SHEKU BAYOH INQUIRY

The Sheku Bayoh Public Inquiry

Witness Statement

Dr Kerryanne Shearer

Taken by [redacted]

In person on 21 October 2022
And via MS Teams on 13 January 2023

Witness details

1. My full name is Kerryanne Shearer. My contact details are known to the Inquiry.
2. I'm a consultant Forensic Pathologist.

Professional Background and Qualifications

3. My qualifications are MBChB, FRCPath (Fellow of the Royal College of Pathologists), MRCP, DFMS.
4. I have been a consultant Forensic Pathologist for 12 years.

Double doctor post mortem

5. I undertook the post mortem with Dr Ralph Bouhaidar. I was first on call that weekend and so was the primary pathologist. In Scotland we have the double-doctor system for corroboration, so there are always two Forensic Pathologists involved in any post-mortem that potentially might result in court proceedings. I was first on call, which means I take the call for that case and I am the consultant that undertakes the post-mortem. I undertake the external examination, do all of the dissection, but the second pathologist will be there scribing what I am saying and we will discuss the case as it proceeds and share opinions. A provisional report with the findings at the post mortem and a note of further investigations

taken will be provided by the first doctor as quick as possible after the case has been undertaken. A final report (when we have all the extra information that we have asked for during the post-mortem and the results of various investigations including toxicology and histology are available) will be issued and agreed and signed by both pathologists. I will put the report together, the second pathologist will read it and possibly suggest some changes. We will have further discussion and are also likely to ask the opinions of other colleagues. The final report is very much a joint effort but with me leading it and me primarily writing the report.

Information provided regarding the circumstances of death

6. I had been on call that weekend and I got a call about this case. It think it was on the Sunday. It is normally the Procurator Fiscal on call for homicide that phones the pathologist out of hours, not the police. There will then be a discussion between the fiscal and pathologist as to any need for scene attendance and the best time for a post mortem to be arranged. I remember it was a very dynamic situation and there was insistence for the post-mortem to be done as quickly as possible.

7. I have been asked whether I would normally keep a telephone note of these kind of calls from the procurator fiscal. Not normally on file, no. I normally just make a note of the name of the deceased, a date of birth and a time and a date of death. I would also make a note of any notable information the fiscal tells me with regards to the circumstances surrounding the death. I need clerical information to let the mortuary know what to expect and also to be able to clarify the body has definitely arrived at the mortuary. The handwritten notes I make at this time do tend to be limited and the information available at the early stages of the investigation is often limited (and not accurate) and I know this information will be superseded by what I will receive in the coming hours/days so I do not tend to keep this information for the file.

8. I have been asked whether any reason was given for the post mortem being done so quickly. I cannot remember any specifics. Everyone, including myself, was aware of the media attention and the circumstances of this case, and I assume they wanted to get the post-mortem done reasonably quickly so they had

more information as to what may, or may not, have happened. We would normally have undertaken this type of case reasonably quickly anyway because we do any potential homicides either the same day or following day. If I am called on a Sunday, unless it was a very early call and the body had been transferred to the mortuary I would normally undertake the case on the Monday afternoon. That is a standard procedure for any homicide or potential homicide post-mortem.

9. This case was a particularly difficult case to be involved in; although no cases I undertake are terribly pleasant. I cannot remember the specifics but having an awareness of the background to this case with the extensive media interest, the circumstances surrounding the case and obviously the awareness of how the family was being affected.

10. I have been provided with a copy of my initial (provisional) post mortem report (PIRC-01444). In the section background history, you can see a lot of the information was missing as I did not have hospital notes, or general practitioner notes. When this case was reported to me, obviously the background was noted, the police involvement was noted. In the background circumstances I would normally put in the pertinent information I have received from the fiscal and from the police report written yet or, in often in potential homicide cases we get a police briefing paper. I had the information that was available to provide at the time. In this case there was a briefing meeting prior to the post mortem examination and I was given three A4 sides of information (single sided) and I used this information for the majority of the information in the background circumstances of the provisional post mortem report. The post-mortem examination was undertaken on the Monday (4th May), the day after he died (3rd May), and my provisional report was issued on the 6th May. I was asked to put it out as quickly as possible to provide as much information as possible. Normally, for a preliminary report, I would wait for photos and I would maybe wait for more information, but there was a request to have the report available as quickly as possible for this case, I assume because of all the background circumstances regarding the case.

 [redacted]

11. In terms of where this background information came from, as stated above there was a briefing prior to the post mortem examination and written information was provided. Normally, prior to a post mortem examination happening in the mortuary, normally in the conference room, I would sit down with the procurator fiscal and police including the SIO. In other circumstances it would be the police who were providing information, but this was obviously not the case here and it was PIRC that was involved which included attending the post-mortem and taking productions. We tend to keep everything we are given in paper form in the case file and we also have these documents scanned and kept on the computer system.

12. On file I have a copy of the sudden death report (PS-01405). This document is where we normally get most of our information from, but I do not think that was available before I did the post-mortem because normally it takes a day or so for it to come through because it has to actually be done by the police or whomever is filling it out. We are normally provided with an instruction sheet (a single sheet) from the Procurator Fiscal that gives us information including what type of post-mortem they are instructing i.e., a two doctor post-mortem. In the case of potential homicides we more often or not do not receive this document prior to the post mortem.

13. I have been asked whether I have a note of the briefing provided to me by the PIRC. This is noted above.

14. While I did not have the sudden death report at the time of doing the post mortem, I did have a briefing note and often the information from one to the other is repeated. I do not think having a sudden death report would have changed what I had done at the post mortem. The date of receipt of the death report is 6th May, the day I issued my provisional report.

15. I have been asked about the language of the background section from the initial post mortem report and its similarity to the language of the sudden death report. What I normally do is I dictate it and then i will be typed by one of the secretaries. The post mortem was done on 4th May and I think [redacted] nk I dictated it on 4/

5th May because it was requested the report be available ASAP. The death report was received at quarter to three on 6th May. Sometimes they do send it to me and send it to the secretaries at the same time. Normally I would say that in my background circumstances the source of the information I am using i.e. from a police report as I do normally in all my routine and homicide cases.

16. I certainly remember having a knowledge of batons being used because we were specifically looking around the head and neck for external and internal injuries, and I think I commented in my final conclusion that some of the injuries that we saw could have been related to him being hit by a baton. This information was present in the briefing paper.

17. I have been asked if I remember being told that CS Spray and PAVA spray had been used on Mr Bayoh. Yes, that was mentioned at the briefing. Swabs of Mr Bayoh's face, mouth and nostrils were taken to capture any residue from the CS and PAVA sprays. Then I contacted various labs to try and get them analysed, but could not find a lab that would analyse the samples. I have outlined that in my conclusion as well. So, we were aware that these sprays had been deployed during the struggle.

18. I have been asked about the fact that I do not mention in the initial post mortem report that the deceased had been sprayed with CS spray or PAVA. I do mention this in my final post mortem report, which is a lot more detailed than the initial report. The initial report was done quickly to give information quickly. The fact I have not noted it in my provisional report is an oversight but given the sampling I was definitely aware prior to the post mortem.

19. The "pathologist's briefing note" (COPFS-02540) states that *"The pathologist was also informed by John Ferguson that the deceased was Muslim to allow the pathologist to consider and make informed decisions during post mortem".* I have been asked if I was aware that the deceased was Muslim and if so how would this information affect the conduct of the post mortem. Yes, I think I was made aware of this. In terms of how it would affect the post mortem, the main consideration would be to get the post-mortem examination done as quickly as possible because I am aware there is [redacted] place as is a reqirement that the burial take

soon as possible within 24 hours. So that would be the main reason. We were planning on undertaking the post-mortem as quickly as possible, and I cannot remember if his religion was one of the reasons for this. Often in such cases we may be asked to undertake a view and grant examination only, but this cannot be considered in potential homicide cases. In terms of the actual carrying out of the post-mortem, knowing someone was Muslim would not alter how I undertook the examination. I cannot think of anything that would change what I was doing or make me think about things differently. We get a lot of requests in relation to deceased persons who are Muslim or have particular religions, family requests for the post-mortem to happen more quickly, which we are more than happy to accommodate in order to allow the deceased to be returned to their loved ones as quick as possible and as such they are more likely to be buried within the timescale that is required for their religion.

20. The pathologist's briefing note also discusses the fact that I was advises that the deceased had taken drugs. Specifically it states that *"This discussion lead to the potential of a history of drug abuse that may require the taking of head hair for analysis. As the deceased was Muslim the pathologist stated that she would seek guidance from the attending Procurator Fiscal (Mr Bernie Abblett)."* I have been asked to comment on this. I do not have any recollection of the comments that are being attributed to me here. Mr Bayoh had very short hair. I think there was a discussion at the time that Mr Bayoh did not have hair long enough for drugs testing. If someone does have a potential history of long-term drug misuse – especially stimulant drugs – I will often take a hair sample because when there is a suspicion that someone is chronically abusing drugs, their hair grows at about roughly 1cm a month and if we take a couple of centimetres at least, we can look at their drug use over the previous few months. I do this testing in routine cases and occasionally in homicide cases – but, in this case we did discuss it, but the hair was too short. You can take pubic hair, but it does not give you any indication of length of exposure, so it is not a terribly useful test to undertake. As pathologists, we have lots of samples that we take for ourselves that help us in determining a cause of death; for example, histology, toxicology and neuropathology. But at the post-mortem, particularly in double-doctor post-mortems, there are a number of sample[redacted]n this that we take for the police (PIRC in

case) and the Procurator Fiscal. We take these on the instruction of the Procurator Fiscal, and hair is normally one of them. We normally take some pulled hair (pubic and head hair), and that is more for any DNA comparison if they find hair at a scene. These are comparative samples and not pathology samples and have no bearing on the cause of death. We take these samples purely under instruction from the Procurator Fiscal who discuss it with the police/PIRC. We do not do anything with those samples other than taking them, signing the police bags, handing them to PIRC and documenting they have been taken. So the hair in this case that was taken was a PIRC production, and it would have been at the request of the Fiscal in conjunction with PIRC.

21. At the time of performing the post mortem the hospital and GP records were not available. I have been asked whether I would normally have sight of those records before performing an post mortem. In maybe about 70 per cent of cases I may not have them or indeed need them but there are some cases that I will not do the post-mortem without having the records. Those are mainly homicide post-mortems where someone has been stabbed and they have gone to hospital and had procedures carried out like surgery. I need to know what a surgeon has done before I can undertake the post-mortem. There will be some occasions where I will say categorically, "I am not doing that post-mortem." I did not feel on this occasion that having the GP notes or hospital notes was going to make a vast difference to what the actual post-mortem findings would be, so I did not think there was a need to hold things up in this case.

22. Occasionally, cases do wait, but the vast majority we can go ahead and do the post-mortem. A perfect example is a lot of homicides happen at weekends and we do post-mortems Saturdays and Sundays and will not be able to obtain GP notes because GP surgeries are closed. It can also be very difficult to get hospital notes because doctors go off shift and police cannot get statements. But, categorically, and I have done this on several occasions in my career, I will not undertake the post-mortem until I have hospital notes if I think it will be detrimental to the case, but in this case I did not feel the need to do that. In this case, Dr BouHaider and I were in full agreement that we could go ahead with the post mortem without the records.

 

23. I am asked whether, once I had all the available information from GP records, hospital records, witness statements etc, whether there was anything else that I would have done at the time that I did not at the post-mortem?. No, I do not think there is anything. We did absolutely everything that we could do at the post-mortem, every test that was appropriate and available to us.

24. I think that probably was also supported by the fact that the family had their own forensic pathologist, Dr Nat Cary. This was not a defence post-mortem as such, the name for a second post mortem that is done by a third pathologist. Pre-COVID, the pathologist would normally come and undertake their own post-mortem examination; do an external examination again and look at all of the organs usually in the presence of the first pathologist. Another reason for me to get my provisional post-mortem finalised as quickly as possible was to provide this to Dr Cary for him to make a decision about attending in person. From the information provided Dr Cary did not feel that he needed to come and look at the body in person or suggest any other investigations he would have undertaken. He put his report together based on the information that we had provided him, in addition to the information received when various results were received.. I do not think there was anything that we would have done any differently had we got information after the post mortem that was available prior to the post mortem. If I thought that there was information that would have changed things, I would not have done the post-mortem as quickly, we would waited for the hospital notes or for whatever else if I thought that would make a difference to the outcome of the post-mortem.

