The Sheku Bayoh Public Inquiry

Witness Statement

Professor Anthony Freemont

Taken by [redacted]

Via MS Teams

on Thursday 15th December 2022 and Friday 6th January 2023

Witness details

1. My name is Anthony Freemont. My contact details are known to the Inquiry.

2. I am an Emeritus Professor of Osteoarticular Pathology.

Qualifications and experience

3. I hold a BSc (1973), MB BS (1976), LRCP and MRCS (1976). In terms of my Postgraduate Qualifications, I am MRCP (Member of the Royal College of Physicians - UK) 1979; MD London (1984) – this was in relation to lymphocyte migration into diseased tissue. MRCPath (Member of the Royal College of Pathologists) 1986; FRCP(E) 1991, FRCP 1995; FRCPath 1996.

4. I worked at the University of Manchester from 1980 and as a Professor of Osteoarticular Pathology from 1994 until I retired from the university in 2020. I worked as a clinical academic, so I was also partly employed by the Central Manchester Foundation Trust (I retired from the NHS in 2018). In 2010 I was awarded the named chair, Procter Professor of Pathology, by the University, and in 2017, I also became a research Professor in Biomedical Egyptology.

5. Following my retirement, I was contact by the Home Office and asked to assist with a backlog in medico-legal fracture cases. Consequently, I have continued working, on an as needed basis, as an osteoarticular pathologist at the Robert Jones and Agnes Hunt Hospital NHS Trust. I have taken on 13 new cases in the last 10 working months and been involved in the training of another osteoarticular pathologist to take over from me in due course.

6. I undertook work for the coroner for more than 30 years, specialising in the process of fracture. For the coroner, I was also a general pathologist and appeared in the coroner's court giving evidence on cause of death in sudden death cases like any pathologist would. In medical legal terms I specialise in bone and joint diseases, and I particularly have skills in and around fractures, their causes and their healing.

7. I have experience over 30 years of dealing with fracture cases which involved decomposed tissue. In a portion of case, mainly in adults, fractures are found at post mortem in which the patient's tissues have undergone decomposition. This varies from internal decomposition through decomposition that is more extensive and sometime generalised. As Professor in Biomedical Egyptology, my work involved using a variety of techniques to examine mummified human tissues. In all cases involving decomposed tissue, I use special techniques to aid examination of significant events in decomposed tissue. The include the special histological stains, such as those I requested in the case of Mr Bayoh.

Reports

8. I have been referred to two reports I prepared as instructed by the Crown Office and Procurator Fiscal Service (COPFS). My first report is dated 3 May 2017.[1] My final report is dated 3 July 2017.[2]

9. I have been asked why the final report refers to the earlier report as a "draft". The reason that there are two reports is that I wanted to give a report as quickly as I

[1] COPFS-00036
[2] COPFS-00037

possibly could, but once I produced that first report I said, “I will now need to do some more tests.” I then went ahead and requested some more tests on the tissue that I'd been sent. The outcome of these were incorporated into a final report. All the final report did was to add to the draft initial report.

Instructions for report

10. I was instructed as an expert by COPFS. I received a letter of instruction from COPFS dated 16 March 2017.[3]

11. I am referred to the following paragraph within the letter of instruction:

“Further toxicological investigations revealed the presence of alcohol, MDMA and a drug known as Alpha-PVP within Mr Bayoh’s blood.”

12. I have been informed that, whilst the Inquiry has heard evidence that Mr Bayoh had been drinking alcohol the night before the incident on 3 May 2015, the toxicology results did not show the presence of alcohol in Mr Bayoh’s blood. I have been asked if this would have had any significance to the investigations that I was carrying out. No, it would not.

13. I have been asked if I received all of the information noted within the letter of instruction as being contained within an encrypted pen drive. Yes, I certainly had most of those things and probably all of them, but I can't say off the top of my head. At the time I examined the slides, things like the Drug Control Centre Independent Analysis that, whilst of importance to the case and my ultimate interpretation, were not relevant to my initial examination of the slides which I prefer to do as objectively as possible.

Sound of Rib Fracture

14. Within the letter of instruction, I was asked by COPFS to consider whether Mr Bayoh’s rib could have been fractured during CPR. I have been asked, in my

[3] COPFS-03578

experience, if a rib was fractured during CPR would it have been audible. Yes, if a rib is fractured during CPR you can hear the crack.

15. I have been asked if this would have been the case for the type of fracture that I saw in this case. That's a difficult question to answer in the sense that I don't believe that this fracture was caused by CPR. Were the fracture to have occurred during CPR, you would have heard it, but did it occur during CPR and was it what was heard by the person performing CPR? I think the answer to both is "no" in the sense that I don't think this fracture was caused by CPR.

16. I have been informed that the Inquiry has heard evidence from police officers that they heard a rib breaking in Mr Bayoh's chest area during CPR. I have been asked if, in the event the rib fracture was caused by CPR, this matches with the officers' accounts. If the incident occurred and was caused by CPR, then yes, she would have heard the sound. It isn't clear, however, what is meant by the reference to "the chest area". The first rib is right up at the bottom of the neck. That might be called the chest, but I'm not too sure that a layperson would describe it that way.

Staining of Slides

17. I have been referred to page 2 of my final report[4] and the reference, under the heading "Report", to my examination of six microscope slides and one tissue block, labelled F15 542 – AM. The report states that the slides were stained with H&E and Perls and I have been asked what this means. H&E stands for haematoxylin and eosin. Haematoxylin is a blue stain and eosin a pink one. It's the standard stain combination used in all histopathology. They would be the stains that histopathologists would routinely use to identify what was occurring within the tissues. This is made possible by differential colour staining of things like the nuclei of cells and cartilage staining blue, and bone and proteins stain pink.

[4] COPFS-00037

18. The Perl's stain is for iron. Where there is haemorrhage of long standing in life, you get iron leaching out of the red blood cells naturally or after decomposition and this stain allows you to pick that up. So these stains would have been used to examine what was happening in the tissue at the time the rib was removed from the body.

Decomposition

19. I have been referred to page 2 of my final report, where I state:

"Histologically the most striking feature, other than the fracture, was the degree of decomposition in the tissue with widespread evidence of gas formation and subsequent compression of some soft tissues. In addition many of the landmarks used to age fractures, particularly early fractures, were missing as a consequence of tissue decomposition."

20. I have been asked whether the decomposition would have occurred before or after the samples were taken from the body and fixed in the tissue blocks and slides. My understanding was that no fracture was seen originally at post mortem, but that on review of the radiology the fracture was noted. This triggered the pathologist to return to the body to remove the piece of bone containing the fracture. Although bodies are refrigerated, that doesn't stop decomposition occurring and, particularly, you get infiltration of the tissues by gas-forming bacteria. So, the decomposition occurred before the fracture was taken and was a consequence of body storage.

21. I have been asked if the tissue, once it is in the slides, is fixed in such a way that it does not decompose further. Yes, tissue is usually fixed with a fixative called formaldehyde and that prevents any further degradation of the tissue.

22. I have been asked if the degree of decomposition in this case suggests that there was an issue with the conditions in which the body or the tissue samples had been kept. A body can be deep frozen, but the usual way for a body to be stored is within what amounts to a refrigerated room. There's lots of access doors so that you can get out individual bodies. [redacted] haven't

completely prevented all the processes of decomposition. Clearly, I cannot answer with absolute certainty as I haven't seen the refrigerated room or the body, but looking at the tissue I don't think there was anything abnormal about the way in which the body behaved after it was placed into the cold room. I don't think there was anything negligent about the way in which the body was stored that comes from the appearances that I saw within the tissue.

23. I have been asked what landmarks are used to age fractures and how they are affected by decomposition. A brief list of the landmarks is set out within my report. When you break a bone, the first thing that happens if the person is alive when the bone is broken is that you get haemorrhage into the tissues. That haemorrhage tends to remain in and around the fracture site. Then the haemorrhage – which in addition to red blood cells, contains the contents of the blood as well – starts, by a chemical reaction, to form a molecule called fibrin, which is a long fibril-like material, as its name would suggest. The fibrin gradually increases in amount until eventually it becomes visible down the microscope. It binds the red blood cells together. Part of the haemorrhage brings cells called platelets and other components of the blood into the fracture. This then initiates a cellular reaction within the tissues, which we think of as fracture healing.

