic stimulant abusers.

41. I am referred to paragraph 1B at the top of page 3 of my report where I refer to *“observed cardiac lesions”*. The lesion I am describing is the fibrosis, wavy fibres, muscle nuclear changes discussed above. There are some areas where there are vacuoles. A vacuole is a word for nothing, meaning it’s a clear space. The overwhelming impression that I got when looking at the heart, and it was clear to me then, that it looked like a methamphetamine user’s heart.

42. I have been referred to paragraph 3 on page 4 of my report where I mention observing *“myocardial remodelling of both ventricles”*. This can be a normal occurrence of the heart and is a reparative process, where cells change size and position. I only recall changes in the Left Ventricle. As I have not had an opportunity to re-examine the slides I can’t opine about the right ventricle. Myocardial remodelling is the way the heart heals after a myocardial infarction. It is also the way that the heart responds to a chronic pressure overload, namely hypertension. Many of the antihypertension drugs recently on the market work by inducing “reverse remodelling” and restoring the heart back to its normal shape.

43. Remodelling causes cells around an injured area to increase in size in an attempt to fill in the area where cell damage occurred. Unfortunately, stimulant drugs somehow cause this process to occur in places it doesn't need to occur. Generally, the result is an enlarged heart. I realise that my report refers to remodelling of both ventricles, but I don't remember, and never had the opportunity to photograph the heart for the record.

44. I've been asked if a person has myocardial remodelling, would I expect the heart weight to be abnormal. It is not possible to say in this case. Given Mr Bayoh's height and the heart weight recorded in the final post-mortem report, his heart falls into the very broad range of normal. This is a little surprising as nandrolone, which the decedent was also taking, also causes remodelling. I have written a paper about a weightlifter who was on nandrolone, and he had remodelling. Heart weight is an independent risk factor for sudden cardiac death, regardless of the underlying aetiology. It probably accounts for a fair number of sudden deaths. I'm more used to seeing an enlarged heart in such cases than not.

45. I'm asked if it is possible the changes observed were a result of heart arrythmia being caused by the drugs and then the ongoing CPR that happened prior to death. It is possible because of the presence of interstitial fibrosis, of the type present here, disrupts normal cardiac conduction, and can cause sudden death from cardiac arrhythmia. I am a little hesitant, absent having the slides to review to reach a more definitive conclusion. I believe that there was some mention of microinfarction in one of the reports which probably would not have been a consequence of CPR as too little time had passed between collapse and death for more advanced changes to have been apparent. Based on the amount of time between the beginning of CPR, and Mr Bayoh's death, you might also see the wavy fibres. With special staining you might be able to elicit microscopic evidence of damage to some of the cells, but it wasn't performed in this case.

Cause of Death: Drugs

46. On page 1 of my report, I state: *"In addition, the three drugs found in Mr Bayoh's body were, at a minimum, contributory causes of death and quite possibly the actual cause of death. Very little data about the effects of Alpha-PVP exists, but the little that has been reported suggests it may well have been the sole cause of death."*

47. Since the death of Mr. Bayoh in 2015, hundreds of reported cases of alpha-PVP deaths have been described in the medical literature. Alpha-PVP is a cathinone derivative (active ingredient in marijuana) related to methamphetamine and amphetamine. Presumably all the drugs share a common mechanism of toxicity, namely elevated levels of the neurotransmitters, dopamine, norepinephrine and even cocaine. Elevated levels of these compounds can leave identifiable histologic markers. Unfortunately, no systematic study of human hearts (let alone other parts of the body in the body) exposed to alpha-PVP have never been published nor are they likely to be as we now know the drug is far too dangerous to give to humans.

48. In answer to question 1 on page 2 of my report, I state:
"All of the drugs detected (Alpha-PVP, MDA and MDMA) cause acute and chronic cardiotoxicity. Any, or all of them, might have been the cause of death, but it is impossible to determine which drug actually did."

49. I am asked whether regardless of the issue of damage to the heart, could all of the drugs detected still cause acute and chronic cardiotoxicity and which, if any, of them might be the cause of death. Regardless of whether tissue damage was observed, all three drugs mentioned could have caused sudden death. The explanation is simple: these drugs work by altering the electrical system, at the molecular level within the heart, Interaction with the potassium channel appears to be a characteristic of most cathinone derived drugs, however the ability to disrupt membrane channels varies from drug to drug, simply because at the molecular level the structure of each drug is slightly molecular different. Some drugs would fit the molecular channel better than others

50. The three drugs mentioned also share the ability to increase blood catecholamine levels. Elevated catecholamines cause visible tissue damage. Unless pre-existing, there is no time between symptom onset and death for necrosis, in the form of contraction band necrosis to occur. That would explain why contraction bands were not seen.

51. Among clinical reports about the three drugs, MDMA deaths are relatively uncommon, MDA deaths are much more common, and alpha-PVP deaths are now the most common of all. It is questionable whether MDA is cardiotoxic at all. If I were to write my report again, I would say that we really need to rule out alpha-PVP as a cause of death, just based on the clinical presentation, though the mechanism is not known with surety. Results in animals cannot be generalized to humans, and no human studies of this drug would be permitted as it is too dangerous. Widespread though abuse of the drug may be, we don't know anything about the human pharmacokinetics or tissue disposition of alpha-PVP.

Genetic Testing

52. I believe that in cases of sudden death, when any autopsy is said to be negative, the decedent should undergo genetic screening, if for no other reason than for the family's peace mind. Genetic mutations could affect the heart or the brain and my occur in other members of the family. Of course, at the time of Mr. Bayoh's death this sort of testing was not done except in research cases and would have been prohibitively expensive. Today it is not, and many insurance companies are willing to pay for the testing.