Identification of the deceased

25. I have been asked whether I was made aware by the police or PIRC of any issues regarding identification of Mr Bayoh by his family, or what their understanding was of the family's attitude was towards identifying the body. I honestly cannot remember. What we would normally do is (although not so much now, post-COVID), in a double-doctor post-mortem like this, family members or friends; essentially two people who knew the deceased in life would come to the mortuary and we would have a very brief chat with them, and then we would take

 [redacted]

them into a room with their loved one being behind a window on a trolley where just their head would be exposed, and we would have to ask them to let us know that this is their loved one, and that would be the identification in life.

26. Identification is something that is normally decided by the Procurator Fiscal. They tell us what they want to do from an identification point of view. Normally, we have a discussion with the Fiscal pre-post-mortem to confirm how we are identifying the deceased, because ultimately, it is the Fiscal's responsibility to release the deceased after they are finished with their investigations and they have to be happy that identification has been done optimally. Cases will be different. It may be that there are no family or friends available to identify, it may be that police knew the deceased well enough to also do the identification in life if they have a criminal record and they are known to the police. It may be if they are not suitable for viewing due to injuries or decomposition and DNA may be used for identification. There are all sorts of ways bodies can be identified. I am not sure why this was done in this way, but we normally take our instruction from the Fiscal.

27. I can see from the initial post mortem report that the body was identified by fingerprints and by John Ferguson and Peter Grady. I cannot remember why family or friends were not involved in this case. I am asked whether I had any concerns regarding proceeding in this way. No, not at all. I am asked if I was aware that the family did not want the post mortem to go ahead at the time that it did. I am aware of these things now. However, I cannot remember what I was aware of at the time. But, from my point of view, I do not decide how an identification is undertaken, I can advise, i.e. if decomposition is such that viewing is not recommended but it is not my remit to decide how this is done. So even if I had knowledge that the family did not want it to go ahead, I cannot influence what happens next. It's not a pathology decision. I'm instructed to undertake the post-mortem. Once a person dies particularly a non-natural death, the death is referred to the Fiscal and it would be very difficult for a family to stop a medico-legal post mortem happening and we often have to do post-mortems on people whose family may have religious objections, because unnatural deaths normally require a post mortem and this is a legal decision.

28. I have been asked what would happen if the family were to make a request for the post mortem to be delayed. This would be a very unusual request as usually they request for the case to be done sooner. Yes it is possible for a request of that kind to be granted within constraints and we normally do our best to accommodate requests. All of our double-doctor suspicious cases are done in the afternoon, as we have morning lists of post-mortems which are our routine post-mortems. We normally do six, although now we do eight because it is busier. These are people who have been booked in normally for several days, and these are people who have funerals booked for maybe a day or so after the post-mortem. We keep afternoons free for suspicious post-mortems. Where post-mortems need to be rescheduled, what we would say is we cannot do it first thing in the morning because we do not cancel routine cases in order to do suspicious cases and the fiscals would never expect that because they also deal with the natural deaths and would not want families to end up having to cancel funerals etc. But the actual instruction for the post-mortem and any delay would have to come from the Fiscal.

29. I have been asked about the timing of the post mortem. This is usually determined after a discussion with the Fiscal. There was obviously massive media interest on everything that was going on in the background and they wanted the post-mortem done as quickly as possible, and that is what we did, hence the call on the Sunday and we did it on the Monday. But again to reiterate that these cases are often done as quickly as this one so it was not unusual timing.

30. I have been asked about the training of pathologists in relation to religious and cultural considerations during an autopsy. We certainly learn about that during our training. We are aware and, as much as we can, we try and help. The main problem is that once it goes into the Fiscal domain, it now becomes a legal requirement. There will be cases where, for example, if someone has committed suicide by hanging and they are Muslim and there are objections to a post-mortem being undertaken, as long as we have a specific set of circumstances and we're 100 per cent happy that it's definitely a suicide death, we may do

 

something called a view and grant examination. This involves an external examination the body of the deceased while considering the deceased's history and the events surrounding the death, then we can give a cause of death. This does not involve an invasive examination of the body.

31. We do try and take all of that into consideration. I think the main thing that we do is we try and do cases as quickly as we possibly can. If a death is reported and family are wanting things done quicker, then we will try and fit them in as quickly as possible. Our waiting times can be a week, two weeks depending on how busy we are, but we try and accommodate family by doing cases quicker. Legally, they may require to have a post-mortem done and we take instruction from the Fiscal and have a discussion with them about the best way forward. If we are doing a case earlier, it means there is another case that may have to wait longer so a discussion needs to be had. "If we're doing one earlier, who do we delay?" or, "Are you happy that we do view and grant this case?" They have to be happy as well and we have to come to a conclusion together. It's not something that is specifically in our training, but we certainly learn about it as we are training and when we are consultants.

32. In terms of CPD, we have modules that you can do associated with the NHS. There is an equality and diversity module you need to complete yearly. Generally, we are all educated adults, have friends who are Muslim or are various religions. We will try and do everything we can to help because we completely understand how difficult it is for family members.

Final Post Mortem report

33. I have been referred to the final post mortem report (PIRC-01445). At page 3, under external findings we describe "The body was that of a dark brown skinned adult male of heavy build." I have been asked to explain this. The use of the words dark brown skinned is a visual description rather than a racial description. In relation to the words heavy build, it may be taken to refer to someone who was overweight. However, in this case, it was a reference to his muscular build. I

would have been better using the term muscly because he obviously was not overweight given his BMI was normal.

Internal findings at post mortem

34. At page 10, the report states *"The liver (1800g) appeared congested and showed focal pale areas".* I have been asked what significance this has. It does not really have any significance. It is just very much a visual description that we see in a lot of people who have died. It is very non-specific. It does not tell you anything about the mechanism of death. It does not tell you about the cause of death; it's just a gross description of the liver, a common finding in death, especially in people who have been resuscitated over a period of time as well. It's not anything to do with any pathology.

35. Continuing at page 10 of the report, there is a heading of "musculoskeletal system". Here we have recorded *"various areas of haemorrhage to the back, legs and wrists, including a rim of subcutaneous haemorrhage measuring 11 centimetres by 1 centimetre around the left wrist."* I have been asked if this is consistent with the use of handcuffs. Yes, it is. I have said that in my conclusion as well, that it would be in keeping with him being restrained by handcuffs, which I knew about prior to the post-mortem examination.

36. At page 9 of the report, we record that facial dissection that was performed in this case. I have been asked in what circumstances facial dissection is undertaken. Facial dissection is only performed in cases where it is indicated. Yes, it is not something I would do lightly. The reason it was done in this case was because we were given information about him being hit with batons, being face down on the floor, and another reason, with him having dark-coloured skin, it can be very difficult sometimes to see injuries, especially bruising. There were several reasons. Equally, even if he was a Caucasian man, he still would have had a facial dissection undertaken because he had some injuries and we wanted to see exactly what was going on underneath the injuries. Also with blows to the face, even with the most minor external injuries, you can still get internal damage, broken bones like a broken jaw, broken nose, broken orbits that I would not

 [redacted]

necessarily see externally. I would not have hesitated in doing a facial dissection in this case.

37. I have been asked whether I anticipated that the family would want to view the body after the autopsy had taken place. Given the way the identification was carried out, I would have been aware that Mr Bayoh's family had not had an opportunity to view the body, so yes I would assume they would want to view him. However, the way the mortuary staff expertly reconstruct the body makes it difficult to tell that a facial dissection has been carried in the vast majority of cases. As such I would not have avoided doing a facial dissection for this reason. I would have had no concerns regarding the family viewing the body after the post mortem. We repeatedly do post-mortems where families have not viewed their loved ones, and then they will view them afterwards. A lot of the time in the routine cases, it will be in the undertakers rather than in the city mortuary because the city mortuary is not the ideal place to come to view your loved one. But, no, routinely people will see their loved ones after the post-mortem has been done.

38. At page 9 of the final post mortem report, it states: "*Following the CT examination which was undertaken after the post mortem, the 7th cervical vertebra and left 1st and 2nd ribs were re-examined […] Soft tissue overlying the front of the posterior part of the left first and second ribs (just adjacent to the thoracic spine) was removed and revealed focal possible soft tissue haemorrhage measuring 0.5cm in diameter overlying the 1st rib. Underlying this there appeared to be a fracture through the rib. There was no evidence of injury to the left second rib.*"

39. I have been asked why the report states that there "appeared to be" a fracture. By this point, the body had begun to decompose. Things had begun to break down, and the fracture was tiny and very well-hidden. We often have this with bony injuries where we are not 100 % sure if there is definitely a fracture there. We then take histology, and we either confirm or exclude it by looking at it down the microscope. So that is why I kept that in and to make it clear that it was really difficult to find, and we were not 100 % sure just on gross findings that it was fractured.

 

40. This is why it was not apparent during the initial examination. The first rib is hidden away behind the clavicle, deep in the shoulder girdle. During the post-mortem, there was not anything obvious. Normally what draws our attention are things like soft tissue haemorrhage or bleeding around it, and there was not anything obvious there at the time. The fact that it was also not present, obviously, on the initial x-rays that were done is another indicator, but it has only been picked up on CT scan which shows such things in much greater detail. It was really hard to see, hence why we did not see it at post-mortem.

41. Similarly, I am asked about why I describe "possible" the soft tissue. Again, after death, things begin to break down, tissues in the body begin to change colour, and they can become quite darkened, so post-mortem changes can mimic what may be haemorrhage, and it can be really difficult to differentiate what was ante-mortem and what was post-mortem. That is the main reason. We just were not sure, with the naked eye, if there was definitely haemorrhage there.

Further Investigations - histology

42. I am asked about the investigations that list at pages 10 and 11 of the final post mortem report. Histology is a standard investigation that we do on probably about 95 per cent of our post-mortems. It's best practice. It's in all the guidelines of best practice, especially in double-doctor post-mortems. You categorically have to take histology samples because you are looking for disease processes in organs when you may not necessarily see anything grossly, and ultimately, you are looking for a cause of death. As well as confirming, you also have to exclude various disease processes. We take tiny little samples, maybe a centimetre and a half by just a few millimetres in thickness. This is standard in most post-mortems.

43. For the heart, I've taken a heart slice. I also taken tissue from the lungs. Also, I took some liver, some thyroid gland because this thyroid gland was slightly enlarged, and pancreas, adrenal glands (little glands that sit above the kidneys that produce steroid hormones) and kidneys. I also took some histology from the

kidneys. I also took some histology

rib fracture that we found on the CT that was done afterwards. The tissue we take varies from post-mortem to post-mortem, depending on what you are finding at post-mortem, but in this case and in the double-doctor cases, the best practice is to take a wide range of samples from all of the main organs in addition to anything else that you think is appropriate..

44. The next thing listed is neuropathology. Again, standard in this type of case where there has been blunt-force trauma to the face and head areas, I would always keep the entire brain to be looked at by a neuropathologist. We have a professor of neuropathology that looks at all of our forensic cases, he will fix the brain; do his external examination; section the brain; and take pieces of tissue from the brain to look under the microscope. Then the brain goes back to the body, which is what happened here. That is detailed in my report. That, again, is best practice in this sort of case where there has been trauma. Even if you are not seeing anything externally on the brain, there is still a chance that, when you cut the brain, trauma is seen or, when you look at it down the microscope, that there is potential traumatic injury, hence why we kept the whole brain, and the spinal cord in this case.

45. I have been asked whether Mr Bayoh's family were aware that the brain and spine were retained. They should have been and I assume it was done; however, I do not have any dealings with the family. The family discussions come through the liaison officers, via the fiscal, but I think normally they would explain these things, but I cannot say what happened in this case. Normally I think that sort of information is given as it is a reason for a delay in the body being released. In saying that, suspicious cases are different because normally there is a defence post-mortem, so the body is kept anyway, but depending on availability of neuropathologist – if they are out of the country or because we only have one that we use – it can delay the release because the body cannot be released until the brain has been looked at, sampled, and the remainder is put back to the body. We do not give a death certificate until that brain has gone back to the body because the body cannot leave the mortuary until that has been done. The

 

Fiscal or whoever family liaison would normally have an understanding of what information was passed to family.

46. The next sample we took is toxicology. Again, toxicology is standard and routine and best practice in all double-doctor cases. We have to take toxicology in all of these cases. It was particularly important in this case given the history of potential drug use and, potentially, him being on drugs at the time of the incident. Our standard samples, if we can obtain them, are blood samples (best because they give the most accurate account of antemortem levels of various drugs). We were able to get some blood in this case. Again, we always have two different samples if we can. Best standard is urine with blood, that is mainly because if you are looking for alcohol, it's a good comparison to know the alcohol levels of blood and urine, and it can give you an idea about the degree of intoxication in hours before their death. There are other drugs that are can be found in urine as well, and if the drugs have been in their system and metabolised, they may no longer be present in the blood, but we may find them in the urine. If urine is present, we will take urine. If urine is not present, we will take vitreous humour, which is the fluid that is in the back of the eye – but I did not have to do that in this case because I had a urine sample.