24. For the first day and a half you can see the haemorrhage but the red blood cells gradually break down and become less visible. The next feature to appear is the formation of visible fibrin. Ultimately a material called fracture callus starts to form. It's a bit like plaster of Paris, in that you get emergency new bone forming to try to repair the fracture defect. Initially callus is a primitive bone-like material made up of protein, but it doesn't start to mineralise until about two weeks after the fracture occurred. Some part of it forms on the inside of the bone, but most of it forms on the outside. This material will grow from the two sides of the fracture, on the outside of the bone particularly, and eventually join the two bone ends together. It's fairly flexible material, but once the bone ends are joined together by this material, you then get stability of the fracture. Finally a set of processes occur, which are mineralisation of the fracture callus occurs and then, over a matter of months, the restructuring of the callus so that it conforms to the shape of the bone again.

Signature of Witness [redacted]

25. The very earliest processes of haemorrhage and fibrin formation and the initial cellular responses leading to callus formation can be masked as a consequence of decomposition. Particularly affected is the presence of visible red cells. The fibrin, if it has formed, tends to stay. It breaks down much slower in the situation of decomposition than do cells. You lose these early direct markers, evidenced by the amount of haemorrhage, which decreases anyway with time. The formation of fibrin, which you should still be able to see in part or whole following decomposition, is followed by the earliest stages of cell activation and the formation of very early fracture callus.

26. When I was using the landmarks that I saw in the tissue slides to age the fracture, I took into account the fact that the tissue was decomposed and the effect that that would have had on those landmarks. The effect of decomposition was the main reason why I went on and did the extra tests.

27. I have been referred to pages 2 and 3 of my final report, where I state:

"From my experience of many cases in which there is tissue decomposition, established osseous vital reactions in response to the fracture tend to remain visible, particularly early osteoid formation. There was no evidence of this in the sections I was reviewing."

28. I have been asked what conclusions I draw from this. The osteoid, once it's laid down, is really bone without the mineral. Once the osteoid has been laid down, it will be broken down very slowly by the processes of decomposition, so you should still be able to see it. That it wasn't there helps ascertain a timeframe over which the fracture could not have happened. Early fracture callus should always be present by about four-five days. Sometime between one and a half and four days, you would start to see the formation of osteoid, and we don't see the formation of osteoid here. It is relatively resistant to breakdown by decomposition; therefore we can use its absence as one timeframe point.

29. I have been asked what signs of a vital reaction I would ordinarily expect to see. Before osteoid forms, the cells in and around the bone start to lay down a

fibrous material, and that material and the cells within it form a shape that's very much like the new osteoid/bone will be. That starts forming at about 36 hours and I would have expected to have seen that had the fracture occurred 36 hours or more before death. I didn't see any. Would I have expected to see it with decomposition? Yes, I think I would have expected to see some with this extent of decomposition. Not seeing this means we can begin to move the timeframe over which the fracture had occurred back from 36 hours. All the times that I give are related to the time of cessation of a circulation, which is the bone and joint pathologist definition of death.

Haemorrhage

30. I have been referred to page 3 of my final report, where I state:

"There were very few intact red cells visible. However in the marrow and the periosteum there was red amorphous material that had all the characteristics (other than the presence of intact red blood cells) of recent haemorrhage. After haemorrhage occurs a clot starts to form. By about 4 hours after the initial fracture it is possible to start to see the [fibrillar] *clotting elements of blood – fibrin strands – start to appear under the microscope. Established fibrin strands (seen from about 12 hours onwards) are largely resistant to early decomposition, such as was present in this case. These fibrin strands were not seen. Indeed, no fibrin was seen."*

31. I have been asked if haemorrhage is a vital reaction. I am glad that this question has been asked, because it sounded as if I might have been saying that it was. Haemorrhage is a reaction to the fracture. Almost immediately after you break a bone, and it bleeds. The vital reaction is when the cells in the tissue start to repair the fracture. That starts at around about 36 hours. First of all, blood contains red blood cells, but it also contains proteins, which when activated – and that is just a purely chemical reaction – form these fibrillar strands that once enough have formed can be seen with the microscope, which is one way that we recognise that fibrin has formed. Another is by the use of special stains particularly MSB (see paragraph 76). That is just a reaction that occurs inside or outside the body. If you cut yourself, you get a scab forming, and a lot of that is

made up of fibrin, and that does not involve the cells within the tissue itself. It all comes from the blood.

32. I have been asked what conclusions I draw from the presence of haemorrhage. There is a debate as to whether or not haemorrhage can occur after death. I've done some experiments, which I haven't published, on animal bones. Within the bone marrow are big blood-filled spaces, and if you break the bone after death , then those blood-filled spaces can spill out blood, but the amount of blood is small, and it doesn't always occur. If you've got a blood flow to the tissue, i.e. the heart is still pumping, then you will get much more haemorrhage. Then it becomes a question of experience as to whether or not there is sufficient haemorrhage for one to say whether there was or not a circulation at the time of fracture. The amount of bleeding in this case could be seen macroscopically. Figure 1 in the appendix of my report shows a picture that must have been taken by the pathologists when they removed the bone. I didn't see the bone itself. I only saw photographic images, tissue sections and the tissue block. I can see this haemorrhage, which extends into and through the soft tissues adjacent to the fracture site. When I looked down the microscope, I could see what I thought was haemorrhage despite the decomposition. The amount of haemorrhage suggests that the fracturing occurred in life, and that was backed up by some of the other things that I saw. Again to be sure I was looking at haemorrhage, I requested a special stain, glycophorin A, to be undertaken (see paragraphs 71-74).

33. I have been asked what conclusions I draw from the absence of fibrin. Again, it's a time point. It's not a very accurate time point because before you can see fibrin down the microscope there has to be a certain amount of it. It’s very difficult to see individual strands of fibrin, and so you see what's called a fibrin clot where you get a lot of these fibrils together. That was what gave me the upper time limits. I would have expected, by 24 hours for instance, to see quite a lot of fibrin and by 12 hours to have been able to visualise some fibrin. The fact that I hadn't, suggested an upper time limit to when the fracture occurred relative to death, which could have extended to 24 hours prior to death. But I thought it was probably much less.

Osteocyte necrosis

34. I have been referred to the section titled “Fractures” on page 3 of my report, where I state:

> *“2 hours – osteocyte necrosis at fracture edge first seen. Probably established by 6 hours and certainly by 24 hours (NB. This only occurs in life, apparently intact osteocytes remaining in the tissue until well after death even in the face of decomposition).”*

35. I have been asked if the osteocyte necrosis (loss of cells through dying) confirms the fracture occurred ante-mortem. Yes. It's slightly counter-intuitive because the processes of decomposition mimic necrosis, but the cells within the bone material are called osteocytes and they are largely protected from what's going on outside in the decomposition process. The process of loss of osteocytes at fracture sites is almost certainly due to a process known as apoptosis. Apoptosis is cell-mediated cell death, so the cells effectively commit suicide, and that will only occur in life. They don't have the ability to kill themselves once the body has died, so the implication is that if you see necrosis, the fracture occurred during life.

36. Within figure 3 within the annex of my report it's very clear that there's a zone of osteocyte necrosis up against the fracture edge. In the bone beyond it, you can see the nuclei of the bone cells that were still alive when the tissue was taken.

37. The timing of the start of recognisable osteocyte loss is still a matter of continuing research. A lot of the work osteoarticular pathologists undertake is related to abused children, and they're mainly infants. My experience of timing of the onset of osteocyte apoptosis comes from looking at infants, and in infants osteocyte death can be appreciated in fractures an hour old and then the number of dead cells increases with time. There is some evidence that it might be a little bit delayed in adults, which is why I said two hours, a time coming from my own

experience. The fact that you can see the necrosis suggests based on all things I've said, the fracture occurred probably at least two hours before death.