53. Many experts in the field of genetic forensics believe that as many as 30 per cent of all sudden death cases, where the autopsy is negative, will be found to have a genetic mutation present. In relation to the heart, among others, this would include genes for channelopathies and long or short QT syndromes. Interestingly, the same genes are sometimes found in epileptics who die suddenly as in non-epileptics who experience sudden death.

54. I am asked what would be the type of genetic screening I would recommend? Depending on the testing approach, the process may be expensive. Genetic testing is not standard and when testing is done it is the common practice to ask for a "panel," or a group of genes frequently encountered in specific diseases. In the United States the cost is often covered by insurance companies. One of the most studied genetic diseases, is called long QT syndrome, and the diagnosis can be made simply by recognizing the pattern of the EKG even when the patient is alive and asymptomatic. All that is required is a routine electrocardiogram. I didn't see anything in any of the material that I was asked to review suggesting that there were records of Mr Bayoh going to his GP and having had a cardiogram performed. In-vitro studies of human heart tissue show that alpha-PVP can increase the duration of the QT interval which, in turn, increases the risk for sudden death. Taking alpha-PVP, or a drug like it may cause death because the drug has made the QT interval longer, something that cannot be detected after death. Other cardiac gene mutations have been identified associated with a gene called TITN, a very large protein the comprises most of the cardiac skeleton and cardiac muscle. Mutations of TITN are thought to be present in more than a quarter of cases where nothing is found at autopsy. If testing is not done for the gene there is a very good chance that the actual cause of death may be missed or misdiagnosed.

MDA

55. I have been referred to the toxicology report dated 12 June 2015. This is appended to the final post-mortem report[4]. I am referred to the 'Interpretation and Opinion' section of the toxicology report on page 2, where it states:
"MDA is formed from MDMA by metabolism but is also sometimes encountered as an illicit drug either on its own or as a constituent of Ecstasy tablets. If its presence in blood is due to metabolism of MDMA, it is usually present at a lower concentration than MDMA, as in the present case."

[4] PIRC-01445

56. I am asked about the possibility that the MDA was a metabolite of MDMA. MDMA is metabolised to MDA. But the absolute quantitative post-mortem value cannot reliably be used to determine the cause of death or even what the MDMA concentration was during life. It simply shows that the drug is present. So many changes occur in drug concentrations after death that it's naïve to argue that a specific post-mortem blood level measured in blood taken from an unknown anatomic site in the body, where the post-mortem interval (between death and sampling) is not known especially when neither the actual time of death nor storage condition of the cadaver are known with any precision. In addition, MDA and MDMA have different affinities for fat where both drugs stored fat begins to dissolve after death and releases stored drug stored within it. So yes, MDA was there. What the numbers mean or whether the MDA is a by-product of MDMA in this case is irrelevant.

57. I am referred to the blood samples, and the fact that there were two different types of samples - the hospital blood and the blood taken at the post-mortem examination. The hospital blood has been taken prior to death. I have been asked specifically about the hospital blood and the possibility there of the MDA being a metabolite of MDMA. Since MDA and MDMA were present in both pre- and post mortem blood, it is difficult to imagine that some conversion to MDA did not occur during life. And in fact, controlled studies show that some conversion occurs during life occurs. I think it is irrelevant since MDA appears far less toxic than MDMA. I would reiterate the absence of correlations between levels measured in living and dead. Post mortem blood levels are almost always show an increase from life. For example, it is not unheard of for patients with zero fentanyl at the time of death to have massively high fentanyl levels at autopsy 48 hours later. This is because fentanyl is absorbed by fat and slowly released, even in life. Nobody is sure of the rate it is released in death, another illustration of the consequences of fat drug storage. The situation is no different with MDMA.

58. I am referred to paragraph 2(a) of my report headed 'Methylenedioxyphene (MDA)' and asked why I have put an emphasis on this drug rather than MDMA. I didn't intend for it to read like that. It is just intended to raise the possibility since it

has the same mode of action, namely serotonin elevation, that it might contribute to the now known massive effects of Alpha-PVP. MDMA deaths are rare, but they're more common than MDA deaths.

Nandrolone

59. I have been referred to paragraph 3(a) of my report headed 'Nandrolone'. I state: *"High doses of nandrolone elicit cardiotoxic effects including cardiac remodelling and injury."*

60. I have been asked to explain what I consider to be a high dose of nandrolone. I am unable to answer that question. To the best of my knowledge, post-mortem nandrolone levels have never been reported. It is reasonable to suppose, however, that a regular user is presumably ingesting more than an occasional user. The literature on that subject has grown substantially since my report was written. Reported deaths in body builders, usually males using this drug regularly, have increased.

61. I am asked to elaborate on how often a person would have to take nandrolone to be considered a regular user. I really don't know, and I do not believe there is an agreed definition.

62. Within the same paragraph, I state:
"There is also evidence that, by methods yet to be determined, nandrolone facilitates the occurrence of myocardial arrythmias, the apparent cause of Mr Bayoh's demise."