47. I took two urine samples because there was the history of potential steroid use. For that, a urine sample has to be sent to a lab in England. It is not something that our lab will test for, so that is why a separate urine sample was taken to be sent away. The facial swabs, mouth swabs, nasal swabs, and sections of lung are unusual, and the only reason they were taken is because of the history of PAVA and CS use. We were not sure at the time what we would be able to test for. We knew our lab did not test for them. I think PIRC were involved in trying to source if there was anywhere that we could send those samples to. But at the time of the post-mortem, we did everything that we could to have the samples available, but after their research and my research, we could not find anywhere that would actually process the samples. Given that he did not seem to have any ill effects from either of those sprays, and had none of the complications that we see, (spasms in the back of the throat, spasms in their lungs, breathlessness, and very itchy eyes and redden eyes) it did not sound as if they had any

it did not sound as if they had any

detrimental effect on him. As such, it became less and less necessary to find somewhere, but we did try, but I could not find anywhere that would process those samples in the UK.

48. I took some bacteriology and virology samples. The main reason for that was because I was given the information that he was behaving very erratically. He was very agitated prior to police involvement, and that can be a symptom of an infection of the brain. I did not see anything grossly, but you can get a viral encephalitis, which is an inflammation on the surface of the brain, and you can also get bacterial infections that can cause you to not behave like yourself, be completely out of character, be very angry; and I wanted to exclude that as potentially a reason for his behaviour prior to the incident happening.

49. I have been asked whether I have a copy of the virology report as there is not a virology report appended to the final post mortem report. The virology results are stated in the report. We get hardcopies of them that come in via post. We can also access them online as well. We can access them through the hospital system because our samples go to Edinburgh Royal Infirmary, but we normally get a hardcopy of all the bacteriology and all the virology, and that's what I would have taken. I normally incorporate them into my report in that I put all of the results in there. I have provided a copy of the virology report (WIT-00004). They do not give an interpretation. They're all negative results so there was not interpretation to be had.

50. In the final report, there's a list of further investigations which includes skeletal survey and CT examination. I have been asked what the difference is between the two. A skeletal survey is plain X-rays, whereas a CT is computerised tomography; actual pictures of the body, pictures of the bones, soft tissues and of the organs. CT is much more detailed, whereas the skeletal survey is just looking at bones.

51. I have been asked to explain why both were carried out in this case. We do not always do CT pre or post post-mortems; we occasionally do a pre-mortem CT. In some places it's done more frequently than we would do it, certainly. In this case

[redacted]

we had already done the post-mortem. we had looked at all the internal organs, but when you are doing the post-mortem, unless you physically dissect out all of the bones, which is a very invasive thing to do (I would never want to do that if I could possibly avoid it). We had already looked underneath the skin at the tissues in the arms and the legs and the back, but the last thing I wanted to do was make any more cuts in the body and deform the body any more than we already had. As such, we decided to visualise the bones with a skeletal survey, so it means that we could see the bigger bones that we had not visualised – mainly the long bones, so your arm bones, hands, leg bones and feet. We could be categorically 100 percent sure that there were no fractures there that we would not have been able to see at post-mortem.

52. Normally we get indicators if someone does have fractures in a long bone because externally you will probably see bruising or you might see some tissue swelling, but we wanted to just be 100 % sure that we had not missed any fractures of any bones that we were not able to visualise. We visualised the skull, so we knew that there was not anything there. We visualised the rib cage because all of that internally we can see. However, we cannot visualise the bigger bones without cutting into the arms and legs, so we wanted to do that. Because we had already seen all of the organs, we thought the easiest thing to do would be a skeletal survey because we just needed to see the bones, we did not need to see the soft tissues. But the skeletal survey could not visualise the neck bones well, so the radiologist could not comment on the c-spine which was really important; we wanted to know absolutely categorically that there was no fractures there. We then went to CT and that is when they picked up on the first rib fracture that we had not seen at post-mortem and had not been seen on the on the skeletal survey. I made that decision to do the CT scan in consultation with Dr Bouhaidar. Everything that is done afterwards is always a discussion between the two of you just to make sure you are both in agreement about what is being done. Dr Bouhaidar has a special interest in post-mortem CT as well, so he is our expert in our department with regards to that.

The Heart

53. The heart has four chambers. It is a globular structure. The two large muscles that make up the heart are the left ventricle and the right ventricle, so they are what pumps the blood around the body. We look closely at the heart, looking at the coronary arteries, because coronary artery disease can kill people suddenly, and we also look at the valves and the heart muscle, and we cut into the heart looking at the chambers and serially slicing to see the inner parts of the heart muscle because there may be scarring there that you cannot see externally.

54. In this case, I did full heart histology in that I took a mid-ventricular slice, so I took the middle part of the heart. The heart slice is fully embedded in paraffin wax, and we get slides cut so that I can reconstruct a slice through the heart look at the back, front; right and left side of the heart and differentiate, the left and right ventricles. I look at slides representing each part of the heart to tell if there are any disease processes present and exactly where it is in the heart.

55. At page 13 of the report, it states *"The heart was mapped and the sinoatrial (SA) node and atrioventricular (AV) node sampled."* I am asked to explain what we mean here. Mapping is a block diagram of the heart slides. This allows anyone looking at the slides to identify a particular slide and understand which part of the heart it contains. The sinoatrial and the AV nodes, again, are sampled separately so we know exactly where they are and exactly where they've come from in the heart. I have provided a copy of the block diagram which I created to go with the heart slides (WIT-00002).

56. I did this because Mr Bayoh died very suddenly, I wanted to make sure that there was no heart disease that would have played any role in his death. Also, with the history of drugs that that we were given, when people chronically use drugs, it can cause heart disease and microscopic scarring in the heart, so I wanted to get as much information as possible as I could from the heart. This is our standard protocol for doing that. The conduction system is the nerve pathways, and you can get damage to those nerve pathways that can, again, cause the heart to have irregular rhythms, and stop, and people can die. Taking full heart histology is best practice for examining the heart in Scotland in this kind of case.

57. So, again, we do not do this with all of our cases. Such detail is only done with specific cases such as this one involving the sudden death of a young person. Normally I would just take little bits of the heart and I would look at them all together. So, from that, I can work out what is the left and right ventricles, but I would not be able to say which part of the ventricle – but, for a lot of the cases, it does not matter; I do not need that amount of detail. But in cases such as this, I do need that amount of detail. I need to know exactly, if there is pathology, where the pathology is.

58. When we state here that "no evidence of significant abnormality", we mean that we cannot see anything in the heart that would cause concern in terms of playing any role in his death; there are no chronic changes, no acute changes, so his heart looks normal down the microscope – and also grossly as well.

The Lungs

59. I also took bits of lung. Again, this is best practice to take samples from each lobe of each lung, so the left lung has two lobes, the right lung has three. I tend to take five sections, and that again is just tiny little sections that are put in paraffin, and cut and stained, and then we look at that under the microscope.

60. In relation to the lungs, the report states: *"There are extensive congestive features and areas of pulmonary oedema. There are widespread areas of subpleural chronic inflammation and pigment laden macrophages."* I have been asked to explain the significance of this. The congestion and oedema, again, is something that we see fairly routinely at post mortem, especially in people who have been resuscitated for a period of time. It just means that the lungs are a bit wet, they have got more fluid in them; it's not a definite indicator as to the underlying reason that the person has died. It is not specific to anything because it can happen with a whole variety of deaths.

61. The lungs are covered by a surface called the pleura, and the pleura lines the chest wall and also covers the surface of the lungs. Just underlying that surface, there was some chronic inflammation. This tells me something chronically has affected the lungs. He might have had a previous infection, he might have had a

DocuSigned by:
previous infection, he might have

previous episode of pleurisy but it is nothing significant in that it would not have played any role in his death; it's some sort of chronic process that has happened, but would not have caused him any problems, certainly acutely. The pigment-laden macrophages – macrophages are cells that basically mop things up that are not supposed to be there. So, if you have an infection, your body has a response to that; it produces various cells to help you fight that infection, and one of these can be macrophages. We can also see it in people who smoke things tobacco, cigarettes or cannabis or other kind of drugs, because they are putting something into the lungs that is foreign that the lung is not used to it, and will automatically try and remove it, and to do that, it produces these cells. We see them a lot in all sorts of cases, commonly in younger people. Again, it is not a significant finding, as in they have got nothing to do with the cause of death; they just show a chronic process of some kind.

62. In relation to the thyroid gland, the report states, "The two sections taken were reviewed by an Endocrine pathologist. Although autolysis and only two sections being available limits assessment, the appearances would be in keeping with a multi-nodular goitre. There are very focal areas of chronic inflammation but no evidence of malignancy." I am asked to explain this section. It shows that Mr Bayoh's thyroid was slightly enlarged; and a multi-nodular goitre is a sign of thyroid disease, normally hyperthyroidism where you get just little nodules on either side of the thyroid gland. Again, some people can be completely asymptomatic with it, some people can be on medication for it. But again, there is nothing to suggest that is anything to do with his death, it's just an incidental finding that we sampled just to clarify this.

63. In relation to the left first rib, the report states that *"a fracture is confirmed but there is no evidence of obvious associated haemorrhage and a special stain for iron is negative."* Earlier, I explained the use of the description "possible" haemorrhage as it was not clear if it actually was haemorrhage. On examination of the rib histology, I could not see anything histologically to support that. That is why we do the histology. But I do not like changing my initial gross findings because I think it then takes away from what I am thinking at the time, so that is why I often keep the gross findings as possible or probable and then use the

histology to confirm or exclude what I was thinking at the time. We could not see any obvious haemorrhage there.

64. An iron stain is something that shows historical haemorrhage. Again, it goes back to macrophages, and they mop up the pigment, the haem that is produced when something bleeds. If something has been bleeding for a period – and again, it is very difficult to say specifically how long they have to have been bleeding for macrophages to start mopping this up – then we will get this iron stain that basically lights up with blue colour and tells us that there has been bleeding there. But if that stain had been positive, then the bleeding would more likely have occurred prior to the incident; the fracture would have more likely predated the actual incident, the scenario that happened, and the resuscitation – but we could not see any pigment to suggest that. But I am aware it was then sent to a Professor Freemont who did more specialist stains and thought the fracture happened prior to even the incident occurring.

65. The radiology results are discussed at page 14 of the final post mortem report. The history here was that an x-ray of the body was carried out however the lateral spine could not be visualised. The body was then re-x rayed on 27 May; however, it was not possible to get meaningful imaged due to decomposition. Consequently, CT scan was carried out on 28 May. the post-mortem report refers to a skeletal survey being carried out post-mortem, but then talks about the body being re-x-rayed on 27 May. The hope was that the re-x-ray would stop a CT being required, but it's not been possible to get visualisation of the c-spine so we have decided to go to CT instead. The radiology report letter is dated 4 June and has the date 13 May in the heading. This is a typo and may have been the date of the first x-ray of the body.

66. The neuropathology results are recorded page 14 and the neuropathology report is appended to the post mortem report. The findings do not provide any explanation for the cause of death. The changes which the neuropathologist reports to have seen in the brain are consistent with the circumstances of Mr Bayoh's death.

67. Page 15 of the report records our conclusions in relation to the cause of Mr Bayoh's death. We states that there was *"no evidence of natural disease that would have played any role in death".* I can specifically confirm that there was no evidence of any heart disease. We note a number of minor blunt force injuries and explain that internal examination of the head and face showed areas of bruising in keeping with blunt force impact. This was in keeping with the reports of baton use. The use of batons on the head was something that I was aware of prior to starting the post mortem.

68. This continues that "internal examination revealed a fracture to the left first rib, just beside the spine and this could have been sustained whilst he was being restrained, albeit the possibility of it occurring during resuscitation cannot be completely excluded." I have been asked to comment on this. It's very, very rare to see a first rib fracture as a consequence of attempted resuscitation, but it can happen. I have been asked whether the first rib fractures I have seen as a consequence of CPR were isolated rib fractures or whether other ribs were fractured in addition to the first rib. These are historical cases, so to know that off the top of my head is difficult. But I would imagine it would not have been isolated. There would have been other rib fractures associated with that, because it would be, again, even odder to have an isolated first rib fracture from resus and not have other ribs involved. Ibe 99 %sure that other rib fractures would have been fractured as well.