Initial Interpretation

38. I have been referred to page 4 of my final report, where I state:

> *"The man sustained an isolated fracture of the first left rib. Further interpretation on H&E stained sections has been made difficult by the degree of post mortem decomposition. However, on balance, I felt that the residual histological features, indicated that the fracture occurred in life, certainly within twelve hours of death and probably within six."*

39. I have been asked how I came to this initial conclusion. The two things that made me think that this occurred in life were the disappearance of osteocyte nuclei , which I've just discussed, and the amount of haemorrhage that was visible macroscopically and microscopically because, although I couldn't see red blood cells, I could see other features that looked as if they had a distribution of a type that you would see with haemorrhage, and this was later supported by glycophorin A staining.

40. I couldn't see any fibrin. That gives us a top end (i.e. the furthest from death the fracture occurred) of 24 hours, 12 hours or maybe 6 hours. I feel that I could have seen evidence of fibrin by six hours, but my experience says that may not always be the case, which is why I've given a slightly higher value of 12 hours, by which time I would have expected to see fibrin.

41. I've now got bleeding and osteocyte loss at the fracture site saying the fracture occurred prior to death. I've opined a time for the closest the fracture could have occurred before death that comes from the osteocyte necrosis. This I have said was almost certainly 2 hours before death. I used "almost certainly" because whilst I can't exclude completely osteocyte necrosis occurring earlier, because I have seen it as early as 1 hour before death in infants, I have not seen this process in fractures occurring earlier than 2 hours before death in adults.

Similarly I've given a limit of 12 hours to the furthest from death the fracture occurred based on their being no fibrin formation. and I think, because I didn't see any fibrin at all, that I could probably reduce that down to six hours. Osteocyte necrosis and no fibrin formation are the two main elements of the fracture healing time frame that I've used to give a time range during which the fractures occurred before death.

Isolated first rib fractures

42. I have been referred to page 4 of my final report, where I state:

"Isolated fractures of first ribs are rare, and I needed to consult the scientific literature to find information to assist in this case."

43. I have been asked what experience I have of first rib fractures. Very little, which is why I needed to consult the scientific literature. I may have seen a few cases of first rib fractures, but I can't remember before ever having seen any that were isolated. As this was the only time I had seen an isolated first rib fracture, I had to go to the literature to find out more about this, and that bore out my experience in that it's said to be very rare indeed.

44. I must be honest, I am a little bit surprised that the police officers heard a cracking noise that is thought might have come from this rib fracturing during CPR. If I'm correct about the way in which I've interpreted the literature around CPR, then it's unlikely that they were hearing the first rib fracture, but of course it is entirely possible that the noise came from a different rib cracking, but if that was the case why were no other fractures identified? I know that it was not possible to pick out the first rib fracture initially radiologically or at post mortem. The question that's lurking in the back of my mind is whether or not there were other fractures that came as a consequence of CPR that weren't detected at post mortem. That's entirely possible, I think. Again, I've published on fractures not being detectable at post mortem or radiology, though this was in infants. So was this an isolated first rib fracture? All the positive information in this case points towards the 1st rib fracture having been [redacted]hat I've got

that there might be an undetected fracture is the noise that was heard by the officers of a cracking sound. From their description maybe one of the ribs other than the left first fractured. Would this have been spotted at post-mortem? It might not have because CPR, of course by definition, occurs after death. So, there may have been no local haemorrhage, which is one way the pathologist recognises small fractures, and even a little crack to one of the ribs might have given rise to the noise reported by the police.

45. On balance, I felt that the ante-mortem fracture of the first rib was probably isolated. I feel that the pathologists might have noticed something else had more easily visible ribs been broken at the same time as the first rib, and that's why I've given the interpretation that I have about isolated first rib fractures.

46. I have been asked if I would have seen the images from the post-mortem CT scan carried out on Mr Bayoh's body. I almost certainly wouldn't. I am very reliant on the expertise of the radiologist to interpret those, so I tend to believe what they're saying rather than trying to check up on them because I just don't have that expertise.

47. I have been referred to page 4 of my final report, where I cite a publication from the British Journal of Sports Medicine from 2004 about isolated first rib fractures. I have been asked if there is now any more recent literature in this area. I checked again prior to this interview and I didn't find any literature that I felt added more to that particular publication. The important thing about the publication wasn't that the authors described a single case of isolated rib fracture, but that they carefully reviewed the literature up to that time. After my check, I felt that any literature that I had seen didn't add to what was given in the 2004 paper. It's interesting that quite frequently people record fractures but don't say whether there was anything that led up to them. This paper from 2004 said, "Well, these are the circumstances in which people had said first rib fractures occurred," and they quoted references, which I checked, and I think I would have come to the same conclusion as they did from those.

 [redacted]

48. There may be some literature that I've missed since 2004. I tend to use a search engine called PubMed, which looks at 5,000 journals. Sometimes some of the reports about forensic medicine don't appear as those journals the forensic medicine publications are in are not amongst the 5,000. I also do a Google Scholar search which searches more widely but, once again, that can miss certain journals, and the forensic medical journals are some that are missed. The way that journals are ranked is by the number of times publications within them are cited in the same literature base. Only certain publications will get onto the PubMed database, they tend to be the ones containing the most highly cited papers within the field and whilst forensic pathology papers often guide practice they are not cited in the journals selected by the publishers of biomedical search engines. I wasn't able to find any other papers which added anything to that one from 2004. I thought that article was an accurate interpretation of the literature. I selected that paper not just the publication's report, but the publication's review of the literature.

Cause of Mr Bayoh's Rib Fracture

49. Taking account of the causes of first rib fractures that are quoted from the 2004 publication on page 5 of my report, I have been asked whether I would consider anything I have read within the witness statements in relation to the circumstances surrounding Mr Bayoh's death to be a potential cause of the fracture. Yes. In my report I've said that I think this is most likely caused by falling onto an outstretched arm, because my understanding was threefold: first, I think there's a description of an altercation that occurred a few hours before death, and then there was difficulty on behalf of the police in controlling Mr Bayoh's actions. Second, I didn't read anywhere anything that related to a direct force being applied to the rib, and the literature that's cited by the paper in 2004, and the paper itself, say that it's actually very difficult to give direct trauma to the first rib. Third, the 2004 paper I quote says that fractures can sometimes be caused by a violent contraction of the muscles acting on the first rib. However, these occur near the centre of the rib around areas of bone thinning caused by the passage of the subclavian vessels across the bone (around the scalene tubercle which is about halfway along the line off the bone). The radiology report

reads: '*There is well defined, linear lucency in the medial, posterior aspect of the left 1st rib, proximal to its junction with the 1st thoracic vertebral body.* This is much further back along the rib and therefore this key criterion for defining muscle contraction as the cause of solitary first rib fracture cannot be met.

50. The best way to understand the anatomical setting of the first rib is actually to look at yourself. If you run down the neck and then go across towards the shoulder, you come across a dinge and the front of it is the collarbone, the clavicle, and the back of it is a great pile of muscle that's on the top of the shoulder blade. When you press down very hard in that area, you suddenly come across something firm, and that's the first rib.

51. If the fracture was caused by a direct blow and did not fracture any other bones – so it's not just an isolated first rib fracture but it's a first rib fracture in the absence of a fracture of the scapula (the shoulder blade), or the clavicle (the collarbone) – then it would be a very, very targeted action through an area that's maybe a triangle, 3 by 3 by 3 inches. Could the tip of a boot have caused that bone to fracture and not damage the clavicle? I found that difficult to envisage.

52. I cannot completely exclude the idea that a chance kick or blow happened to miss all the other bones and only connected with the first rib, neither can I fully exclude severe muscle contraction caused a fracture in a place along the rib that is not usual for this aetiology. However, from everything that I've read about the case, the most likely cause, I felt, from the ones listed was a fall onto an outstretched arm. That seemed to fit with everything that at the time I'd heard about the events leading up to Mr Bayoh's death.

53. I am referred to page 5 of my first report[5], where I state:

"a) This man sustained an isolated 1st rib fracture in life, definitely within 12 and probably within 6 hours of death.
It is unlikely that the fracture was caused by CPR.

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b) Because of the anatomy, whilst a direct blow could cause the injury, it is unlikely in the absence of fractures of other adjacent bones.
The most plausible cause is an indirect injury such as falling on an outstretched arm or a blow to or a fall onto the shoulder away from the bone.
c) Moving a previously fractured rib such as in CPR could well lead to a sound."