63. I am asked at what levels does nandrolone cause arrythmias. I can't attest to a specific level since it's a post-mortem level. Nobody is ever going to give permission to give increasing amounts of nandrolone to a human volunteer and determine at what level arrhythmias occur and determine how much drug would have to be administered to cause an arrythmia. What information is available is mostly from animal studies. In the case of nandrolone a blood level is hardly

needed to determine the cause of death. Chronic nandrolone causes reverse myocardial remodelling and myocardial fibrosis, no cause of death from acute single use has ever been reported

Rib Fracture

64. In this report I also comment on the rib fracture. At page 5 of my report, I state *"The decedent was involved in multiple altercations, including a prolonged encounter with the police. None of the witnesses reported seeing anyone kick Mr Bayoh, although there seems to be general agreement that there was a good deal of thrashing and kicking on the part of the decedent. The autopsy report makes no mention of any truncal bruising and, in fact, the fracture was only identified by x-ray, never having been noticed at the time of autopsy. On the other hand, rib fracture is a known complication of CPR. In fact, fractures of the ribs and sternum are the most common internal injuries related to resuscitation. Such must have been the case in this episode."*

65. I'd like to revise this opinion somewhat. On two points: (1) The rib fracture wasn't identified by the x-ray. It was only identified by CAT scan. (2) Rib fractures are common in CPR. They tend to occur on the left side and much more often in men. That all makes sense given what CPR is and how it's done. Traditionally when you saw a fractured first rib, that meant massive trauma. We now know that is no longer true. CAT scanners have become more common now and first rib fractures are much more easily visualized, at least so says the peer-reviewed literature including the following article: *First rib fracture: A harbinger of severe trauma?*[5]. On reflection, I now can't say with certainty what the cause of the fractured rib may have been. While it is possible that a first rib fracture could be caused by CPR, I wouldn't be comfortable commenting on causation due to the unknown occurrence rate.

[5] WIT-00013

Professor Sheppard's Report

66. I have been referred to Professor Mary Sheppard's report[6], dated 1 December 2015. I have read the first few lines of the section headed 'Microscopic Examination' on page 4 of the report. I have specifically done published peer reviewed research on the problem of contraction band necrosis (CBN) and published one of the most cited papers on the subject. Contraction bands can be present, but they an utterly non-specific finding. Their presence merely signifies that there is too much free calcium within the cell. The lesion itself does not explain why, except that it is often associated with catecholamine excess. No other disease can be diagnosed by the presence of CBN. Even though very little is known about the mechanisms of alpha-PVP toxicity, we do know that this drug causes elevated levels of norepinephrine, a neurotransmitter known to cause contraction bands. The leukocytes she observed in the capillaries along with edema surrounding blood vessels are all changes that could be CPR related.

67. I would disagree with Professor Sheppard. Three types of fibrosis are recognized: interstitial, infiltrative and replacement. Clearly, the first two types were not present, but I believe I saw small areas of interstitial fibrosis and even indicated the slide numbers where I saw them in my report. In my experience and that of others, this kind of fibrosis is enough to cause lethal arrhythmias. I agree that there were no abnormal infiltrates and that the muscle bundles were normal. Unfortunately, I was never given the opportunity to photograph the abnormalities I identified.

68. In answer to question (b) on page 5 of the report, Professor Sheppard states: *"While CS/PAVA may have an effect on the heart, there is no evidence pathologically of any damage to the heart."*

69. It is absurd to even suggest that the pepper spray had any effect on the heart as there was no effect seen in the trachea. If the irritants had made their way into

[6] COPFS-00027

the lung, it would have burned its way along the trachea on the way down to the lung.

70. In answer to question (d), Professor Sheppard states: *"The deceased has no cardiac abnormality identified at his death. However this does not rule out sudden cardiac death due to an electrical abnormality the cardiac channelopathies."*

71. I would add to Professor Sheppard's point about channelopathy, that many sorts of mutation could occur at a molecular level. Although genetic mutation are now recognized as a possible cause of unexpected sudden death, they were not in 2015. I would simply disagree with Professor Sheppard that in the slides I mentioned, I saw evidence of interstitial fibrosis.

Dr Soilleux' Report

72. I have been shown Dr Elizabeth Soilleux's report[7], dated 14 February 2016.

73. I have been referred to paragraph 60 of the report, where Dr Soilleux states: *"Neither the report of the macroscopic examination of the heart at autopsy nor my own microscopic examination of the histological material retained at autopsy (detailed in appendix 2) show any evidence of a pre-existing heart condition, indicating that pre-existing cardiac pathology is unlikely to have contributed to death."*

74. I have been shown Appendix 2 of Dr Soilleux's report, as referred to in paragraph 60. Some of the conditions Dr Soilleux lists are not channel-related, they are different aetiology. However, Dr Soilleux is absolutely correct. At the time of Mr Bayoh's death, testing would have cost an enormous amount of money, but now for a fraction of the original testing fees for inheritable disorders are available. The best estimates I have seen, begin about the time of Mr. Bayoh's death and they indicate 20% to 30% of people in his age group who experience sudden

[7] COPFS-00031

death with an otherwise normal autopsy, have a genetic abnormality. I would disagree with her findings in so far as she says there was no pre-existing cardiac pathology - I saw scars. I should add that, if the body was not cremated, genetic testing still may be possible

Dr Cary's Report – examination of the heart histology

75. I have been shown Dr Nat Cary's report[8], dated 23 October 2015.

76. On page 4 of the report in his description of the heart, Dr Cary states: *"My own examination reveals the following: Heart: Sections of the left ventricle show some variation of the myocyte size with scattered hypertrophy of the myocytes. In one section there is a focus of marginating neutrophil polymorphs. Overall, in sections of the left ventricle there is no evidence of scarring or myocyte disarray. Sections of the right ventricle all appear to be normal."* At page 5, he concludes at point 2. *"I agree there is no evidence of any underlying natural disease process that caused or contributed to death."* I agree there is no apparent evidence of underlying natural disease, however, I did see interstitial fibrosis and myocardial remodelling of left ventricle. The marginating that Dr Cary noticed is in fact a sign of dying. The change in myocytes is not normal either, but more likely a consequence of hypertension or frequent stimulant abuse. Marginating of neutrophils is a sign that the individual is dying or dead. I have been asked whether these changes may be a consequence of CPR. The change is not so much a consequence of CPR but rather the failure of CPR to restart the heart. It is an agonal process.