69. Whilst I have seen it in other cases, it is rare because the first rib is hidden away behind the shoulder girdle, so it is very difficult to get to that when you are resuscitating, but you cannot completely exclude categorically that it has not happened during resuscitation. However, it is probably more likely during the restraint procedure given that he has been on his back, potentially with someone on his back or knee on him or body on him would be the more likely cause.

70. I have been asked whether the pressure or force could have been to the front of the body. Potentially it could have been, if it was applied directly over the area of where the rib hides; you've got the clavicle and the rib is hidden underneath but, again, given that there is no damage external to that – so there was no damage

to the clavicle, there was no damage to the subcutaneous tissues overlying it – it probably makes that less likely that it has been to the front and it is probably more likely to the back, but it is impossible to be specific. It is a really unusual fracture, because it is so hidden away and deep into the soft tissues. I think one of the common ways is to have your arm out and the transfer of forces up towards there and the rib breaks, but you can get it from a direct impact onto the area so either on the back or potentially on the front, but more likely on the back. The conclusions state that we found no injury that would account for Mr Bayoh's death.

71. The conclusions discuss the drugs taken by Mr Bayoh, at page 15 and 16, and specifically state that either the MDMA and the Alpha-PVP could have caused a fatal cardiac arrythmia. I have been asked whether there would be any pathological evidence that Sheku Bayoh died from a fatal cardiac arrhythmia. Everybody dies of a fatal cardiac arrhythmia because that is always the terminal mechanism: the heart stops beating. It goes into a rhythm that it cannot sustain, and it stops beating, but it is not something you can prove pathologically. When you go back to the medical notes which document a respiratory then a cardiac arrest, that is the fatal cardiac arrhythmia. In my view, clinically they have proven that, even though pathologically I cannot confirm that, but just even looking at the medical notes and the sequence of events, Mr Bayoh has ultimately died of cardiorespiratory arrest, which is a fatal cardiac arrhythmia. What you can say is that with MDMA, it's a stimulant type drug, so it increases heart rate, it increases blood pressure and it can cause cardiac arrhythmias. It can also affect the person's breathing because the heart's not beating properly. So you do not necessarily have a pulse, but your heart function can affect how your lungs are functioning as well.

72. I see cases regularly of people who have died purely from taking MDMA, and their cause of death is MDMA toxicity or ecstasy toxicity. So we know that this drug can kill people. It is not necessarily dose-dependent either. Some drugs, you have to take a reasonable amount of it for it to have any ill effect, but a lot of stimulant drugs like MDMA and cocaine, are not necessarily dose-dependent: in other words, you do not have to take a huge amount of it for it to potentially kill

you. Just having it in your system potentially means that it could have caused your death, but the actual arrhythmia we cannot prove pathologically because it is what is going on in the conduction system.

73. MDMA and Alpha-PVP are stimulant drugs. They have the other effects, including psychomotor effects which could potentially explain Mr Bayoh's behaviour in the lead up to the restraint in terms of him being agitated, delusional, and the behaviour he displayed.

74. I have been asked, if it were the case that a friend of the deceased had taken the same drugs and not suffered ill effects, whether that was relevant. Drugs have different effects on different people. I could take a drug and it could kill me. Another person could take the same drug and be fine. If a friend had taken the same drug as Mr Bayoh and was fine, that does not take away from the fact that Mr Bayoh's taken it and the drugs are in his system and potentially played a role in his death. This is because we all respond to these things differently. One person's response is not the same necessarily as another person's response. Additionally, there may be differences in doses taken or other substances taken. There's the fact that Mr Bayoh's was restrained by the police and the ensuing struggle against that restraint. This is something that, presumably, Mr Bayoh's friend was not exposed to while taking drugs.

75. In relation to the CS Spray and PAVA spray, at page 16, we concluded that these did not appear to have an immediate effect on Mr Bayoh. The report continues "From the literature available, it would appear specific side effects include bronchospasm and laryngospasm and patients with pre-existing respiratory disease (which did not appear to be the case here) are more at risk from severe effects." I have been asked to explain bronchospasm and laryngospasm and their symptoms. They're spasms in the muscles. The larynx, commonly called the voice box, is at back of the throat and bronchial are the lungs. With these sprays or powders they can affect the mucosa, which is the lining of the back of the throat, or the lining of the lungs and it basically causes them to go into spasm. It's almost like what you see in someone who has an asthma attack. With both conditions, individuals can suddenly become wheezy and have difficulty with

breathing and speaking. They may feel like their throat is closing over. Such individuals become very unwell very quickly, and it's very apparent that those effects are happening, which did not seem to be the case here. I have been asked how quickly you would expect to see those effects. This is not my area of expertise; however, my understanding is that the CS Spray and PAVA have very acute effects in that they cause the eyes to be sting and be very itchy and red immediately. The actual bronchospasm and laryngospasm are not something that should happen routinely. That tends to happen in people with pre-existing respiratory and heart conditions, which makes it much more dangerous to use them on someone who has asthma or who has bronchitis. Mr Bayoh did not have any history of any respiratory problems. I don not think it would have been delayed by minutes for him following the spray and only take effect, for example, once Mr Bayoh was on the ground being restrained, then to suddenly become breathless or his airways closing a few minutes down the line when he is on the ground. But I would defer to an appropriate expert on this point. From a pathology point of view, this not part of my remit. This is simply my knowledge from reading the literature provided to me.

76. I have been asked whether sleep apnoea would be included in the definition of a pre-existing respiratory disease in relation to CS spray and PAVA. I do not think so. Again, perhaps this a question for a respiratory physician or expert in relation to the CS/PAVA spray. Sleep apnoea takes place when an individual is asleep. They have episodes where they stop breathing for short periods of time. It tends to happen in overweight people or I presume muscly people as well where they have got quite big chunky necks, but to my knowledge it does not require any treatment as in inhalers or steroids or any actual medication. Treatment is usually with a machine to help with their breathing overnight. Mr Bayoh was an otherwise fit and healthy person who exercised regularly at the gym. He did not have any problems with breathlessness, wheezing, which are the pre-existing issues that I would be worried about. It is these kind of issues the spray would trigger because of its potential effect on the airways or the mucosal lining of the airways, which I do not think is relevant to sleep apnoea. Again, I would defer to the opinion of a respiratory physician or other appropriate expert on this point.

77. I have been asked whether seasonal rhinitis would be included in the definition of a pre-existing respiratory disease in relation to CS spray and PAVA. No, I do not think so.

78. In the report we discuss the issue of excited delirium. At page 16, we state *"Excited delirium syndrome is described as a life threatening condition that has a variety of causes but is largely associated with drug intoxication, in particular stimulant drugs (MDMA and alpha-PVP are both stimulant drugs). It can include paranoid and aggressive behaviour as was reported in this case and has no pathognomonic findings at post mortem."* I have been asked to comment on this. Because of the type of case that this is, as in a death whilst being restrained and someone who has drugs on board, I am obliged to discuss excited delirium. It's not a diagnosis I can make. Excited delirium is a psychiatric diagnosis – I am not a psychiatrist. It is not a pathological diagnosis, but it potentially gives an explanation of behaviour rather than a reason why someone has died, but I am under obligation to discuss it just because of the circumstances surrounding this case and the actual pathological findings, but it is not a diagnosis that I would ever make.

79. At page 17 of the final post mortem report, we discuss the restraint: *"In terms of the history of restraint here, Mr Bayoh was reportedly face down with his hands cuffed in front of him (this is supported by the presence of injury 16), his legs were tied around the knees and ankles and at least four officers were restraining him. Post mortem examination showed the presence of petechial haemorrhages within the eyes and whilst these are not specific and can be seen in someone who has been resuscitated, they could indicate a degree of asphyxia."*

80. I have been asked to comment on this. Given the position Mr Bayoh has been in during the restraint, we are looking for potential asphyxial signs and given the position he was in, we are considering the potential of positional asphyxia in that he is face down, his chest is forward, which can alter how you breathe, so you cannot inhale and exhale the way you would normally because your chest is pressed against something. We also considered the potential of mechanical asphyxia as reportedly during the res[redacted]s back to restraint pressure was applied to his

hold him down. These are a number of factors that can affect breathing and could have been associated with him stopping breathing in the first place. I have been asked whether a combination of positional and mechanical asphyxia might be possible in a restraint situation. Yes, that is possible.

81. Finally, in the report the cause of death is a narrative cause of death: "1a Sudden death in a man intoxicated by MDMA (ecstasy) and alpha-PVP, whilst being restrained". The drugs taken, Alpha PVP and MDMA have cardiac toxic effects. Alpha-PVP is similar to MDMA in that they both cause your heart rate to increase, they cause your blood pressure to go up, and they increase the rate or the force that your heart has to beat. The Alpha-PVP would have worked synergistically with the MDMA with all of that happening to the heart in addition to the acid that's circulating from the restraint and the struggle. All of those factors together would have accentuated each other, and hence why the cause of death is given as a long narrative that encompasses all of those factors.

82. I have been asked about the toxicology report dated 12 June 2015 which is appended to the final post mortem report (pages 23 to 25). The report speaks about examining samples of blood and urine collected at post-mortem and at hospital (ante-mortem blood samples) were analysed for alcohol, prescription drugs, and drugs of abuse. The Alpha-PVP found to be present in the hospital blood at 0.07 mg/L, and in the post-mortem blood it's 0.29 mg/L in one sample and 0.31 mg/L. I have been asked to explain why the Alpha-PVP levels in the post-mortem blood are higher than the hospital blood samples. This is often the case and this can be the problem with post-mortem toxicology. After someone dies, the drugs can redistribute in the post-mortem sample so they can become artificially higher than what they would have been ante-mortem. That is why it can be really difficult sometimes for us to interpret post-mortem toxicology levels because often the levels can be slightly higher than what they are ante-mortem. That is why it is really useful to have ante-mortem blood and why we always try and get, if possible, a hold of ante-mortem samples because they are much more accurate samples to test.

83. Additionally, you get post-mortem production of various things. Some drugs still continue to break down after death; they redistribute through the blood. It depends on you getting a clean amount of blood taken from exactly the right place. All sorts of factors can affect what level you get at post-mortem, hence why the ante-mortem samples are always the gold standard. Things like alcohol are produced post-mortem because that is how the body responds to death; the cells break down and they start producing alcohol. You could actually have no alcohol ante-mortem and a reasonable amount in post-mortem sampling because it will be produced after death. There are a number of factors that can affect post-mortem levels of drugs, which makes post-mortem toxicology really difficult to interpret and it is not an exact science.

84. The toxicology report states in relation to MDMA. At page 2, *"Five adults who succumbed to MDMA overdosage had an average post-mortem blood concentration of 1.8 ranging from 0.6 to 2.8 mg/L."* I have been asked to comment on the fact that Sheku Bayoh's post mortem blood levels were at the lower end of that range. As explained previously, these types of drugs –stimulant type drugs – are not necessarily dose-related and just because you have a very low level does not necessarily mean that it is not going to have adverse effects on you. Additionally, the combination of MDMA being taken with another type of stimulant drug is a significant factor in itself.

85. In relation to Alpha-PVP, the report states *"It is not clear from the literature available what effects would be expected from specific blood concentrations."* In 2015, we knew relatively little about Alpha-PVP. In terms of its toxicity, but we knew it was a kind of synthetic cathinone. We could take our data as to what we knew about that drug class, which is the fact that it can cause euphoria, agitation, and cause arrhythmias. Again, Alpha-PVP being a stimulant drug its effects are not necessarily dose-dependent. So even having a low level does not mean that you are not going to die from it, especially when you start combining this with other stimulant drugs.

86. I have been asked whether there has been any further research since 2015 which provided a fatal range for Alpha-PVP. I have provided copies of the few

case reports that I am aware of in relation to this (WIT-00008, WIT-00009 and WIT-00010).

87. I am asked to confirm that toxicology testing was negative for alcohol. Yes, there was no alcohol found in the post mortem samples and they did alcohol testing on the hospital sample as well. There was no indication that he had taken any alcohol. There was not even any produced post-mortem but that is because we did the post-mortem so quickly after death.

88. I have been asked about the microbiology report which is appended to the final post mortem report at pages 26 and 27. I have been asked about the handwritten notes on the letter. This is my handwriting. This appears to be my notes of my discussion with Dr Robert Weir. At page 26, next to Staphylococcus epidermidis I have written "normally see if shunt etc." Next to the words "Stenotrophomonas maltophilia" I have written "ubiquitous in environment" – I'm unsure what I have written after that. Further down the page I've written, "If no previous intervention i.e. shunts very likely all post-mortem contaminants."