54. I have been asked if CPR, particularly if it was making a cracking sound, would have led to displacement of the fracture and, if so, would such displacement have been evident. I do not have sufficient experience of this rib to respond directly, but extrapolating from fractures of other bones, yes, in general, the more you move the bone ends, the more likely it is for a fracture to become displaced, but the covering of the rib is a very dense, although thin, fibrous tissue layer. To actually displace the bone ends sufficiently for the fracture to move in either direction, you'd have to have considerable damage to the surface material (the periosteum), which I would conjecture might be associated with damage to some other structures such as blood vessels around the bone ends. In addition, I do not think the radiologists' description in her report is of a displaced fracture.

55. Whether doing properly targeted CPR would cause the first rib to move, I don't know, it is outside my expertise. However, from my knowledge of anatomy, my own experience of ribs that get fractured at CPR and my understanding of the literature about 1st rib injuries in relation to CPR, I doubt it. This is because it's such an unusual structure by comparison with other ribs. It's short, it's flat, and it's way up inside the neck, whereas in CPR you're applying pressure over the heart which is much further down in the chest. The ribs that are over the top of the heart, are longer, thinner, and I think more likely to fracture than transmit the force of CPR up into the structures at the bottom of the neck. I have looked specifically at the literature but I haven't managed to find evidence that occurs.

Histological Stains

56. I have been referred to page 5 of my final report, which contains a summary of the conclusion that I initially reached within my first report. I have been asked how my views developed between my first and final reports. The additions to my report that came between the first and the second version were related to

reviewing a series of histological stains that show up particular elements of the tissue. Although we don't understand the chemistry, certain stains will detect things that we're interested in seeing, such as fibrin, and evidence of haemorrhage, hence requesting extra, differently stained tissue sections. I couldn't remember with certainty when interviewed whether we stained them in Manchester, or they were stained for us at the original laboratory, but everything I have now seen confirms it to be the latter.

57. I have been referred to an e-mail that I sent to Alasdair Macleod, COPFS, on 5 June 2017.[6] From this e-mail, it looks as if working through the Procurator's Office I requested a series of stains be performed.

58. My e-mail requesting the stains refers to "MSB" and I have been asked what this is. That's a stain called Martius Scarlet Blue, and that will be looking for fibrin.

59. My e-mail also refers to "PAS" and I have been asked what this is. This is the Periodic acid-Schiff. One of the things that I noticed histologically was round lumps of pink material in the haematoxylin and eosin stained slides that could have represented material from haemorrhage left following decomposition. However, I wanted to exclude a number of other possibilities as to the cause of these and one of them is fungi. One of the sets of organisms involved in decomposition is fungi and were they present they should have stained up with Periodic acid-Schiff.

60. The PTAH referred to within my e-mail wasn't done. That's usually used for looking at muscles. There was an elastic van Gieson, which I think was to replace the Weigert stain, then there's the glycophorin A. Glycophorin A is a protein that's found in red blood cells, and I wanted to see if I could find a surrogate marker for red blood cells that were no longer intact due to decomposition. As they decompose, they would have released the glycophorin A that could have remained in the tissue at the site of red cell breakdown, so I asked for that. The way that's done is by immunohistochemistry. There, an antibody is raised against the glycophorin A and we use a technique called immunohistochemistry

[6] COPFS-04310

which is shortened in the email to IHC to show if there was and where the glycophorin is. This is discussed further in paragraphs 70-77

61. I am not sure why the stains are slightly different to those that I requested, but they are not meaningfully different. I suspect almost certainly that by the time I received the slides I'd forgotten what I asked to be done, and so listed only the stains that arrived.

Further inquiries

62. I have been referred to page 6 of my final report, which makes reference to the further inquiries and investigations that I carried out between my first and final reports. These further inquiries included a request that I made for additional information as to why there was tissue decomposition. I have been referred to an e-mail from Kerryanne Shearer to Alasdair Macleod, dated 17 May 2017 at 12.21.[7] The e-mail from Dr Shearer contains the text of a previous e-mail sent to her by Alasdair Macleod:

> *"As you are aware the Crown have instructed Professor Anthony Freemont, an Osteoarticular Pathologist at Manchester University to provide an opinion on inter alia the mechanism of the fracture to the deceased's left first rib which was discovered by a CT scan on 27 May 2015.*
>
> *Following examination of tissue samples in Manchester on 27 April 2015 Professor Freemont has approached the Crown with a number of questions.*
>
> *Professor Freemont is looking to reconcile why the fractures are in decomposed tissue and cannot find a sequence of events. I would be grateful for clarification of how it was identified and at what stage. Was it on the basis of finding bruising or palpation of the fracture at post mortem examination or was it at post mortem radiology?*

[7] COPFS-03737

Professor Freemont wishes to know what ancillary post mortem features which might indicate the ante mortem nature of the fracture.
Finally, Professor Freemont notes that the two pieces of bone on the slide were fractured. He states that the fractures looked different one being transverse and the other diagonal. Was this because the same bone was cut lengthways and the pieces placed into the block at different angles?"

63. The e-mail from Dr Shearer in response states:

"With regards to the letter noted below I can confirm that the bone was indeed cut lengthways and the pieces must have been placed in the block at different angles to explain the differences.

With regards to the finding of the rib fracture, there was nothing obviously seen at the time of the post mortem (4/5/15). A skeletal survey was undertaken (13/5/17) after the post mortem which showed no obvious bony abnormality but because the lateral spine could not be visualised a CT was undertaken (28/5/15).

This showed 'There is well defined, linear lucency in the medial, posterior aspect of the left 1st rib, proximal to its junction with the 1st thoracic vertebral body. This involves the anterior cortical surface only'.

Following this we went back to the body (29/5/15) which revealed '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'. This fracture was sampled. There was only very focal possible soft tissue haemorrhage associated with the fracture so no real definite gross indicators to confirm it was definitely ante mortem.

64. It is interesting when you read those. If you take the wording as being accurate, the radiology says, "This involves the anterior cortical surface only" and the

pathology says: “a fracture through the rib.” You can see that the radiology, I think, is saying that there’s only a partial fracture of the bone and the pathology is saying there’s a complete fracture through the bone. I think that indicates some of the difficulties that the radiologists have in identifying fractures in these sorts of settings because, as far as I can see, there is a full thickness fracture of the bone.

65. I have been asked if I was provided with photographs of the rib. Yes, they are in my report. Figure 1 within the annex to the report says: “Macroscopic image of the 1st rib taken at time of post-mortem examination.” It was one of four images attached from the email entitled, “rib pictures, part two.” You can see there’s a green arrow that points at the dark red area that I believe is haemorrhage. I think the pathologists performing the post mortem interpreted it the same way although I note the phrase “*so no real definite gross indicators to confirm it was definitely ante mortem*” is carefully worded. Taking together the extension of the haemorrhage into the soft tissues overlying the fracture site, the structure of the first rib, and the density of the soft tissue coverings of the rib alluded to in paragraphs 54 and 55, and the advantage that I had in knowing the microscopic features of the fracture that included the degree of decomposition, and the results from the special stains (particularly the density of glycophorin A immunostaining), neither of which I believe was known by the pathologists at the time of removal of the rib, I concluded that this was sufficient evidence to say the haemorrhage was the result of an antemortem fracture. My exact wording was “*Although haemorrhage can occur in fractures after death (e.g. fractures caused by CPR), the bleeding, if it happens at all, is minimal. The amount and distribution of the glycophorin staining is therefore much more in keeping with antemortem haemorrhage.”*

Sickle Cell Trait

66. I have asked if I was aware that Sheku Bayoh had sickle cell trait. No, I have no recollection of this.

 

67. I have been asked if Mr Bayoh having sickle cell trait would have any significance to the signs that I saw. No, sickle cell disease affects the red blood cells but not the other clotting elements as I understand it. Sometimes down the microscope the intact red blood cells of someone with sickle cell trait can appear sickle shaped rather than donut shaped if the patient has been ischaemic – they haven't got enough oxygen in their blood. This is much more common in patients with sickle cell disease – you can sometimes recognise sickle-shaped red blood cells, which is where the disease gets its name from. When tissue is fixed, the red blood cells change their shape anyway and sometimes they can appear sickle shaped, but my understanding is that it would not have affected the clotting process.