77. Dr. Cary does not appear to consider the possibility of genomic error as the cause of death. In decedents with negative autopsies, the best data suggest that in 20 percent or more of cases a genomic cause can be identified.

Professor Crane's Report – examination of the heart histology

[8] COPFS-00196

78. I have been shown the report of Professor Jack Crane[9], received 31 May 2016. At page 6, Professor Crane addresses the question *"whether the deceased had a heart abnormality, if so what impact would this have had on his death".* In response he states *"There is no evidence of underlying heart disease. I have examined the microscopic sections taken of heart tissue, including the sections of the conducting system. There is no evidence of inflammation, recent necrosis or fibrosis. I am satisfied that there was no cardiac abnormality which would have caused or contributed to death."* Again, I disagree that there was no evidence of underlying heart disease. I saw small areas of interstitial fibrosis. Fibrosis is metabolically active scar tissue in the heart. Fibrosis is metabolically active scar tissue in the heart. Professor Crane makes no mention of molecular/genomic disease, but that may not be relevant as the significance of these disorders was not have been recognized by most in 2015. Most importantly, Professor Crane appears to be unacquainted with channelopathies and other genetic mutations associated with sudden death

79. Overall, I understand that I have come to a different conclusion on the heart histology, than Professor Sheppard, Dr Cary, Dr Soilleux and Professor Crane. I accept that Mary Sheppard will have seen many more hearts than I. But I don't think she was really looking for these changes. I certainly had no trouble seeing them.

Excited Delirium

80. I am aware that the diagnosis of Excited Delirium is controversial in the UK. I debated Dr Nat Cary on this topic more than 20 years ago at the Royal College of Pathologists.

81. I was particularly incensed the following article in the Lancet *End the use of "excited delirium" as a cause of death in police custody*[10]. I did a PubMed search

[9] COPFS-00134
[10] WIT-00040

on each author of the Lancet article. None had previously ever had written a paper on Excited Delirium or any issue related to it. This article relied upon no controlled studies from the scientific literature, and there is no evidence that the authors had ever seen an actual case. In the United States the National Institute of Mental Health no longer uses the DSM-5 diagnosis and disapproves the DSM process and its designation of Excited Delirium as purely a psychiatric disease.

82. Neurochemical studies showing derangements in brain dopamine metabolism, particularly in the region of the brain's nucleus accumbens have been observed and published by many authors (though many of the studies were animal.) The same derangements have been demonstrated in the brains of all stimulant deaths in humans (when the effort to measure them was undertaken). Many published studies have quantitated the physiologic changes that occur during restraint in man and animal. None have found changes that could remotely be considered lethal. I am aware that some organisations refer to Excited Delirium as 'Acute Behavioural Disturbance' (ABD), which is appropriate, especially in this case where the work-up to prove ED was not complete.

83. I have seen many litigated cases of real and alleged ED. In my experience, which appears to be the experience of those who specifically study the disease, the overwhelming number of those effected with ED are chronic drug users, mainly chronic stimulant abusers. Although the syndrome is occasional seen in schizophrenia, I have yet to see a case where the decedent wasn't a chronic stimulant drug user. All the decedents had changes secondary to stimulant abuse in their hearts and elsewhere. The best explanation for ED appears to be the nexus between simultaneous disruption of brain and heart homeostasis at the same time. The much-discussed Lancet paper is not believable. It was written by individuals with no published research on the subject and no experience with the clinical management or pathological examination of decedents with this disorder. Contrary to the way it is portrayed by the media, lay, medical, and enforcement sources, ED, though a plague on society, is a rare disease.

84. The only paper to accurately estimate the true incidence of ED was published over 20 years ago in Los Angeles (Stratton). It included 400 individuals with apparent ED requiring paramedic evaluation. Most left the scene before paramedics arrived. Of the 400 ED patients there were only 19 deaths. The implications are clear – ED is a very uncommon disease. For most pathologists, police and first responders, managing an ED patient is a once in a lifetime event.

85. A widely quoted ED paper, *Excited delirium: a psychiatric review* by Maurice Lipsedge[11]. calls for ending use of the term excited delirium (ED) and, like many similar papers argues it is a fictitious entity, used to obscure underlying police misconduct. I have studied ED for 25 years. I can remember my very first case. It was a prostitute, and she was caught by the guards, running down a hotel hallway screaming and naked. She was quickly brought to the trauma centre where she was found to have a temperature of 105 degrees Fahrenheit. She was given a powerful anti-psychotic drug and bathed in ice. It saved her life even though she had cocaine levels too high to measure without dilution. After hospital discharge she reverted to her old pattern of drug use and several months later came back to the same hospital with the same symptoms. She couldn't be resuscitated. I think lack of knowledge and limited exposure to the disorder, not to mention political correctness, are the issue here.

86. In the second last paragraph on page 3 of my report, I state:
"The results of thorough in vitro studies of human brains harvested from those who succumb suggest that most, probably all, of the symptoms listed are as a consequence of dopamine excess." I would now add to this that excessive concentration of norepinephrine are also partly responsible, especially for the cardiac damage (norepinephrine is the heart only neurotransmitter).