89. Post-mortem microbiology is notoriously difficult because after you die you produce bacteria, and you also have a lot of bacteria, we call commensals, that line various parts of the body; line the mouth, the lungs, the gastrointestinal tract, that can then be picked up when we test when it is actually just normally there. We often consult with the microbiologist, to see what they think in terms of are these bacteria important or not. Nine times out of ten, they are not and are post-mortem related. We came to the conclusion, in this case, that it was post-mortem related, and that was also supported by the fact that neuropathology did not find any infection or inflammation in the brain.

90. On gross examination we did not see any signs of infection. Neither did Prof Smith, who did the neuropathology and he would obviously have looked at the brain as well and he also sampled the brain. If there was any infection on the brain, you would see changes on histology down the microscope, and there was not any.

 

91. In relation to the radiology report, it mentions that *"the levels of decomposition meant that meaningful images could not be obtained and the patient subsequently then attended for a CT examination"*. I am asked whether the degree of decomposition was within normal parameters. Even though they're refrigerated, bodies still can deteriorate. It should not be rapid, because they are refrigerated, but you do expect a degree of deterioration. This is talking about all internal deterioration, so internally it will deteriorate more. That doesn't necessarily mean that externally there's been a deterioration. That is not what the CT report comments on.

92. When we went back to body to take out the rib, I cannot remember thinking he had deteriorated far more than what I would have expected, but it was a long time ago and it was not something I would have made note of at the time. However, this deterioration is not the external body; the deterioration that the CT's talking about is internally. We would expect that to break down, and gas gets produced even though the body is refrigerated, but it is not necessarily talking about what the body looks like or what we would see if were looking externally.

93. I have been asked why the degree of decomposition affects an X-ray? I think they could not see what they needed to see on the X-ray; they could not visualise the spine. They could not get the pine into a position to be able to see it clearly. The cervical spine has seven vertebrae that normally lie in almost a C-shape. So they could not have got it into the right distribution to be able to visualise the bones properly, to be able to make sure that they were definitely not fractured. Decomposition *per se* should not stop any sort of bony X-ray being a problem; it's more the soft tissues that get affected by decomposition. The bones will be there for a long time; they do not tend to break down. It is more the soft tissues that are surrounding them.

94. The difference with a CT scan is that with a CT is you can reconstruct the images; you cannot do that on a bone X-ray. You X-ray the bones and it's like taking a picture. You get a picture, you cannot break that down or rearrange it. Whereas a CT scan takes thousands of very fine photos of the whole body and the radiologist, on a computer, can manipulate that, can build it up, can give you 3D pictures. Bone X-rays are just the plainest, simplest thing that you can do so

if a structure is not in the right position or things are not positioned in the right way, then that is what you are going to get a picture of. You cannot do anything about that. You are not going to be able to reconfigure it or change it, whereas in CT you can. So even him lying in the same position, you could not visualise the C spine on X-ray, but on CT, you can see what they need to see. It's a much more detailed way of doing it. It's three-dimensional image rather than 2D, which is what X-ray is.

95. I have been asked whether there any suggestion that the decomposition impeded the CT examination. I suppose it did because they're not going to see what they would have done normally, but it did not impede things overall because the only reason we were doing it was to get a good look at the area that we had not been able to see on the X-ray. We are using CT more and more, but ultimately the post-mortem is the gold standard. Post-mortem will never be replaced by CT scanning because actually visually looking at something is much more accurate than what a CT scanner gives, especially for smaller structures.

96. Ultimately, albeit what they were looking at some of it had gas formation and other signs of deterioration but we did not need that information because we already had all of that information from the post-mortem. We had seen everything, we had looked at everything closely but what we did not have was a detail of that particular area of the body, and that is what we really wanted and that is what the CT was able to give us that. From that, it obviously picked up on the fracture that we had not seen and the X-ray had not picked up on as well so we were able to then go back to the body to look at that specifically, we would not have been able to do if we did not have the CT.

97. I have been referred to a section in the radiology report (appended to the final post mortem at page 29), under the heading of the cervical spine where it says, *"C7 remains intact and in place. There is significant gas within intervertebral veins throughout the visualised cervical and thoraco lumbar spine."* The cause of the air-gas formation is post-mortem production. We see it even in someone who has been CT scanned the day after they die, you'll still see a degree of gas production in vessels. Lots of post-mortem artifacts are seen in CT, and this is

why we need radiologists who have expertise in post-mortem CT scanning. Post-mortem CT scanning is completely different to antemortem CT because the changes that take place immediately after death will affect what pictures they take, and so it has to be interpreted with that in mind.

98. This section continues: *"However, there is particularly linear distribution of air within C7 extending from the vertical body to the posterior elements bilaterally. Although this may represent artefact, given the rib findings detailed below, direct visualisation is advised."* Direct visualisation is us looking at this part of the body again, which is what we did. When we went back to the body to look at ribs one and two, we also looked at the seventh cervical vertebra, and it was uninjured. There were no abnormalities so I think it was just artefact that they were seeing on the CT. That was one of the reasons that we went back to the body to look.

99. The next paragraph states *"there is a well-defined, linear lucency in the medial, posterior aspect of the left 1st rib proximal to its junction with the 1st thoracic vertebral body."* I have been asked about the significance of the "well-defined linear lucency". That's what was potentially a fracture. When we went back, that's what we thought possibly was a fracture and then took the histology and confirmed it was a fracture.

100. I have been asked about the post-mortem chest X-ray, as this is not mentioned in the post-mortem report. Was it taken before or after autopsy, and by whom? This was the first skeletal survey following post mortem on 13 May 2015. This report is appended to the final post mortem report at page 28. Part of the skeletal survey will be looking at ribs. It has not been referenced on the actual report. I have been asked about the fact that this X-ray did not show up an obvious fracture. I think it just shows that the rib fracture was difficult to find and small, so not picked up on the plain film. It shows it was not an extensive fracture; it was not displaced, or fractured in more than one place, so it was a relatively minor fracture.

101. The neuropathology report by Professor Colin Smith is appended to the final post mortem report at page 20. It is dated 20 May 2015. The report states *"Neuropathological examination has demonstrated changes consistent with an evolving global ischemic brain injury. There is no evidence of significant traumatic injury to this brain and no infectious disease such as meningitis or encephalitis. No natural disease is noted to account for death. The changes all appear secondary to cardiac arrest with resuscitation and short survival period."* I have been asked when these changes in the brain would commence. They can start within several minutes. Professor Smith would have had information about Mr Bayoh going into cardiac arrest and the transfer to hospital and the on-going resuscitation are all in keeping with that kind of timescale.

102. What happens is Mr Bayoh is in cardiac arrest, so the brain is not being perfused but resuscitation is trying to push oxygenated blood to the brain. The brain is being starved of oxygen for that period of time, and that's when it's becomes ischaemic, because often when people die very quickly, if they are in cardiac arrest and they are maybe only resuscitated for a few minutes or if they are not resuscitated, you will not see any changes in the brain. But because with him he had on-going resuscitation for a significant period of time, so they have continued to try and pump oxygenated blood to the brain and that is why there has been time for those hypoxic changes to develop. It can take a few minutes up to several minutes for him to start seeing things down a microscope, but as Prof Smith says it's very, very early changes, and would all be keeping with the cardiac arrest, resuscitation, and the shortness of viable period. However, Professor Smith would be best placed to give the specifics because the research and the data changes reasonably frequently on this topic.

 

Forensic Strategy Meeting – 12 May

103. I have been asked about the forensic strategy meeting that took place on 12 May 2015. I have seen the minutes of this meeting (PIRC-04161). At page one, I see that I give an update on the cause of death which was that it was unascertained pending further tests and examinations being undertaken. At page two, I am asked about the following paragraph: *"PIRC to provide scientific research papers to Dr Shearer regarding the effects of CS/PAVA spray. PIRC to prepare and submit a package to Dr Shearer with: GP medical records, Hospital medical records, statements from hospital staff, statements from witnesses to the deceased behaviour and actions in the lead up to his arrest and witnesses to the arrest, CCTV compilation and photographs."* I am asked whether I received this package. The final post mortem report makes reference to the GP records, hospitals records and witness statements so I had these. I am sure I saw CCTV – I believe that someone from PIRC came into the office and showed this to me.

104. I am asked whether the scientific papers were of any significance to my findings. They were. I have another folder in my office with all of the scientific papers to do with this case and they were useful. The papers were not anything I could not access on my own, but they were useful papers to read. I think obviously the reports were that both sprays had been used, and I was going to have to try and make some comment as to whether I thought it played any role in his death. PIRC were obviously researching it and passed on whatever information that they had found about it. I think they were just trying to be helpful and what they provided was very useful.

105. I have seen the letter of Dr C Walker dated (COPFS-02380) dated 23 June 2015 regarding analysis of post mortem urine from Mr Bayoh. I cannot remember seeing this letter. My reading of the letter is that Dr Walker is not implicating the steroids in death in any way. I completely agree with this. We did not implicate steroids in the cause of death because, to my knowledge and my reading, the acute use of steroids does not kill you *per se*, it tends to be the long-term use can have cardiac effects and cause problems with the heart. This was another one of the reasons that we did such a detailed examination of the heart, and we could not see anything that would say that he had any long-term effects from that.

 [redacted]

Supplementary Post Mortem Report – Caffeine

106. I have been asked about the supplementary post mortem report dated 5 November 2015 (COPFS-05138). This report was needed as tablets were found at the scene. The tablets were analysed and some of them were caffeine or perhaps the tablets said caffeine on it, I cannot quite remember. Caffeine is not a drug that we would routinely ask for in toxicology; I have never tested for caffeine before in any cases, so it was just to make sure that we had definitely checked for that and that was not another factor in his death. I asked the lab to check the post-mortem blood and hospital samples for caffeine to see if he had taken an excess as another potential reason that he had a cardiac arrest. However, looking at the data, reasonably low levels of caffeine have been detected; certainly nothing up to what has been seen in papers that look at fatal cases, so we discounted that as being a potential factor in his death.

Supplementary post mortem report – sickle cell trait

107. I have been asked about the supplementary post mortem report dated 3 October 2017 (COPFS-00040). This following on from the positive test result obtain that Mr Bayoh was a carrier of sickle cell trait. I have been asked what experience I have of sickle cell disease or sickle cell trait. Absolutely none whatsoever. I was a medic for clinical medicine for a number of years before I went to pathology and I think I maybe saw one or two cases of people with sickle cell who came into hospital acutely unwell – not with the trait, with the actual disease – but I've never come across it at all in forensic practice. This is all new, definitely I have no experience of it whatsoever.

108. I have been asked who requested the sickle cell testing. It was Dr Elizabeth Soilleux. I was given her initial report suggesting that testing should be carried out. It is not something that we at all considered at post-mortem, as far as I remember, and we probably should have done to be fair. This case was reviewed by all of my colleagues. In addition to Nat Cary and other forensic pathologists, nobody thought about sickle cell. With his ethnic origin, we probably should have. It was a learning point for all of us and we will certainly be

thinking about it in the future. Dr Soilleux suggested the testing and we had taken genetic samples at post-mortem, so we did the testing and he tested positive for sickle cell trait rather than the disease itself.

109. I have been asked to comment on the significance of Sheku Bayoh being a carrier of sickle cell from the point of view of cause of death. I have now gone and now done a great deal of reading on this topic. Sickle cell trait, for the vast majority of people, would seem to be something that they never know that they have or if they do have, they do not have any ill effects from it whatsoever. There are papers, and I think Dr Soilleux references some papers that discuss people who are carriers of sickle cell disease who are under extremes of stress. For example, there are papers about people in the army in the desert, exercising and dehydrating in high temperatures. There are a few papers about people under anaesthesia. It seems to be profound states of hypoxia that can cause sickling in people with sickle cell trait. As such, looking at this case, given everything else that is noted in terms of the drug intoxication, the struggling against restraint and the restraint you can see why in that whole situation he would be hypoxic to a degree and as such sickling could be occurring.

110. I have no experience in looking at sickle cells down the microscope. Dr Soilleux comments on there being lots of sickle cells on histology. Then Professor Lucas is asked to comment on this specifically, and he has a vast amount of experience in sickle cell. However, the difficulty is that you can get sickling post-mortem: your normal cells can sickle post-mortem because there is a degree of hypoxia so it is a difficult one. I think it is really useful to know about it and I think absolutely we have to consider it being somewhere in the cause of death. However, reading Dr Soilleux's report (and perhaps this is just my interpretation) she seems to put a huge amount of weight on it.