68. I have been asked if Mr Bayoh having sickle cell trait would have affected the fibrin at all. No, it shouldn't have done. If fibrin had formed it would have been visible, and the disease should not have affected the ability of fibrin to form. It doesn't affect that bit of the chemical reaction that you see in the blood.

69. I have been asked if the sickle cell trait could account for the absence of intact red blood cells. No. The red blood cells do break down more easily in sickle cell disease, but that's usually because they're breaking down in areas where red cells usually break down, so it should not have influenced the timing of the effects of decomposition and certainly not in Sickle Cell Trait.

Review of Special Stains

70. I have been referred to page 7 of my final report, where I state, under the heading "Review of special stains: 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. This showed this material had no integral structure, was not caused as a by-product of fungal infestation, and had features seen in blood clot."

71. I have been asked to identify the features seen in blood clots to which I am referring. When blood clots it doesn't clot as a whole, early clots form in layers.

 

When processed the material separates into the layers. It's called lamellation. Sometimes in processed tissue the lamellae round up. Interpreting these appearances is made much more difficult by tissue decomposition which is why I asked for the special stains which are designed to identify special molecules in the tissue.

Glycophorin A

72. I have been referred to pages 7 and 8 of my final report, where I state, under the heading “Glycophorin A”:

“This recognises a molecule on red blood cell walls. Normally in tissue (even necrotic tissue) red cells are restricted to the inside of blood vessels. If there has been haemorrhage into the tissues, even after the red cells have broken down, it is my limited experience that glycophorin A may be detected sticking to the tissues and the areas of soft tissue haemorrhage (this includes fracture sites). Glycophorin A was seen in marrow (red blood cells are manufactured in the marrow, and this is therefore an expected finding) and also in large quantities at the fracture site.

Although haemorrhage can occur in fractures after death (eg fractures caused by CPR), the bleeding, if it happens at all, is minimal. The amount and distribution of the glycophorin staining is therefore much more in keeping with antemortem haemorrhage.”

73. The lamellar material stained with glycophorin A, which can be seen in figure 5 of my final report. Figure 5 also shows heavy stating for glycophorin A in the fracture gap and into the soft tissues on the surface of the rib. In my opinion this is strong evidence for there being blood that could only have been present in these tissues and in this amount if it was caused by antemortem haemorrhage.

74. I have been asked if sickle cell trait is relevant to this. No. Sickle cell disease is so called because the red cells change their conformation and instead of looking like donuts down the microscope they look like sickles. Patients with sickle cell

disease have two abnormal haemoglobin genes. Having this disease offers some protection against malaria. Sickle cell trait (SCT) patients have one abnormal and one normal haemoglobin genes. People with SCT usually do not have any of the signs of the disease, but, in rare cases, a person with SCT may problems, most often when there are other stresses on the body, such as dehydrated or after strenuous exercise. There is a growing literature about glycophorin genes and resistance to malaria, but I know of no linkage between the two, and neither know nor can find reports of a difference in glycophorin A expression on the surface of sickled cells and normal cells. The red cell membranes contain glycophorin A so if you get haemorrhage in someone with sickle cell disease then you would expect to still find the glycophorin A.

Elastic Van Gieson Test

75. I have been asked what the elastic van Gieson test was for. It allows you to see pre-existing structures that contain a molecule called elastin (elastin fibres stain black) and collagenous fibrous tissue (collagen stains red). It does not differentially stain fibrin. I used this to try to establish if the lamellated material was collagenous or whether some of the rounded structures were blood vessels. Blood vessels have elastin in their walls which would stain black. The lamellated material in the fracture gap could have been periosteal collagen forced into the marrow cavity. In which case it would have stained red, but the staining is a non-specific yellow.

Martius Scarlet Blue (MSB)

76. Martius scarlet blue was designed as a stain for fibrin. Depending on the age of the fibrin it stains the fibres - fresh (orange-yellow), mature (red) or old (blue). None of these colours are present in my figure. Within figure 4 of my final report, in referring to a rounded structure in the marrow it says “MSB shows internal lamination.” If you look at the top image within figure 4, there’s a round structure with the arrowhead virtually over the middle. The same structure appears in the bottom image (PAS). If you then look at the MSB, you can see the same rounded structure that’s made up of different layers. There’s a sort of dark grey area around the outside top, and then there’s a paler grey area around the inside and separating the two is a brownish line. At first it looks like a blood vessel, but it

remains solid over multiple tissue sections. You can think of a tissue section as a slice from a loaf. It might contain a hole, but in later and earlier slices the hole disappears and others may appear. Looking at multiple tissue sections gives you a better idea of the overall structure, and a hollow structure might be expected to reveal its lumen (central hole). There is a special blood vessel in bone marrow called a sinusoid. It is large with a thin wall a little like a sausage skin and contains no elastin. It is one structure that you might have expected to show a lumen across serial sections. Its wall would be blue with the MSB but this structure isn't. This structure is laminated so something has been laid down on something else. That's the way that the blood starts to clot. True blood clot contains fibrin, but it needn't necessarily have microscope-visible fibrin associated with it. You rarely see very early fibrin because there's just not enough of it or the fibres are too thin.

Periodic Acid Schiff (PAS)

77. The PAS I also asked for was to exclude the presence of fungi which have long fibre-like "tentacles" and can form rounded bodies. They are found associated with decomposition. The sugars in their walls stain a bright pink. The lamellae and rounded bodies are a muddy mauve/bluey grey

Osteocyte Necrosis

78. I have been referred to the paper annexed to my final report, titled "Death of Osteocytes: Electron Microscopy After In Vitro Ischaemia". This paper states, at page 26 of my final report:

> *Concerning the original question of the viability of osteocytes under total ischemia, our observations provide good arguments for the assumption that osteocytes can tolerate no more than two hours of total ischemia. If this is so, they behave like all other nucleated human cells which in general can endure in vivo ischemia at 37 C (temperature is of course very important) for a maximum of two hours before sustaining irreversible damage.*

Signature of Witness … [redacted]

79. I have been asked how relevant this paper is to my findings in Mr Bayoh's case. This was one piece of information amongst many that I have employed to study the significance of my observations around osteocyte necrosis. I included it because, it did show the state of understanding of osteocyte cell death in 1986, really before apoptosis became widely recognised as a mechanism by which all cells might die. This was part of the evidence I used to understand the significance of a review I was undertaking of fractures. In 2017 this review was partially complete. This review is now a peer reviewed publication *Histological ageing of fractures in infants: a practical algorithm for assessing infants suspected of accidental or non-accidental injury* (WIT-00027).[8] I presented my evidence underpinning the possibility that histological detection of osteocyte apoptosis can occur within an hour of death.

80. What I have done is review a huge number of cases that were sent to me at the Manchester Royal Infirmary and the University of Manchester over a 32-year period. The focus was fractures sent as medicolegal cases, but there were also fractures sent in my normal clinical practice and uniquely some from a major study of the biology of fracture healing I undertook with funding from the Medical Research Council, considered by many as the most prestigious funder of medical research in the UK.

81. The trigger for my review was a lack of understanding of the mechanisms to age fractures in potentially abused infants. Fractures in infants, because of the settings in which they occur and as they are usually related to abuse, are very difficult to age, because everybody says, "No, I have nothing to do with hurting this child."