87. Here I am talking about studies by Deborah Mash. She held a federal grant to operate a brain bank. In cases diagnosed as ED in life, post-mortem brains were taken at autopsy, frozen and placed on dry ice. It was an easy matter to send

[11] PIRC-03395 (a)

frozen brain slices to Dr. Mash's lab in Florida where they were studied for a number of neurological diseases over a period of years.

88. I am asked if I'm aware of any researchers replicating that research where those changes seen by Deborah Mash have been seen in the brain. I'm not sure. I lost interest in the area, I found it too politically difficult to deal with. It is my understanding that many others have measured dopamine concentration in human and animals and have been doing so for over a quarter century (using biochemical and immunohistochemical methods).

89. I have been asked about the Royal College of Psychiatrists position statement on "acute behavioural disturbance" and "excited delirium". It has been explained to me that they have said that the term excited delirium should not be used. Specifically that they highlight that the term *"has also been controversial when used in a way that minimises the role of restraint in understanding why someone has died following the use of force by police or health services, particularly those from ethnic minority backgrounds."*[12] I have been asked to comment on this. My first comment would be that I do not follow the writings of the Royal College of Psychiatrists. but I do follow the position of the American Institutes of Mental Health which, as of 2022, recognizes the disorder as quite real. My second comment is that it seems strange to me that Royal College of Psychiatrists would even concern themselves with ED since it is a medical emergency with which psychiatrists would have little experience. Finally, at the risk of repetition, one simply cannot dismiss the official leadership of the American mental health care establishment; it will no longer support research based on unreliable DSM5 criteria.

90. Excited delirium is rare, and this has been proven in one large study, by a researcher named Stratton, and published in the American Journal of Emergency Medicine. The author was the director of the Paramedic System for the city of Los Angeles. Every individual diagnosed in the field, in the City and County of

[12] WIT-00021, at page 3.

Los Angeles with ED, was included. Approximately 400 cases met the diagnostic criteria for ED but only 19 deaths were recorded. So I don't think ED is common at all. I think it's a one in a lifetime experience for most policemen and most coroners. I know nothing about the racial characteristics of the people who succumb, although the two people that I saw were both white.

91. The conclusion is supported by the American College of Emergency Physicians formal recognition of ED. The endorsement is very important because it was reached by the doctors who actual render emergency care to ED victims. It is also devoid of political judgments.

92. I'm advised that the American Medical Association (AMA) adopted a policy in 2021 confirming that current evidence does not support "excited delirium" as an official diagnosis and opposes its use until a clear set of diagnostic criteria has been established. I was not aware of this.

The physiological effect of restraint of the deceased

93. The third question I was asked by the PIRC was, "What is the physiological effect of restraint of the deceased in the circumstances of his arrest?" In my report, I respond to this at page 6 with the answer "*Given the details of this situation the effect of physical restraint would have been di minimus."* I then explain *"It has been postulated by some that the mechanical obstruction of chest movement, called asphyxia, because air exchange within the lungs is diminished, could be caused by multiple officers overlying the body, thereby leading to asphyxial death. There is no evidence that sufficient pressure was applied to the decedent's lungs."*

94. I have been asked what I mean by "sufficient pressure"? At page 7 of my report I make reference to experiments in which weights of up to 250 pounds were applied to maximally restrained volunteers with "no clinically significant effects were observed". I am advised that the Inquiry has heard evidence that a police officer who weighed 25 stone (or 350 pounds) may have lain across the upper

back of Sheku Bayoh. It has been explained to me that this matter is disputed; I have been asked to comment on whether this might be sufficient pressure, if the Chair makes a finding that this has taken place. In my view, it would certainly cause measurable changes in oxygen delivery to the myocardium, but I believe these changes would likely be trivial. As a rule, practically, you cannot apply that much weight simply by lying across the back. Miklowitz published a study of restraint where 250 pounds (roughly 18 stone) was placed on the back. Simultaneously, they measured her VO2 max, the maximal amount of oxygen supplied by one breath. They measured this continuously on electronic equipment and they found a decrease, but it wasn't enough of a decrease to cause clinical symptoms.

95. In the United States, the general approach for the plaintiff is to argue that increased weight on the thorax inhibits adequate chest movement resulting in asphyxia and that experiments done on healthy volunteers are inapplicable to the situation. The assertion is hardly proven. One frequent argument is "Well, the people that are tested in the laboratory are not obese," In fact studies of obese restrained volunteers have shown the diminution of oxygenation is real and measurable but not life threatening. Note that the medieval practice of peine forte, where stones were placed on a naked man tied to the floor. Very good records were kept of these "legal" proceedings. In some of the cases up to 350 pounds of stones were placed on the victims and the process would go on for days before the decedent or the prisoner would die. Granted the weight was on the anterior chest and not on the back, but I've yet to see any proof in any controlled study that the position of the stones on the thorax makes any difference makes a difference.

96. The ancient or historical materials certainly argue against asphyxia from moderate chest compression because, we already know that weights over 350 pounds do not prove fatal. Furthermore, it's gratuitous to assume that lying across somebody would transfer their entire weight from one body to the other. Finally, some years ago I published pointing out that the oxygen reserve capacity

in the body was so great that you just could not, in a normal healthy person, deprive them of enough oxygen by applying the weight changes described above

97. Dr Sheppard's report mentions that she in fact is writing about positional asphyxia. I read the paper: she said the man died of drugs – and I don't remember which drug - and police restraint. The paper was written by two of her graduate students named Krexi. In her report for this case Dr Sheppard relies on the paper authored by O'Halloran[13] in relation to the link between restraint and sudden death. The problem with the O'Halloran paper is that it contains no research, consisting merely of a collection of anecdotes, there are no specifics about past drug use or past medical history, nor any detail about the autopsy. The cases are presented because all the deaths occurred while struggling with the police.