111. I agree that it is something that we should definitely consider as being in the cause of death somewhere. But I think the bottom line is that if he has suffered a degree of sickling due to hypoxia, that has happened because (1) he's taken drugs, (2) he's been restrained, (3) he's struggling, so the sickle cell albeit may be involved in the cause of death but in my mind it's not the most major factor;

 [redacted]

there are much bigger factors there and the only reason that it may come in to play at all is because all of those other factors have happened, because without those factors, in my opinion, he would not have died. If you take sickle cell out of the equation, would he still have died? Yes, because he has drugs on board, because of the restraint, the potential asphyxia and all of the other's factors. If you took those factors out, and you just had the sickle cell, would he have died? Absolutely not. It's a bolt on factor that may have had a role to play but certainly is not the main factor in his death.

112. Having read the reports of Dr Soilleux and Professor Lucas', it's difficult to tell from their reports how much weight they are putting on the sickle cell trait. In my mind the causes of death and how I report and how my colleagues' reports are that we tend to put the most important thing first. Sometimes it's difficult to do because all factors are important. In this case, it is difficult, there are lots of important factors so one thing cannot come first. But they have both put the sickle cell right at the beginning of the cause of death so I do not know if that means in their mind that it is the most important factor. It is something that requires to be clarified. I would have it in the cause of death, maybe not under Part one, maybe under Part two, as a potential factor that could have played a role. Or perhaps under Part one, adding to the narrative and adding in "A man with sickle cell" as well at the end. Perhaps something along the lines of "Sudden death in a man intoxicated by MDMA (ecstasy) and alpha-PVP, whilst being restrained, who had sickle cell trait". But I think we have to bear in mind that, the sickle cell trait would not have had any role to play had he not had all of the other factors causing a hypoxic state that ultimately ended up in him dying.

113. My other comment on the reports would simply be that Professor Lucas is not a forensic pathologist. Consequently, he, quite rightly, does not comment on the restraint part of things. In relation to Dr Soilleux, she is obviously a very experienced pathologist. The pickup was excellent in giving us that diagnosis of the sickle cell trait. However, I am not aware that she is a forensic pathologist. If she is, I would be interested to know how much weight, in terms of where either or both of them would put that. Did they weight it the same as the restraint and did they weight it the same as the drugs? Or do they think it's more important

Signature of Witness .....[redacted]

than the restraint or the drugs? I cannot tell that from the reports, but it would be useful to clarify that.

114. I have been referred to a DNA diagnostic test result following a referral for a sickle cell disease and thalassaemia (COPFS-03590(a)). The report states *"The presence of the heterozygous pathogenic HBB variant, c.20A>T, p.(Glu7Val) was confirmed. No other pathogenic variants were identified in the HBB gene. MLPA analysis of the HBA and HBB genes repeatedly failed to give interpretable results due to the low quality of the DNA sample, therefore full thalassaemia screen is not possible. SHEKU AHMED TEJAN BAYOH was a carrier of sickle cell disease."*

115. I have also been referred to an email dated 5 June 2018 in which I send this report onto COPFS and say: "They appear to have managed to complete the vast majority of the screening and this was negative. Although, we are unable to complete a small proportion so could not definitively say everything was negative."

116. I have been asked to explain this. I'm just talking about the full thalassaemia screen that was not possible. One of the problems are submitting post-mortem tissue, albeit this is probably the best-case scenario because this tissue was taken so quickly after death, but it is not the same as being able to take somebody to the hospital and taking a blood sample from them. There is going to be some degeneration in the tissues, so they are not always able to complete all of the tests that they can do. As much as we try to take the tissue as quickly as possible and get it frozen to stop it from breaking down, there is still a process whereby it gets taken at post-mortem, it gets refrigerated, transferred to the Royal Hospital and then it gets frozen. So there's still a small lag that gives a chance for tissue to break down but these are the kind of limitations that we have in managing these post-mortem samples, unfortunately.

117. The email was just to make the COPFS aware that we have tried, and checked the vast majority of things but there were some tests that could not be completed. The bottom line is that he was a sickle cell trait carrier,

Signature of Witness [redacted] that he was a sickle cell trait

which is what we were looking for in the first place. The thalassaemia was more of a side line because it was seeing the sickling of cells and noting his ethnic origin coming from Sierra Leone that put him in the category of being at a much higher risk of having sickle cell.

118. I have been asked to explain Thalassaemia. Thalassaemia is an inherited blood disorder. It's increased in specific ethnic origins. Essentially, the body does not make enough of haemoglobin so it causes anaemia. There was nothing in Mr Bayoh's background to suggest that he had anything like that.

119. I have been asked whether there is any additional testing that could have been carried out if the sample had been of better quality. I think they may have got a full thalassaemia screening done, presumably if it would have been better quality and then we would have known if that would definitely have been negative, but there was nothing to suggest that he had thalassaemia and the main reason for doing the genetic testing was for sickle cell.

120. I am asked to clarify whether the fact the sample was of low quality had any impact on the finding that Sheku Bayoh had sickle cell trait. No because they were able to get a genetic mapping for that so it did not matter.

Report of Dr Steven Karch

121. I have had sight of the report of Dr Steven Karch (PIRC-02526(a). I have been asked to comment on the following paragraph, at page 4, *"High doses of nandrolone elicit cardiotoxic effects including cardiac remodeling and injury. There is also laboratory evidence that they m[a]y provoke arrhythmias. As myocardial remodeling of both ventricles was apparent on my examination of the heart, it seems only reasonable to conclude that nandrolone contributed to the process, as did all of the other stimulant drugs. There is also evidence that, by methods yet to be determined, nandrolone facilitates the occurrence of myocardial arrhythmias, the apparent cause of Mr. Bayoh's demise"*

122. I completely disagree with what he says, we did not see what he was seeing in the heart. The heart was completely normal histologically. This case was seen

by myself and Dr BouHaider, every other colleague in my department, Dr Nat Cary, Professor Jack Crane and Mary Sheppard, who's a professor in cardiac pathology. They all stated the heart was normal. I assume he was saying the nandrolone is playing a role because he was seeing chronic changes in the heart. There was nothing to suggest that the nandrolone has had anything to do with this man's death so I'd completely disagree with what Dr Karch says.

123. I am asked to comment on Dr Karch's suggestion that there's laboratory evidence that nandrolone may provoke arrhythmias. Yes that is correct in situations where nandrolone has caused chronic damage to the heart. There's no evidence to suggest that nandrolone can cause arrhythmias without that damage to the heart having been created. Normally it does its damage through chronic toxicity which there was no evidence of in this case.

124. I am asked what my understanding is of the term myocardial remodelling. At page 4 of his report, Dr Karch explains that *"myocardial remodelling is the term generally used to describe changes in the size, shape and structure of the heart after it has been injured."* Myocardial remodelling is possibly an American term, it is not a term that I would use. The myocytes are the heart cells so that is where the word myocardial comes from. Myocytes are the tiny muscle cells that make up the heart, they are like little rectangles with a little nucleus in the middle. When the heart is insulted or damaged, like any part of the body, you get an inflammatory response and ultimately it heals by either the myocytes becoming much larger and the nuclei becoming irregular and that's called myocyte hypertrophy and you see that in people with conditions including chronic hypertension, (high blood pressure), valvular heart problems and obesity. In addition, you get scarring, so you get the myocytes replaced by scar tissue, which is probably the remodelling that he is talking about.

125. Events like a heart attack can cause remodelling and you will get damage to the heart and scarring that we will be able to then see at post-mortem. Chronic use of stimulant drugs can cause scarring in the heart muscle because at some point the heart cells become hypoxic; they're not getting the circulation that it requires so little bits of them die off and cause scarring. I see it frequently in

cause scarring. I see it frequently

drug related deaths, especially stimulant deaths where you can have chronic damage from chronic drug use, but there was nothing like that in Mr Bayoh's heart. His heart looked completely normal. There was nothing to suggest any past insults and as such, I do not understand his conclusions.

126. In Dr Karch's report at page 6, he is asked the question what is the physiological effect of the restraint of the deceased (i.e Sheku Bayoh) in the circumstances of his arrest? His answer is that *"Given the details of this situation, the effect of physical restraint would have been de minimis."* He further considers that the restrain is irrelevant when considered in combination with the drugs and CS spray/PAVA. I am asked to comment on this. Pathologically, I cannot say whether the restraint used was adequate or excessive. However, at the end of the day, he was restrained. He was in a position that would have hampered his breathing, hampered his heart, and was intoxicated with drugs . As such, there was a combination of factors that all would have interplayed with each other, and it was difficult to say what has been the more important factor. I think all of them together is important. They have all happened together and resulted in his death. It would be interesting to know what his actual qualifications are and background to be able to say such things.

127. At page 2 of his report, Dr Karch states *"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."* At page 7 and 8, Dr Karch states *"The inherent cardiotoxicity of these drugs, together with obvious pre-existing heart disease, just makes the probability of cardiac arrests even greater".* So essentially, it seems to conclude that the cause of death is due to taking drugs and heart disease. As discussed, the heart was completely normal. I do not consider that Sheku Bayoh had heart disease. As discussed, I accept that drugs may have played a part in his death.

128. I have had sight of a consultation note dated 4 June 2018 (COPFS-04194(a)). It is a consultation attended by, Ashley Edwards QC, Alistair MacLeod from COPFS and myself. I don not remember this consultation. However, I am content to accept that I attended the meeting.

129. The opening paragraph of the note states "*In the main the expert medical witness reports I have seen are broadly in agreement. Dr Karch appears to be out there on his own with his finding of histological abnormalities in the heart. To the best of my knowledge I thought he was a toxicologist and am not sure if pathology is his area of expertise. I was however reassured with the opinions of Professor Sheppard and Dr Cary.* The note suggests that I may have read these reports of this experts either before or during the consultation. I have no memory of this now.

130. The note continues *"In a classic case of mechanical asphyxia, you would expect to see more florid asphyxial signs, including more pronounced petechial haemorrhages. You can also see soft tissue and bony injury of the chest, which was not present here. The deceased had bruising on his back and lacerations in his mouth. That is not to say there was no degree of mechanical asphyxia, but that there were no classic signs. The deceased was resuscitated to a considerable period of time, and this can cause petechial haemorrhages. There is nothing definitive to say that asphyxia occurred here."* I have been asked to comment on this. Petechial haemorrhages are very nonspecific. I see them a lot; I see them every day in the Post Mortem room - a lot of them associated with resuscitation. They are caused when tiny blood vessels pop due to an increase in pressure. When I do see them in cases of asphyxia, some external compression of the neck or mechanical asphyxia, they do they do tend to be more florid, like I have said. You do tend to see them in different places. You tend to see them maybe on the outside of the eyes and the mouth, behind the ears and, especially in cases of mechanical asphyxia, the face may be a bit more congested. However, because of the colour of Mr Bayoh's skin, you would not necessarily see the same degree of congestion as that on a Caucasian person and they would be much redder looking.

131. It is one of those things that are not specific and you have to take them in conjunction with everything else you are finding and with all the other information you are given. At the end of the day, he was face down with officers restraining him. As such he may have had reasons for having both positional and

 [redacted]

mechanical asphyxia in that scenario and haemorrhages would fit with that, but he also was extensively resuscitated.

132. So it is difficult to be specific. It could be either or it could be a combination of both, and that is another reason that the cause of death is a narrative – taking everything into consideration – because it is really difficult from a pathology point of view. Pathologically, I cannot prove asphyxia, I can just surmise that this could have happened, or this may have happened in conjunction with other things.

133. In the consultation note, I'm recorded as saying *"The deceased was pushing up an officer in a press up motion. This shows he was moving and particularly moving his chest muscles. With such use of his muscles, this could have increased his heart rate and blood pressure and been associated with acid production. Being of large build with a lot of muscle bulk, acids could have been produced and the act of struggling would have increased the risk of an arrhythmia."* This is the aspect of the struggling against restraint. He has being restrained, but he was also struggling against the restraint so it is almost like double the effects on his body and what his muscles are having to do. He is pushing against whatever is on him, but he is also using his muscles to try and push against these things increasing the chances of acid being produced.

134. The consultation note continues *"I am asked about the deceased's behaviour beforehand particularly the fact he was running around, striking at cars and wearing minimal clothing. The deceased's heart rate would be up, his blood pressure would be up, he would have been using his muscles and therefore acids could have been accumulating in his bloodstream. Add in stimulant drugs and you can affect the heart rhythm further, increase blood pressure and heart rate and increase the risk of cardiac arrhythmias."*

135. Again, it is excessive exercise. It is excessive use of muscles so there is a chance that in that activity – even before the police arrive – that he has already started some degree of muscle breakdown because he has got drugs on board as well. So already his heart rate is up, blood pressure is up and his muscles may have already started to produce lactic acid before he even comes to the

altercation, then add in the struggling and the restraint and all of it together in combination ultimately led to him dying. So yes, it is important to consider what he has done beforehand as well.