82. There are, however, certain cases where it is known how long before death the fractures occurred. From infants I extracted 169 fractures of different ages following death, at which the age of fracture was known. This was sufficient to perform scientific analysis, which is why a study like this can only be performed in a place that examines a lot of infant fractures. I was able to identify a progression

[8] Histopathology 2019, 75, 74–80

of histological features from the time of fracturing to late healing and to age them as "hours or days before death". I tried to do the same with other age groups (Children and adults). There were only 20 fractures in children in my data set that reached entry inclusion criteria for the study. Although there were 171 fractures in adults, they covered such a wide range of ages, and the same 15 features that I could not perform scientifically meaningful data analysis. In particular I had relatively few fractures (37 adults vs 81 infants) removed from a living person in 48 hours of the fracture, or the person died within the same time frame. However, the study did show that the order of progression of the 15 features was the same as in infants and the timings were similar (except perhaps the elderly where fracture healing, particularly in women with osteoporosis. The most obvious exception was osteocyte necrosis which was regularly seen in infant fractures 1 hour after the fracture occurs but I had no adult cases in which osteocyte necrosis was seen sooner than 2 hours before death. The paper is therefore relevant in that very broadly it says that if you undertake experiments in animals you get similar results to humans. Of course, this is nowhere near as relevant to a carefully managed study in humans, but at the time of writing my report in 2017 I did not have the fully analysed data sets I describe above.

83. I have been asked if I am aware of any more recent publications in timing the appearance of osteocyte necrosis before death. Not from my knowledge, no, with the exception of the paper that I published on infants (WIT-00027).

84. I have been referred to page 7 of my final report, where I state, with reference to osteocyte necrosis:

> *"The timing of this phenomenon is not clear, but the available evidence (e.g. paper 4 attached) and my own experience, indicates that the process can be visualized possibly, as early as 2 hours after onset, probably by 6 hours and definitely by 24 hours. So its presence at the edge of a fracture indicates that the fracture occurred in life and at least 2 hours before death."*

85. "Two hours after onset" means two hours after the fracture.

86. I have been asked if I can definitely say it is two hours. No, I can't be more accurate than that. If you use two hours as a time point, then you can be sure that you would have seen established osteocyte necrosis. What I can't say is would I have seen any osteocyte necrosis at one hour. I have always thought of it as something that accumulates over time, more and more cells undergoing apoptosis over a period of time, so eventually you reach a point where the loss of cells is clear. I have been asked if it could have happened closer to death than two hours. Yes. In infants, I've seen established osteocyte necrosis as soon as one hour after death. I have it in my mind that I have seen it stated in writing that the process occurs a little bit later in adults, but I've searched high and low through all the publications that I hold and I cannot find that statement.

87. In this case my data and experience have to be triangulated against the timing and nature of the events on the morning of Mr Bayoh's death including his interactions with the police and others. Could the fracture have occurred less than two hours of death? Data available to me now, but not in 2017 have shown that it could. I don't think it was because the osteocyte necrosis was well established, but, yes, the new data show reasons why there might be an element of difficulty now in Mr Bayoh's case in saying, "*at least 2 hours before death*".

Steroid Use

88. I now understand that Mr Bayoh's GP records from 2011 note that he had been taking anabolic steroids for approximately 2 years, on a 6 week cycle. I have also had sight of the letter from Dr C Walker dated 23.06.15 (COPFS-02380) providing the post mortem toxicology results confirmed the presence of anabolic steroids in Mr Bayoh's urine. Certain steroids will weaken bones. The effects of anabolic steroids however, should not have caused the bones to get weaker and indeed, I saw no evidence of a loss of bone material in the segment of rib I was sent.

89. The tissue called "bone" is what makes up a bone, and so you must be very specific about what you're talking about. Osteoporosis is where you lose bone tissue from a bone, and I saw no evidence of that in the sample of Mr Bayoh's

 

bone I examined. Had it been present that would have weakened the bone. Anabolic steroids on the other hand are used by athletes to increase their muscle bulk, so when I saw that the publication on 1st rib fractures, was referring to athletes, I thought, "Oh, well, this might be significant in this case."

90. The regular use of anabolic steroids is one of the reasons that in paragraph 87 I said that new data show reasons why there might be an element of difficulty now in saying in Mr Bayoh's case the fracture occurred at least 2 hours before death. Since 2017 when I wrote my reports on Mr Bayoh there has been an increased interest in osteocyte apoptosis. Importantly it has been shown that apoptosis occurs if the bone sustains microdamage (damage short of fracture) where it is believed to initiate repair processes. It could be argued that the faster this happens the quicker healing starts to occur, in which case seeing apoptosis at one hour in infants is a reflection of the "high quality" of these infants' osteocytes to initiate repair. It has also been recognised that osteocytes develop an increased tendency to display unstimulated apoptosis with age, in effect as they age they function poorly; one manifestation of this being an increased propensity to commit unstimulated suicide. Unstimulated osteocyte death can be so bad that in elderly men with low testosterone poor osteocyte function can lead to severe, debilitating osteoporosis.

91. Over the last 5 years this has become increasingly treated with exogenous testosterone-like drugs, particularly nandrolone, the drug, or a relative of the drug Mr Bayoh had been taking. It has recently been shown that nandrolone works in this setting by restoring osteocyte function towards normal and particularly by inhibiting the abnormal driver of apoptosis. It is not known whether or how nandrolone affects osteocytes in younger men but as a moderator of osteocyte function there are arguments to support both nandrolone making the cells more like those of infants and therefore more likely to demonstrate early stimulated apoptosis in response to bone damage; but also by inhibiting apoptosis it might make the cells less likely to become apoptotic. There is no definitive answer as to which of the two is correct, but on balance the data are slightly more supportive of the first. Thus, instead of apoptosis being seen at 2 hours, in a man taking nandrolone it might appear earlier.

Signature of Witness [redacted]

92. There are no data in the literature to say by how much nandrolone could have pushed back the appearance of apoptosis in this setting, but the implication is that an assessment of the closest the fracture could have occurred relative to the time of death might need to be revised downwards bringing it closer to the time of death than the 2 hours the pathological changes on their own suggest.

93. This is a complex area but is well summarised in the following papers *The Osteocyte: New Insights*[9] (WIT-00028) and *Androgens and Androgen Receptor Actions on Bone Health and Disease: From Androgen Deficiency to Androgen Therapy*[10] (WIT-00029). I have cited these two papers as examples of the two elements of the science of osteocyte biology that impact on Mr Bayoh's case. There are many other recent publications with similar messages.

Blow to Shoulder

94. I have been informed that the Inquiry has heard evidence that Mr Bayoh was struck 2-3 times with a police baton on his arms. If the Chair were to accept this evidence I have been asked whether this is a likely mechanism of injury to the first rib.

95. I have no direct personal experience of this area of medicine, or expertise in the area, so my answers are based on my review of the relevant literature. One of the causes of isolated fractures to the first rib is a direct blow to the shoulder. It might be possible to cause a fracture of the first rib by hitting the shoulder. In return I would ask, was there evidence that he was or wasn't struck on the left shoulder? If he was then, yes, that could have caused the rib fracture. It's one of the causes that I listed from that publication that talked about athletes.

96. I have been asked if Mr Bayoh had been struck to the upper part of the left arm, would that count as being a blow to the shoulder. Whether the upper part of the upper arm would in this context include the shoulder, I just don't know.

[9] Annu Rev Physiol. 2020 Feb 10; 82: 485–506
[10] Cells 2019, 8(11), 1318

 [redacted]

Fall to the Ground

97. I have been informed that the Inquiry has heard evidence that Mr Bayoh was tackled to the ground by PC Walker in a manoeuvre described as a bear hug. PC Walker's demonstration of this tackle involved him pulling his right arm across this chest and charging at Mr Bayoh with his right shoulder. If the Chair were to accept this evidence, I have been asked whether this could be a likely mechanism of injury to the first rib. Again based on my review of the literature rather than personal experience or expertise, one could imagine a scenario when Mr Bayoh naturally would put his arms out whilst falling to try and stop himself being injured. Then one needs to consider that were he falling there's not only his weight but the weight of a policeman when he hits the ground. If he protected himself with a straight arm the force coming up the arm and into the shoulder would, I believe from what I have read, be such as to fulfil the criteria outlined in the literature

98. I have been asked if the fracture could have resulted from Mr Bayoh falling onto his back. Again this is outside my expertise but to assist the Inquiry, from my reading and my personal experience most people, when they fall onto their backs, are slightly curved forwards. Analysing the force distribution we're talking about in this scenario there is not a direct injury to the shoulder region. As such it is unlikely that this force would be transmitted from the back through to those ribs quite high up on the chest. So whilst I cannot know for certain I wouldn't have thought so.