98. This theory of positional asphyxia was first proposed in 1992 by a friend of mine named Donald Reay. The experiment was done with primitive equipment. It was a very simplistic design, and all that Dr. Reay was able to demonstrate was that people whose hands were restrained prior to exercise, and then were hogtied, took longer to recover a normal baseline electrolyte level and oxygen level than people who weren't. The theory was retracted in open court in open court, by Dr. Reay who said he was wrong and that the studies from the San Diego group, including Miklowitz which were, were done with much more accurate equipment than his, and that his had been mistaken, although he never did deny that such a theory might be true. In any event, this idea that putting the weight on was introduced by Reay and retracted in front of a judge, and it has been in a published verdict in the US. So, this is what Dr Sheppard relied upon when she added the "restraint" component to her diagnosis.

99. The current theory of the plaintiffs' bar is that it's simple deprivation of oxygen to the heart caused by the pressure on the body. This is an interesting theory, but

[13] O'Halloran RL and Frank JG. *Asphyxial death during prone restraint revisited – a report of 21 cases*. Am J Forensic Med Pathol 2000; 21: 39–52.

there's evidence against it, and I know of no experimental evidence for it. A statement with no experimental evidence behind it is just unqualified hearsay evidence. It's unsupported and it's theoretical, and it sounds like hearsay, at least what we would call hearsay, so I was disappointed.

100. A page 7 of my report I refer to a large body of literature that suggest that maximal prone restraint positioning *"has very little clinical effect."* I am asked to explain what I mean by "very little clinical effect?". In the experiments I referred to, a volunteer would be hog-tied, and then every exhaled breath collected on an inhalation, an exhalation –and the amount of oxygen and carbon dioxide measured in each breath. In other studies volunteers were also hogtied following violent struggling (from memory I think this of continuous beating a boxing bag prior to being hogtied). When that, protocol was followed, volunteers they did show measurable decreases in oxygen, but only on the order of 10 per cent which is interesting, expected and not dangerous. To put it in perspective, to qualify for a lung transplant, at least in US transplant centers, one needs to lose 80 per cent of their lung capacity before being considered a candidate for lung replacement. Yet studies on so called "positional asphyxia demonstrate loses of less than 10 per cent.

101. It has been suggested to me that there are experts in restraint who would consider that the results of these studies are clinically significant. That such experts would counter that laboratory studies are not able to mimic a real life restraint and struggle against restraint as they experience must be carried out in a safe and ethical manner. The volunteers have no concerns for their safety and consequently do not have the same physiological state as an individual being restrained in a real life situation. I would say this is the common response because when you challenge this theory, they always say, "Well it's just not relevant to humans." In fact, catecholamines, which are the hormones that mediate the stress response within the body, have been measured in people that are exercising violently. The exact amount the heart is working can be determined, and so can the amount of oxygen the heart is consuming. so you're not hypothesising. Volunteers pounding the wall with boxing gloves for 15

minutes, to the point of exhaustion, who were then restrained and hogtied have had the cortisol, epinephrine, norepinephrine and dopamine levels measured. The levels measured proved to be the same as those students who were only restrained without exercise. Dr Ho has done those studies and the results published in respectable peer reviewed journals.

102. Published controlled studies in the peer-review literature show that obesity has no effect on position when being restrained. There is reason to believe that pressure exerted across the thorax would diminish a person's ability to breath but not seriously unless the person was seriously ill with respiratory issues, hypertension or a chronic drug user.

103. The commonly raised objection that I've seen in case-after-case is "Oh yes, but this doesn't apply to fat or old people. It's not similar to the situation seen on the street." These experiments, I think, are life-like as can be. I think it's on this other side to show that the studies that have been performed and published in peer-reviewed journals are not comparable to the situation in the street. The only way I think they're not comparable is they're not comparable because if you're a drug user, you'd be automatically excluded from the study.

Dr Cary's Report – restraint and cause of death

104. At page 6 of Dr Cary's report he states with regard to the cause of death: *"In relation to the possibility raised of an excited delirium syndrome, this is not a diagnosis that I consider to be appropriate as a cause of death, accepting that it is used as such in North America. In my opinion, it's very much more appropriate to stick to the facts rather than invoke syndromes. The facts here are that there was an acute behavioural disturbance and stimulant drug misuse, both features that are highly relevant to the cause and circumstances of death. One of the most dangerous effects of stimulant drug misuse was not present here, namely hyperthermia. In terms of possible role for restraint, I support the opinions expressed that petechial haemorrhages in the eyes may indicate a degree of asphyxia, in this case most likely originating from compression of the trunk in the face-down position rather than any compression of the neck, for which there is no*

evidence. In terms of any role for restraint, this cannot be separately considered from struggling. As is commonly the case in acute behavioural disturbances, the deceased displayed remarkable strength and stamina. Ongoing restraint and struggling in these circumstances is very likely to lead to significant metabolic disturbances with early breakdown of muscle releasing potassium, which can precipitate cardiac dysrhythmias and the development of metabolic acidosis. Indeed, in my opinion, given the presence of a background of potent stimulant drugs, this case cannot be viewed simply as an example of a case of sudden death during restraint. I therefore entirely support the cause of death proposed, namely: 1a sudden death in a man intoxicated by MDMA (ecstasy) and alpha-PVP whilst being restrained. The only suggestion I would make would be to substitute the phrase 'whilst being restrained' with 'in association with struggling and restraint."