136. I think that also comes in with the sickling. If it is prolonged over a longer period of time, you have got more chance of him being more hypoxic, and more chance of them sickling as well, which means that the quality of the blood that is reaching the organs is not ideal because it is not the correct shape for carrying the oxygen that is required. That is basically what happens in sickling: the cells become a funny shape so they cannot bind oxygen the way cells normally would, and it means you are getting hypoxic tissues, or hypoxic blood being taken to tissues, which just adds to the hypoxia.

137. I have been asked if there is any data available regarding the number of people who die from drugs toxicity alone – particularly in relation to the use of stimulants such as of MDMA and Alpha-PVP. One difficulty related to this is that a lot of the time you get people who have taken multiple drugs like in this case and if they have taken multiple drugs, the cause of death will be multiple drugs. The other problem that we have is, especially in the cohort of people that we get for post-mortem, a lot of them who have taken stimulant drugs have also taken things like heroin and methadone. A lot of our drug deaths are multi-drug toxicity. We do get the odd cocaine death and MDMA death, but a lot of them tend to take other substances at the same time.

Professor Freemont's report

138. I have had sight of Professor Anthony Freemont's report dated 3 July 2017 (COPFS-00037). I am referred to the paragraph in the consultation note (COPFS-04194(a)) in which I discuss his report *"Professor Freemont has timed the fracture to have occurred at least 2 hours before death. I have seen first rib fractures but they are not usually so posterior. They are rare but can happen. It potentially could have been caused by such a blow to the front of the shoulder however Professor Freemont is very specific. You have an opinion from a respected professor of bone pathology I cannot argue with his findings. Professor*

Freemont' is much respected in his field and his report appears well referenced." I am asked to comment on this.

139. Professor Freemont is very, very specific in his report. I think it would be very useful for you to get a second bone opinion, but I cannot dispute what he is saying. He is an expert in his field. I very rarely take bone histology to look at down the microscope and, if I do take it, I do tend to send it off to an expert to have a look at if I want any kind of specific comment. So I would not dispute what he is saying.

140. I have been asked about the special stains that Professor Freemont requested be applied to the rib tissue samples. He discusses at page 6 of the report that he requested "Martius Scarlet Blue (MSB), Periodic Acid Schiff (PAS), Elastic van Giesen (EVG) and Glycophorin A". I have been asked about the use of these stains. Most of these stains, I would have never been involved in requesting before. Professor Freemont explains in his report at page 7 that *"EVG, PAS, MSB - These stains allow the nature of necrotic tissue to be probed. They were specifically deployed to allow the amorphous red material described above to be analysed."* At the same page, the report states that Glycophorin A *"recognises a molecule on red blood cell walls".*

141. Essentially, they are all for him to have a better look at the tissue and we use special stains reasonably frequently. I use special stains on post-mortems all the time because it means that I can look at particular parts of the tissue more clearly, or if I am looking for specific things in the tissues, the special stains will bring that out and make it easier to be seen. They are a reasonably common thing that we use frequently in pathology. As it is post mortem the tissues have begun to break down, he is using the special stains to see if you can see things clearly and to see if he can see things that are maybe being masked or hidden because of the decomposed nature of the tissue, and the glycophorin A recognises a molecule in red blood cells so he is looking for red cells, which is the reason I did the iron stain. The iron stain is a special stain as well. I had to ask for that separately, and I did that to see if I could see any red blood cells,

which I could not. He has done this specific stain that presumably is what his lab uses to see if he can see haemorrhage at a fracture site.

Dr Cary's Report – restraint and cause of death

142. At page 6 of Dr Cary's report (COPFS-00196) he states with regard to the cause of death: *"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."*

143. I have been asked whether I agree with this statement. Yes, definitely. I am asked whether Sheku Bayoh was at particular risk of metabolic disturbance and the development of metabolic acidosis due to his muscular build. I do not know if having more muscle bulk increases the chance of your breakdown to lactic acid. Common sense would say it probably does, but pathophysiologically I do not know if that would be the case. Perhaps a clinician would probably have to answer that absolutely categorically.

144. Continuing at page 6 of Dr Cary's report, he states "*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*." I completely agree, and I have actually now used that in future cases that I have been involved in deaths in custody with restraint and struggling. So absolutely, the struggling in itself, I suppose, you could liken it to something like intense exercise. The body is continually moving. The muscles are continually moving, so you are getting a production of acid in your muscles. You are getting lactic acid production, and

 [redacted]

when your body has an excess of acid it can cause adverse effects on your heart because it alters the conduction system of the heart and can cause arrhythmias. As such, if you have got that in addition to the heart already being under stress due to drug toxicity that is increasing the heart rate and blood pressure, altering the contraction of the heart so if you have already got that in the first place in addition to adding another factor into the mix that is negatively affecting the heart, then absolutely, they can work in combination. As such, the struggling could have played a significant role as well, not just the restraint, the actual movement against the restraint and even any intense exercise beforehand.

145. I have had sight of Professor Jack Crane's report (COPFS-05194). Professor Crane states at page 7: *"The role of restraint is invariably problematical, as (1) restraint restricting breathing may not be associated with diagnostic signs at autopsy, and (2) petechial haemorrhages may indicate asphyxia but can also occur following CPR."* I have been asked to comment on this. I am fully in agreement with this. I have made these points in the final post mortem report. Petechial haemorrhages are not pathognomonic that someone has been asphyxiated. There are other causes and one of the main causes that we see is resuscitation, so you cannot say definitively that the petechial haemorrhages that Mr Bayoh had are due to the restraint.

146. Professor Crane's report continues to discuss restraint at page 7 and states: *"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 e.g. 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 causing hypoxia would have rendered an unstable myocardium 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 cardio-respiratory arrest, restraint of the type described above was taking place."* I have been asked if I would agree with Professor Crane's conclusion on this scenario. Yes, it seems perfectly reasonable.

 [redacted]

Professor Lucas' report

147. I have had sight of Professor Sebastian Lucas's report (COPFS-00084). I am referred to page 2 and 3 of his report:

"There is no doubt that the vast majority of persons with HbAS who undergo life's daily stresses do not suffer any such syndrome. The critical aspects are therefore the level of stress and accompanying elements such as dehydration, drugs, alcohol intake, muscle activity and body temperature.

In evaluating the clinical pathology, the critical aspects are the amount of sicking of red cells and in how many critical organs. There is no rigid morphological case definition – we must acknowledge – that separates harmless sickling from harmful sickling: it is inevitably somewhat subjective, and informed by the observer's previous experience. And it must be acknowledged that changes in the body's tissue post-mortem can contribute to sickling of red cells. But the quantity of sickling here tells me that this is much more than just post-mortem sickling; it happened peri-mortem as part of the death processes.

In the BAYOH case, I am impressed by the quantity of sickling in the organs such as the heart, kidneys, liver, thyroid and adrenal – much more than I expect to see in the organs of those with HbAS who died of unrelated causes"

148. Continuing at page 3 of his report, Professor Lucas states an alternative cause of death of *"1a. sudden cardio-pulmonary failure 1b. sickle cell trait, recreational drug use, struggle against restraint ".* In relation to the three factors of sickle cell trait, recreational drug use, and struggle against restraint, he comments that *"I do not think we can quantify the contribution of the three factors and state with rigor that one is more or less important than the others. It is multifactorial."* I am asked to comment on this. This is a really difficult one and it is not something I have a huge amount of experience in. We do not see it. I've never seen it before. It is not something that we thought about when we were doing the initial post-mortem and putting out the report. It has come further on down the line, when an expert has been asked to look at it has then picked up on

that as a side-line and then the testing has been carried out. My understanding of sickle cell trait was that individuals with the trait tend to be completely symptomatic, but in stressful situations, extreme hypoxia, you can see sickling. My understanding of it is that when you look at post-mortem histology, a lot of things are distorted. It is very difficult to be sure that you are seeing sickle cells due to say sickle cell trait. This is because you will see sickle cells in people who do not have sickle cell at post-mortem, because the cells can alter their shape and things after death. My understanding is t is very difficult to be certain.

149. I do not have Prof Lucas' experience of looking at post-mortem histology in people with sickle cell because it is not something we come across in Scotland. Again, it is related to ethnicity. I think people who work in London probably do quite a lot of these post-mortems with people with sickle cell trait whereas in Scotland it is not so common. I have now done quite a lot of reading around it, and as previously discussed I would not give sickle cell trait the same weight that Professor Lucas appears to in his report. I think it probably should be in there somewhere however, I think putting it in there in 1b with the restraint and the drugs, when he has not been diagnosed, has never had an issue before, and we really do not really know the extent of it, is giving it a bigger role than it deserves, but I fully appreciate that I do not have experience of this condition.

150. I have been asked to explain what type of matters are normally put in part 2 of a death certificate. It is normally things that are potential factors, but not necessarily directly related. Potential contributing factors is the best way of describing it. They potentially may have played a role in death but are not as important – or we don't think are as important – as things that we've put in part 1. Those are the really important things that we put in part 1. If I was going to change the cause of death, even if it was going to be in part 1, it would probably be with a narrative, as in what we have, but "in a man with sickle cell trait.So often with part 1 you think, if you take that out would the person still have died? With this case it's multifactorial – could Sheku Bayoh have died just from the restraint? Potentially, yes. Could Sheku Bayoh have died potentially just because of drugs? Yes. Could Sheku Bayoh have died just because he had sickle cell trait? No.

 [redacted]

151. At the end of the day, Mr Bayoh was restrained and he had drugs onboard, so I cannot separate these because they are two major factors, and we will never know if one could have killed him on their own because they both happened together, but with sickle cell trait, it was very unlikely that he was going to die of that. He was probably never going to know he had it unless he became extremely unwell in the future. We have information in the literature that says that in extreme situations death can occur death can occur, and obviously Sebastian Lucas is saying he has seen restraint deaths with sickle cell, and I have not. Again, you have to look at those individual cases, and I am sure he would agree that without the restraint those people would not have died of sickle cell trait. If you take sickle cell trait out of the equation, is he saying that those people would not have died of restraint? I would imagine possibly not. It is a side factor and it is not the most important factor.

152. Hence why I think when you are putting it into part 1 probably gives it more emphasis than it deserves but, again, as I've said, I fully accept that I do not have experience in this condition. I know what I have read and what I know about the case in general.

Dr Soilleux's first report

153. I have had sight of Dr Elizabeth Soilleux's report dated 14 February 2016 (COPFS-00031). I have been referred to the report at page 29 where she gives consider to the question of whether Mr Bayoh had a heart abnormality. She 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 preexisting cardiac pathology is unlikely to have contributed to death."* So, she comes to the same conclusion as we did in relation to the heart histology, essentially that the heart was morphologically normal.

154. I have been referred to Dr Soilleux's summary at page 33, where she states: *"It is most likely that the death occurred due to a combination of restraint and the*

presence of significant levels of the drugs MDMA and alpha-PVP. The process of restraint may have caused physiological stress in two ways, one or both of which may be relevant. Restraint may have led directly to asphyxia (of either positional or mechanical type) or may have precipitated an abnormal heart rhythm, as a consequence of the very significant self-induced physiological stress due to the struggle put up by the deceased (e.g., doing a "bench press" and lifting up a 25 stone police office). MDMA and alpha-PVP would have significantly increased the risk of an abnormal heart rhythm developing under conditions of physiological stress." My understanding is that Dr Soilleux is a general pathologist not a forensic pathologist. I am not sure if she undertakes restraint cases and drug deaths. However, I completely agree with what she says here.

Dr Soilleux's Supplementary Report

155. I have been referred to Dr Elizabeth Soilleux's supplementary report, dated 10 May 2018 (COPFS-00039). I understand that this report was produced following testing that revealed that had sickle cell trait. AT page 11 of the report, Dr Soilleux states *""Sickle cell trait gives a very coherent explanation for the sudden death. It is therefore extremely unlikely, although not completely impossible, that a channelopathy affected the cause of death."* I have been asked to comment on this.

156. I would agree that it's highly unlikely, given everything else, that he had a channelopathy. We normally look at channelopathies in negative post-mortems. Young people die suddenly and their post-mortem is completely negative, their toxicology is completely negative, and we do not have another reason that they have suddenly died. Within that, there is going to be a tiny proportion that have taken drugs and died, and may also have a channelopathy, but we cannot check everybody's genetics. We have so many reasons that this man has died, it is highly unlikely, but not something that you can categorically exclude even with genetic testing, because you can genetically test people, but there are not genetic tests available for every kind of underlying heart arrhythmia that can kill you. It's not exhaustive and we can only do what we can do based on the information we have.