Press-up

99. I have been informed that the Inquiry has heard evidence that, during the restraint, Mr Bayoh did a press-up. PC Tomlinson gave evidence that he was lying across Mr Bayoh's legs and that Mr Bayoh was able to use his right hand to press up from the ground. PC Good recalled seeing PC Walker lying across the top of Mr Bayoh's back towards the upper half of his body to prevent Mr Bayoh pressing up from the ground. If the Chair were to accept this evidence, I have

 [redacted]

been asked whether this could be a likely mechanism of injury to the first rib. I don't know, but what is described in the publications is falling onto an outstretched arm or a direct blow. The forces working through a push up are different to the “shock loading” of a blow or fall onto a straight arm. That said the literature does discuss that 1st rib fractures can be caused by severe contraction of the muscles acting on the first rib.

100. It is my understanding that one, and perhaps the most likely way in which the first rib could have been broken is by the application of a force in a very short time, shock loading, like hitting the ground on a straight arm, whereas “press-up” sounds rather more as if it's an extended time interval that you're applying load. But of course, if he's got at least one police officer on his back, and he was a reasonably large man himself, very muscular, then the amount of pressure and muscle contraction that it required to do a push-up would be extensive, particularly considering the combined weight of himself and the police officers. However, the main reasons I would use to discount the rib fracture being caused by this press up would be, the fracture was in the wrong place along the rib for one caused by severe muscle contractions and the reports indicate the press up was done with the right arm and the fracture was on the left side.

101. I have been asked if Mr Bayoh managed to push up and straighten his arms, whether it’s more likely that that could have caused the injury, rather than a bent arm. The question refers to “arms” which I interpret as meaning both the left and the right. I think from what I've heard, and from what I've read, that that is more likely to cause the fracture because of the contraction of the lower neck and upper back muscles acting on the 1st rib, but I haven't read anywhere of this sort of scenario where pushing up, even with bent or straight arms, would apply a sufficient direct force to the 1st rib. There are 5 reports of radiologically proven fractures in people who habitually lift heavy weights or can remember lifting a heavy weight in the past, but the direct causal link is tenuous. These fractures were characterised by being in the region of the scalene tubercle and not where Mr Bayoh’s fracture was found, hence making this cause less likely than shock loading.

Signature of Witness [redacted]

102. I have been asked if a first rib fracture would have caused Mr Bayoh to experience any difficulty doing a press-up from the pavement with the weight of a large police officer on his back. I can only offer an opinion from the perspective of the literature I have read. Mr Bayoh has a unique history. He is known to have drugs in his blood stream that I would have thought came under the general category of psychostimulants, the MDMA and the alpha-PVP. How that would affect pain perception, I'm not clear. It's said that fractures of the first rib are very painful. I haven't been able to find anything that suggests that they're more painful on movement, but you'd expect that they might be. I do note that in the Appendix A – Summary of Events of 3 May (SBPI-00306) that Mr Bayoh is reported as pushing up on his right arm and this might have meant he couldn't push up on his left.

103. How his perception of pain might be altered by the drugs that the toxicology suggested that he had been taking fairly recently before his death, I just don't know, but I suspect an altered mood state might have included experiencing reduced pain, but that's way out of my area of knowledge.

104. However, I wrote a paper with our chemical pathologists, years and years ago, about a man who was found dead, hanging off a high-tension electricity cable. He was found to have MDMA in his system. It was thought that climbing up to the height of a high-tension electricity cable was itself a bit of an achievement, but actually going along a high-tension electric cable until you were electrocuted sounds as if your perception of pain and your perception of the world around you can be significantly altered by MDMA.

105. Returning to Mr Bayoh, he may have been able to do the press-ups because he didn't feel the pain. Either wat, the fractured rib would not have affected his ability to do press-ups.

Use of Baton to Control Arm

106. I have been informed that the Inquiry has heard evidence that, during the restraint, PC Paton placed an extended baton across Mr Bayoh's left bicep in an attempt to control his upper arm. PC Paton was applying his full weight to his

bicep. If the Chair were to accept this evidence, I have been asked whether this could be a likely mechanism of injury to the first rib. Although I have no direct experience, from what I've read, the answer is likely to be No. Again, all the comments and all the descriptions seem to be indicate fracturing of the first rib requiring a narrow timeframe over which the force is applied – perhaps parts of seconds – and there's no doubt that a bone is much more likely to fracture if you apply the same force in a single element as opposed to gradually over time as PC Paton is believed to have applied the force from his baton..

Thumper

107. I have been asked if the use of a LUCAS machine, or "thumper", could have caused a fracture of the first rib. I've never really seen one of these machines in action, but I imagine that it's designed to mimic the effects of manual CPR. Therefore, I believe the description that I gave will probably hold for it as much as it would have done for CPR. In the sense that the first rib is a structure right up high in the chest at the bottom of the neck and the force is being applied in the middle of the chest, I can't envisage a situation in which that would lead to an isolated fracture of the first rib. Some of the forensic pathologists may have seen fractures induced by these types of machines, but I haven't. In my mind, I can't envisage how this would have caused an isolated 1st rib fracture.

108. And of course, if the "thumper" is used after death the changes I saw in the rib, particularly the haemorrhage and the osteocyte necrosis, would not have occurred.

109. I think key here is the <u>isolated</u> nature of the 1st rib fracture, and the appearance of the fracture.

Leg Restraints or Handcuffs

110. I have been asked if there is a likelihood that the fracture of the first rib could have been caused by leg restraints or handcuffs being applied. Again, whilst outside my expertise, I can't see that, no. Again, I'm only working off the

descriptions from the publications that I've seen but, again, this fracture was most likely caused by a rapidly applied force leading to either direct hitting of the shoulder or a force that's come up a straight arm. Even with the hands behind the back, I imagine that the forces would be rather different.

111. I have been informed that there is evidence before the Inquiry that Mr Bayoh punched his friend to the head, face and body. If the Chair were to accept this evidence, I have been asked whether this could be a likely mechanism of injury to the first rib. I think the implications from my reading of the literature are that it could, but it would have to be of an equivalent force for a man falling onto his outstretched arm.

112. I have been informed that Mr Bayoh's friend was taken to hospital, examined and then discharged. So, Mr Bayoh's friend was not knocked unconscious. I don't know what that tells us about the amount force that was applied but, obviously, had he been knocked unconscious, then it might have suggested that more force was applied. If Mr Bayoh's friend had been knocked unconscious or Mr Bayoh missed his friends head and hit the ground that might have helped assess the forces being applied but my understanding is we just don't know.

Timings

113. I have been informed the Inquiry has heard evidence that Mr Bayoh's altercation with his friend occurred approximately between 06.30 and 06.45; that police officers first arrived at Hayfield Road at around 07:20 and there was then an altercation between Mr Bayoh and police officers, followed by his being restrained before Mr Bayoh was reported to be unconscious at 07:25, and CPR commenced at 07:29 when it was observed that Mr Bayoh had stopped breathing. The thumper machine was used at some point following Mr Bayoh's arrival at hospital at 07:45. Life was pronounced extinct at 09:04.

114. However, in the ambulance and at the hospital Mr Bayoh was found to have a spontaneous pulse. At some stage in hospital, I don't know when, the

spontaneous pulse was lost and CPR recommenced. This generated systolic pulse waves between 70 and 140 mms of Hg.

115. Although receiving CPR in the ambulance there is an episode of circulation associated. This is described in Appendix A – Summary of Events of 3 May (SBPI-00306). I think from the description with electrical activity of the heart "*Finlayson and Taylor applied a defibrillator in the ambulance and obtained a trace of a pulse. They therefore did not apply a shock to Mr Bayoh*)"[11]. On reaching the hospital at 7.45 "… *Dr Pickering checked Mr Bayoh's carotid artery and found a pulse. She deduced that when the Scottish Ambulance Service had called the hospital, Mr Bayoh was actually in respiratory arrest*."