105. I agree with everything he says except from the inclusion of restraint in the cause of death and the release of potassium from the breakdown of muscle. In relation to the first point, there is no proof as to the connection with restraint. Dr Cary undertook research into restraint and found that, even in obese people, there was de minimis increase in deoxygenation during intense exercise. I would want to know why Dr Cary is disavowing his own research. The article to which I refer is *The effect of simulated restraint in the prone position on cardiorespiratory function following exercise in humans*[14] (WIT-00036).

106. He believes that myocardial potassium increases during difficult physical activity because of tissue necrosis, but in-fact, no necrosis was observed on examination of the heart. High levels of potassium were no doubt present, but the potassium is released via channels in the cell membrane and, even though local pH levels may be greater than >8 (very elevated), potassium quickly returns to normal concentrations when stressors are removed. Finally, Dr. Cary must know that the presence of petechia is a non-specific finding which can, in fact, even occur after death.

[14] Cary, NRB, Roberts, CA, Cummin, ARC & Adams, L (2000) 525 The Journal of Physiology 30-31.

Professor Crane's Report – cause of death

107. Returning to the report of Professor Jack Crane[15], at the second bullet point under the 'final commentary and opinion' section of the report, at page 7, Professor Crane states:

> *"A toxicological analysis revealed that, at the time of his death, there were two drugs circulating in the bloodstream, MDMA (popularly known as 'Ecstasy') and Alpha-PVP, a synthetic cathinone. Both these drugs are psychostimulants affecting the activity of dopamine, serotonin and noradrenalin in the brain. Whilst MDMA is typically associated with elevation of mood and a feeling of wellbeing, Alpha-PVP has been reported to induce paranoia, and hallucinations. It this seems likely that the Alpha-PVP was responsible for the bizarre, aggressive and violent behaviour demonstrated by the deceased. In addition to their psychostimulant affects both drugs affect the cardiovascular system and specially the heart, causing change in heart rate and blood pressure and predisposing to cardiac arrythmias. It is my opinion that it was the combined effects of these two drugs which precipitated a fatal upset in the heart rhythm."*

108. I don't disagree with the toxicological analysis. Alpha-PVP is notorious for inducing a dysphoric state. Users are known to be violent and aggressive. Current theory is that deaths occurring during restraint and exertion are probably due to microvascular disease; the heart is continually being deprived of small amounts of blood and the additional burden of physical combat or agitation leads to an ischaemic sudden death. This is a plausible explanation except I didn't see any microvascular disease. Thus my diagnosis is undetermined, and it still remains that way because the autopsy was incomplete, as the genome was unexamined. It may be that a genetic abnormality is necessary for the Alpha-PVP to react with the potassium channels, but there is evidence that Alpha-PVP probably could cause arrythmia on its own. However, there is no way to

[15] COPFS-00134

determine what actually occurs except in animal studies, the results of which may or may not be relevant in humans.

109. Professor Crane's report continues at page 7:

> *"The role of restraint in deaths such as these is invariably problematical, as restraint which impedes or restricts breathing may not be associated with any diagnostic signs at autopsy. Furthermore, whilst petechial haemorrhages may be an indicator of asphyxia, where venous return to the heart is impeded, these pinhead-sized haemorrhages may also occur in other circumstances, including terminal convulsive seizures and following attempted CPR. I am satisfied that the application of handcuffs to the wrists and where the arms were to the front of the body would not have caused any degree of respiratory embarrassment or interference with breathing. Similarly, the application of the restraints to the legs would not have impeded breathing in any way. If, on the other hand, the deceased was lying on the ground, either on his back or face downwards, and pressure was applied to his trunk, i.e. by a person or persons kneeling or sitting on him, then a serious and potentially life-threatening degree of asphyxia could have been induced. In an individual where cardiac instability had already been induced by drugs, then any form of respiratory embarrassment caused by hypoxia would have rendered an unstable myocardial more prone to the development of a fatal arrhythmia (upset in the heart rhythm). Thus asphyxia could have been a contributory factor in the death if, at the time of his cardiorespiratory arrest, restraint of the type described above was taken place."*

110. I am asked for my comments on these conclusions. Professor Sheppard and Professor Crane don't see any damage to the heart. Although I saw some old scars, and they can cause an arrhythmia by themselves. I would point out that we don't really know what form of Alpha-PVP Mr Bayoh took. There are 11 different forms of Alpha-PVP. The laboratory did not test to distinguish which form it was. Why is that important? Well, because some types of Alpha-PVP are

more than 1,500 times more powerful than others. We don't know what form was ingested. For that reason alone, I am hesitant to say that the drug, alone, was the cause of death. that the drug did it.

111. With MDMA, deaths are very uncommon. Reported Alpha-PVP deaths now number in the hundreds. We simply don't know enough about it except people die after they take it, but I think of all the cases reported they've all been multi-drug. An important ruling in our court system called *Burrage*, describes a man who had bought heroin from multiple dealers and died. It was concluded that the last dealer was responsible for the death, even though all the dealers had sold the same drug - heroin. The most conservative judge on our Supreme Court, asked "How do you know which heroin it was that killed him?" Similarly, how can anyone know whether MDMA, MDA, or Alpha-PVP was responsible? The same applies here.