157. People can be completely asymptomatic and then suddenly it just hits, and that is why you tend to find absolutely nothing at post-mortem in these people. They tend to be completely well otherwise, and something has just happened on that day: they have gone into an arrhythmia that was not sustainable with life but there has been nobody to immediately resuscitate them. It is possible, even in young, fit, healthy individuals, that they have some sort of underlying cardiac abnormality and they get to a reasonable level of fitness, and nobody knows about it, and they just drop down dead one day. So that is a possibility. It's tiny numbers, but because we see it in the media, it sounds as if it Is really common. It Is not common, but it Is possible.

158. At page 12, Dr Soilleux revises her opinion in relation to the likelihood of positional asphyxiation contributing to cause of death. She states *"On balance, I think that positional asphyxia was unlikely, due (a) to the short time period between Mr Bayoh's contact with police and the commencement of his unconsciousness and (b) the fact that sufficient other contributing factors to death were known to be at play, for death to have occurred as a consequence of these contributing factors in the absence of any positional asphyxia."* I have been asked to comment on this.

159. I would completely disagree. You cannot take positional asphyxia out of the equation, especially when you have it in the context of all the other things that are going on. So, it might require less to be in that position for a reduced period of time because your heart is already under strain due the drugs taken, and because you've already run around for a period of time and got a build-up of lactic acid. This is a multifactorial death, and we do not know what the main factor has been. I think all of the forensic pathologists that have been involved in this case would completely agree with that and have all completely agreed with that.

160. At page 13 of her report she says that she would now *"categorically favour the first of the two scenarios in paragraph 59 of my original report, with the additional contributing factor of sickle cell trait, as described in paragraphs 11 - 12 above and in figure 1."* The scenario she is referring to is that *"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 (e.g., the "bench press" described; see paragraph 58) would very significantly increase the risk of a rhythm abnormality developing, which may well be what happened in this case." My comment on this is really the same as my response to Professor Lucas' report. Dr Soilleux is putting a lot more emphasis on the sickle cell trait than I think is justifiable. I think the sickle cell trait may have played a part but it is certainly not up there with drugs and restraint for the various reasons I have already outlined. The reference to restraint encompasses the struggle against the restraint, potential asphyxia and other factors, such as those outlined by Dr Cary, including the build-up of the lactic acidosis. I think that all comes under the restraint as well.

Professor Sheppard's report

161. I have had sight of Professor Mary Sheppard's report (COPFS-00027), dated 1 December 2015. At page 4, in summary, she states *"The heart is morphologically normal. There is no evidence of damage in the right or left ventricle which would indicate 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 of 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 agree with this. It is fully in keeping with our own findings.

162. At page 5, she states in answer to the question of the physiological effect of a, b, c (i.e. the drugs, the CS/PAVA spray and the physical restraint) on the deceased in in combination in the circumstances of his arrest *"The combination of a, b, c 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."* Again, I agree with this and do not have anything to add.

 

163. Continuing at page 5, in answer to the question of whether Mr Bayoh had a cardiac abnormality, She states "*The deceased had no cardiac abnormality identified at his death. However, this does not rule out sudden cardiac death due to an electrical abnormality of the cardiac channelopathies."* I have been asked whether think there would be worth undertaking further testing for cardiac channelopathies. No, because it's like looking for a needle in a haystack. They can do a whole set of genes, even if those genes are negative, it's not going to change the cause of death. Even if he has a channelopathy, we have the scenario involving the restraint and the drugs, and that is still the reason he has died. A bit like the sickle cell, in some ways, the only positive thing that would come out of it is if he does have a channelopathy, then it's normally inherited. However, if we do it for this case, then why have not we done it for the thousands of other deaths that we do not think it has been appropriate for?

164. II can do it if I am instructed to do it, but I do not think it needs to be done, and I do not think it is probably appropriate to do it to tick another box, because we are not doing that for every other post-mortem. We are not doing that for our young drug deaths who are coming in and we do the post-mortem. They die of drugs. We are not doing genetics on all of them. We need to consider where do we draw the line?

165. I'm not convinced ethically that it should be done because it's not something that we would normally do in such a case. It's basically a needle in a haystack. We are chasing something that the only positive to come out of it would be for family. But it's opening a can of worms; do we do that for every young person that has died of drugs, or every young person that has died in custody or for another reason. We have thousands of cases of young people who die. They may have had an underlying channelopathy, but we do not have the resources to be doing all of that testing on every single person just in case.

166. The other thing to be aware of is that you can identify things during genetic testing that cause more anxiety rather than providing answers. For example, a gene that experts do not necessarily know what to do with. If we identify a gene that the experts do not know what it does, you then have opened a can of worms

for a family who potentially might have a gene that they have no clue if it will cause them any problems or not. It's a watch and wait. They will have to submit that on their insurance forms that they have this gene that may affect their life insurance and other things like jobs. It is a really fine balance with genetics at the moment because it is progressing so fast. It is almost progressing too quick for itself, and they are finding lots of genes that they do not know what to do with, and it may be that we all have these genes and we all live perfectly normal lives. But once they are found but we are not sure of their significance, what do they do with it? Do they follow those people up for the rest of their lives?

167. I have had sight of the report of Dr William Lawler dated 22 May 2017 (COPFS-00033). In this report, Dr Lawler reviews a large quantity of material from COPFS which he details at pages 1 and 2. This includes civilian and police statements, an expert witness pack, GP notes and hospital records and various experts witness reports from Drs Bleetman, Cary, Soilleux, Parkes, Lipsedge, Payne-James, Karch and Professors Sheppard, Crane and Freemont. He provides an overview of his opinions at pages 24 to 28. I am referred to the following sections at page 26:

168. *"I think that the struggle, in its totality, is very important in this case because, per se, it must have contributed substantially to the various metabolic disturbances associated with the psychological and physiological stresses just referred to. Under these circumstances, therefore, if it is accepted that the struggle per se contributed significantly to this man's death, then it must mean that the act of restraint (whether necessary or not, and whether performed appropriately or not) also contributed significantly to his death – if only because it was a significant, albeit indirect, contributor to the total stress burden affecting the deceased in general and his heart in particular."* this accords with our conclusions in the post mortem report.

169. The report continues on the subject of restraint at page 26, paragraph 13, in which Dr Lawler discusses the findings of petechial haemorrhages. He states *"I certainly do not think that they must reflect some form of asphyxia – they could be an entirely non-specific finding in association with a cardiac arrhythmia, and*

 

they could even have been a consequence of the intense resuscitation which was carried out." Again, this accords with our conclusions in the post mortem report.

170. He then concludes *"whilst it is never going to be possible to exclude completely the possibility that this aspect of the restraint may have made a minimal contribution to collapse and death (being, perhaps, 'the straw which broke the camel's back'), I think it very unlikely, and I do so particularly in the light of the literature quoted extensively in his report by Dr Karch."* I have been asked whether this is that a conclusion that I could reach on the basis of the same evidence. I do not think I would be able to make that conclusion. I think it is difficult to know because he has obviously been restrained in a position that potentially might have caused asphyxia. I do not think you can minimise it. I do not think I can take that and say it is less important than the restraint or it is more important than the drugs. Again, because of the whole situation and the whole findings, I think they all just need to be put together.

Causation

171. I have been asked whether I consider that the drugs taken by Mr Bayoh materially contributed to his death? Yes, absolutely because if you take everything else out of the equation, as explained previously, and he just was found dead at home with those drugs on board, then that would have been his cause of death.

172. I have been asked whether I consider that the restraint and struggle against restraint caused or materially contributed to the death? Again, it would have to be, yes. If he had just died during the restraint with the struggle and did not have drugs on board, and we did not find anything else at post-mortem, we would have to postulate that that was why he's died, because of the metabolic effects and the hypoxic effects of the restraint.

173. I have been asked whether I am able to comment on which aspects of the restraint were the most significant. No, I don't think you can say. It's a dynamic situation. There is a lot going on. We do not have an absolute clear picture of what has happened, and I think it's impossible to say. I think all of

I think it's impossible to say

these factors have to feed into each other, so I would not be able to separate them out.

174. I have been asked whether, if the Chair made a finding that PC Walker, weighing 25 stones, had been lying across on the upper part of Mr Bayoh's back during the restraint, would be a significant aspect of the restraint. I understand that this is a matter of dispute and is simply a theoretical question. I would imagine if you've got a person weighing 25 stones lying on you, even for a muscular man like Sheku Bayoh, that is a cause of the restraint, keeping him down and also and the asphyxia part of it as well if he is lying on him and he's facedown, depending on how this is affecting his airway. Again, this is just, I suppose, more of a common-sense thing rather than an actual, "I have seen this before," or, "I have evidence for this." I think having 25 stones lying on your back, even if you are a reasonable size, is going to push you down towards the ground, may affect your airway, and presumably will make you try and struggle more against that as well. I cannot see how it would have helped if that, indeed, was the scenario.

175. I have been asked about the petechial haemorrhages and whether they are consistent with the degree of asphyxia. As explained, they're not pathognomonic for asphyxia, so they can be seen in asphyxia, and they can be seen in resuscitation. So it is impossible to say what the causes of those petechial haemorrhages were in this case. It is not possible for me to provide an opinion on their most likely cause.

176. I have been asked whether asphyxia caused or materially contributed to the death? It's a possibility – given the information we've been given the scenario, given his position, given how he's been restrained – that asphyxia has potentially played a role and been part of that whole scenario. I cannot categorically say that it did. It is impossible to prove from a pathological point of view, and what we have to go on is the scenario, how he has been and how he's been restrained. I think it is definitely in there as part of the scenario of how he has died. I certainly would not minimise it or take it out of the equation.

 [redacted]

177. I have been asked whether sickle cell trait caused or materially contributed to the death of Sheku Bayoh? As discussed, in my opinion, would be small print, and would not be one of the major factors and major reasons that Mr Bayoh died. It's something that I would list in part 2 of the death certificate. I understand that factors that are a material contribution anything more than *de minimis.* Then, yes because it does require consideration, but from a small print point of view. I think it has to be considered. You cannot just exclude it.

178. I have been asked whether, on the balance of probabilities, but for the drugs, would Sheku Bayoh have died i.e if drugs were the only factor. Potentially, yes. Similarly, with the restraint, on the balance of probabilities, but for the restraint would Sheku Bayoh have died, yes. If you take the drugs out of the equation, he still could have died and, similarly, if you take the restraint and the struggling out of the equation and just have the drugs, then he could have died. Either of them could be a valid cause of death in and of themselves.

179. I have been asked about whether I can comment on whether Mr Bayoh would have been in first in respiratory or cardiac arrest. No, it is a clinical diagnosis, not a pathological one. It's out with my expertise. Practically, in the whole situation, it is very difficult to know if Mr Bayoh did or did not have a pulse. Was it there but just not felt, or was it felt and it was not really there in the whole dynamics of the situation.

180. I have been asked, if the Chair made a finding that Mr Bayoh was in respiratory arrest prior to cardiac arrest, what, in my view on the balance of probabilities, would have been the cause. Respiratory arrest is normally something that is a problem with the airway. When you go into cardiac arrest, it is your heart that s basically stopped, so you still have a pulse, but you are not breathing. It is normally something to do with your airway being blocked. Or giving that he was restrained face down, his position and asphyxia could have been a factor in that if they were looking at purely respiratory arrest.

181. I have been asked whether the MDMA or the Alpha-PVP cause respiratory arrest. These drugs are stimulan[redacted] ardiac drugs so they do tend to cause

arrest. Whether you get respiratory arrest before you get the cardiac arrest, due to the drugs I do not know. You get other drugs that are depressant drugs that can reduce breathing, but these ones are more stimulant drugs, so they affect the cardiac cycle and they cause arrhythmias.

182. I have been asked whether it be correct to say that some forensic pathologists would have more expertise in restraint deaths than others. Yes, absolutely. For example, I think Dr Nat Cary and Prof Crane are 20 to 30 years in the job, so are very experienced. They will have seen a lot of these types of cases, and consequently these type of cases are then referred to them for expert opinion. So, yes, absolutely, there will be some forensic pathologists who have had more exposure to these types of deaths than others. In terms of my own experience, I see these types of deaths from a homicidal point of view, but it is a very rare cause of homicide. We see it with police-related deaths as well but, again, that's rare. I would maybe only see one a year from a homicide point of view, or police-related cases, maybe one every four or five years.

183. 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..... April 19, 2023 | 1:22 PM BST

Signature of Witness ..... [redacted]