Second Report

112. I have also had sight of my second report[16]. This report also had attachments, which were two photographs[17] and an extract from the judgment of *Garcia v City in the County of San Francisco*[18]. In this report I give my opinion in relation to the restraint and positional asphyxia in more detail and provide a view on the effect of handcuffs and leg restraints. The report covers the retraction of Dr Donald Reay's theory in relation to positional asphyxia which he put forward over 20 years ago, which I've already discussed. I have been asked whether there is anything in my opinion in this report that reviewed that opinion in any way or whether my opinion remains the same. My opinion remains the same. I have no reason to change my mind.

113. My report mentions the hypothesis of positional asphyxia being nothing more than junk science. I accept that there is such a thing as positional asphyxia but not in the sense now in use. The term first came into use at the turn of the last

[16] PIRC-02527(a)
[17] PIRC-02527(b) and PIRC-02527(c)
[18] PIRC-02528(a)

century and was meant to describe a very specific set of circumstances: wen an individual dies of asphyxia (lack of oxygen), because their body is in a position such that the lungs cannot fill with air. Typically, an inebriate passes out on a on a bed, somehow rolls over and manages to slide down between the bed and the wall, and suffocates without ever knowing it.

114. The situation may bear some similarities to a crushed chest but the term positional asphyxia was specifically meant to be applied in situations that were not an extreme crush injury. In the late 1970s the term was redefined mainly by two individuals (Prof. Don Reay and a medical examiner named O'Halloran).

115. I have been asked about the judgment of *Garcia v City in the County of San Francisco*[19] and whether this was a case in which I gave evidence or provided a report. I didn't give evidence. However, I examined the heart, which displayed concentric left ventricular hypertrophy as seen in untreated hypertension (and stimulant abuse), not much different looking than somebody with high blood pressure. Garcia was a very fat man, it was a coldish day, but he had no pants on. He wasn't fully naked though. I think he might have had a t-shirt. He ran up to the window of a convenience store, the kind of window that are made of bulletproof glass. He was screaming at the window but collapsed there. I don't remember other details. The paramedics arrived, noted he was very hot and, as mentioned, autopsy disclosed an enlarged scarred heart. The extract I have provided is the only copy of this judgment I have.

Media

116. I have had sight of an email chain between DSI William Little and myself last dated 10 September 2015 (PIRC-03473). There's an email dated 8 September 2015 from me to William Little in which I mention that I've had voicemails from several Scottish newspapers, and I state *"I've not answered any of them. Please advise if you want me talking to the press."* William Little responds on 8 September 2015 and states *"Yes there has been some press interest regarding*

[19] PIRC-02528(a)

the PIRC engaging expert witnesses and they found out it was yourself and Jason [Payne-James]. Could I ask that if they contact you, you make no comment to them on the nature of the request we have made to you." I can see that I respond on 8 September that, *"Fortunately, all the new[s] papers left voice messages I have deleted but I will be noncommittal if they ask."* I don't have any recollection of these emails. However, I can see that I did correspond with Mr Little about these matters.

117. I am asked whether it is usual to for an expert to speak to the Press in the US once they have been instructed in an investigation. My answer depends on the judge who can prohibit discussion. If the judge doesn't make an order in a case then the expert is free to say anything. I imagine if you say anything too inflammatory, the judge will get angry.

118. I have been shown an article in the Sun dated 1 November 2015 (SBPI-00216). *"Dr Karch told The Scottish Sun on Sunday, 'I can tell you that I saw evidence of heart disease. I am not at liberty to say anymore at this stage.'"* I have been asked why I spoke to the press at that time. I have no idea. I don't remember speaking to them.

119. I have been shown a letter from PIRC to Crown Office, dated 4 November 2015[20]. At page 2, there is a heading "Scottish Sun newspaper article 1 November 2015". I am directed to the following section: *"On Monday 2 November 2015, as a result of the Scottish Sun article, PIRC attempted to make contact with Dr Karch. He responded on Tuesday 3 November and spoke with PIRC investigator William Little. Dr Karch states that in the week preceding the 1 November (having recently returned from abroad he is unsure of the date) he was contacted by a journalist and did indeed make the comment attributed to him in the article dated 1 November 2015. Dr Karch states that he was initially caught off guard but when the journalist went on to ask questions about drugs consumed by Sheku Bayoh, he advised him that he was not at liberty to say more at this*

[20] PIRC-02034

stage and terminated the call. I have been asked if this jogs any memories of this situation. No, absolutely none. This happened over 7 years ago. Over the last 4 years, I have been in significant ill health and have spent protracted periods in the hospital, including prolonged periods on a ventilator, unconscious. I have no recollection of these events.

Miscellaneous

120. I have been asked if I have ever been criticised by a judge in relation to the evidence that I have given. One case where I proved to be right, involved a truck working on a railroad track. It got hit by a locomotive, and investigators reported there were just tiny body parts and pieces. Blood marijuana was measured in blood from some of the tissue fragments. I argued that, as marijuana can remain in the body for as long as a month, I believed the measurement was a meaningless exercise. My opinion was rejected, and the decadents deprived of benefits because it was felt they were obviously intoxicated. Subsequently, the person who wrote the formulas specifying what constituted marijuana intoxication has agreed that her formula doesn't apply post-mortem. Another case was an offshoot of Hurricane Katrina. A policeman was accused of homicide when he shot a looter who was trying to cross the Mississippi River Bridge. The judge felt I didn't have sufficient expertise in gunshot wounds, and I really couldn't argue with him.

121. 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.

Date May 7, 2023

Witness Signature ... [redacted] .....