116. Once Mr Bayoh went into cardiac arrest CPR seems to have given a reasonable systolic blood pressure, probably sufficient to have given some perfusion to the tissues. This is important to assessing the value of the timings I gave to the age of the fracture which is given relative to the time of death. Death is defined as the cessation of a heart beat. Mr Bayoh's exact condition is not clear from the time the police officers thought he had gone into cardiac arrest and the time he reached hospital. Certainly, in the ambulance he is noted to have a spontaneous pulse as he had when reaching the hospital. He goes into cardiac arrest in hospital but it is recorded that CPR gives him a pulse with a reasonable systolic blood pressure between 70 and 140 mms Hg and he is being ventilated with oxygen until declared dead at 09.04.

117. When factoring these time points onto my aging of the fracture 07.29 is about an hour from the first altercation and 9-10 minutes from his first encounter with the police. At 07.45 these times had been extended to about 1 hour 15 minutes and 25 minutes respectively. By 09.04 the times had further extended to 2 hours 30 mins and 1 hour 45 mins.

118. If there is nothing of relevance that occurred to Mr Bayoh in the 6 to 12 hours before 06.30 that is anything that sounds like the sort of event that is described in

[11] Page 7.

the paper I quote as causing this type of fracture - falling on to an outstretched arm being bashed on the shoulder – not just somebody tapping you on the shoulder, it's going to be very hard – or you banging into a door frame with your shoulder but, again, not just staggering but really going for it, then the likelihood is that the event leading to his fractured first rib occurred sometime during his first altercation (with his friend 2 hours and 45 minutes before death) and his interactions with the police (1 hour 45 minutes before death). If we were to have absolute reliance on my timings the altercation with the friend is the more likely of the two, but I have already discussed how my timings might have been perturbed by the fact Mr Bayoh had been regularly taking anabolic steroids for several years. And tissue decomposition made aging the fracture more complex.

Pain

119. I have been asked if a fracture of the first rib would have caused pain. Again, I'm working from the literature, but they're said to be very painful, and the pain isn't described as being in the chest, it's rather more over the back that you are said to feel it, Moreso perhaps because the fracture is said to be nearer the back than the front. So, yes, it's supposedly very painful.

120. I have been referred to the e-mail from Kerryanne Shearer to Alasdair Macleod, dated 17 May 2017, which states:

"There is well defined, linear lucency in the medial, posterior aspect of the left 1st rib, proximal to its junction with the 1st thoracic vertebral body."

121. Yes, that's round the back. The rib is actually quite like a hand, broad and flat, and it curves quite a lot eventually joining the spine at the back. “Medial” would be in the middle, “Posterior” very close to where it joins the spine. It sounds as if it's even further back as the radiology says proximal to its junction with the vertebral body. Therefore, I think he could well have complained the pain over the scapula.

122. That's my reading of the literature, it isn't in my report but I was reading things recently and people say that individuals with a fractured 1st rib have a lot of pain and it's over the scapula, over the shoulder blade.

123. I have been asked if Mr Bayoh would be able to walk with pain like that. Whilst outside my area of expertise it might be argued that if people are able to walk if they fractured their collar bone and that's a common injury, particularly in young people, yes, I don't see that he wouldn't have been able to walk after a fracture of the first rib.

124. I have been asked if movement would have caused pain. I guess so, yes. Certainly when you move bone ends over one another, they hurt. I'm really wandering into areas that are outside my expertise, but I would have thought, just empirically, that moving his arm would have hurt.

125. I have been asked if the fracture and the pain associated with that would have impaired or impeded Mr Bayoh's breathing in any way. No. My understanding of the literature I have read, if you have any major symptoms from these fractures, it relates to the structures around the first rib, and although it sits on top of the lung, most of the effort of breathing comes from the lower ribs and the diaphragm. So, I don't think it would have. Of course, I have no feel for the amount of drugs that he had in his system, and what the effects of those drugs might be on recognition of pain, but that, again, it's obviously outside my expertise, but it's something that needs to be considered I guess.

CPR

126. I have been asked, in terms of the osteocyte necrosis, whether there's any difference in a patient whose heart is beating spontaneously and is breathing spontaneously and that of a patient who is in cardiopulmonary arrest and undergoing CPR. I think that's an interesting question, and one that I've considered quite a lot over the years. Although pathologists describe "osteocyte necrosis," it's really osteocyte apoptosis. As I said in my reports, it's the cells realise their environment has changed, beginning to lose an oxygen supply and responding to bony injury and therefore go into this lockdown of what's called "programmed cell death" or "cell suicide."

 [redacted]

127. The situation in relationship to CPR and tissue perfusion is complex. There's absolutely no doubt that CPR isn't sufficient to completely replace normal oxygenation in the periphery. It's designed to keep the heart contracting and to keep the big arteries supplied with blood.

128. There are two elements to CPR. First of all, CPR is designed to keep blood pumping to the major organs, and the second is respiratory/ventilator support to attempt to keep blood oxygenated. It is recorded that in Mr Bayoh's case CPR included for a while, attempted rescue breathing using mouth to mouth resuscitation and then in hospital respiratory support switched to full intubation and delivery of oxygen directly into the lungs. The object of respiratory support is keeping oxygen going into the lungs, and with the chest compressions keeping blood going through the lungs. We know that the amount of microscopic haemorrhage that you see in bony tissues following unsuccessful CPR isn't anywhere near as much as would have been expected had the person been alive during all that time. By "alive" I mean having their own heart beating, breathing and so on. What we don't know is with CPR how much oxygenated blood is getting through to the periphery, and in this case the ribs really are the periphery. They're right at the very end of the blood supply chain.

129. When people recover from CPR, I do not know of any evidence that suggests that there are bits of them that just die. The implication of that is that in successful PCR you have got enough blood going through for long enough to keep those tissues at least minimally oxygenated. It's balancing our knowledge that blood doesn't travel – that it's difficult to mimic haemorrhage in distal tissue at the far end of the blood supply chain by CPR – with whether or not anything gets through at all. I suspect that, from the fact that people just don't have organs die after they've done CPR, successful CPR means that there is some oxygenated blood getting through; but the tissues are very resilient, and they can survive a little bit without oxygen.

130. What is known in this case is that Mr Bayoh received from the descriptions I have seen very good, sustained and largely successful CPR. From these

descriptions I think there is oxygenated blood getting through to tissues as CPR includes compressions of the chest sufficient to detect a reasonable systolic pressure but also ensuring oxygenation through ventilating the lungs.

131. I have been informed that the Inquiry has heard evidence that rescue breaths were attempted, but Mr Bayoh's teeth were clenched shut and the officers could not fit the mask to give the breaths properly, so the rescue breaths were stopped and the officers just did the chest compressions, but at the same time Dr Pickering checked Mr Bayoh's carotid artery in hospital and found a pulse. She deduced that when the Scottish Ambulance Service had called the hospital, Mr Bayoh was actually in respiratory [not cardiac] arrest.

132. How CPR influences osteocyte apoptosis is something that I've found a bit of a challenge because I didn't know from my own experience and cannot find reference to this in the literature. In some ways, what I've found reviewing the literature from 2017 to 2023 is that, because Mr Bayoh was taking anabolic steroids, his osteocytes would've been pre-primed with a "cell stimulant" . I can't find anything that would suggest that, in itself, the additional androgens, would change the onset of apoptosis in a fit young man., , but it would not be a huge leap to think that it might. How CPR in the presence of exogenous anabolic steroids might have influenced microscopic osteocyte apoptosis is unknown.

133. From paragraph 94 to here I have given a set of long and complicated answers that really could have been said as "I don't know," I have said when something is outside my area of expertise but have also tried to help the Inquiry by using my knowledge and expertise to qualify why I cannot give a direct answer and placed the lack of knowledge into context.

134. What always was clear and has been further clarified by the further literature review I conducted is that this is a complicated case made more complicated by recent discoveries. However, I believe that the arguments I used and conclusions I came to in my final report in 2017, have not, in the most part, changed. The analyses have been made more complicated by: a relative paucity of information on what in the event is a rare fracture, a solitary fracture of the first rib; the effects

of post mortem decomposition; a lack of knowledge of the effects of the drugs Mr Bayoh was taking would have on fracture biology and his behaviour; the effects that CPR have on perfusion of fractured ribs (however the Inquiry have established that for a significant period from 07.29 Mr Bayoh had a spontaneous pulse and that when CPR was applied in hospital it established a good systolic blood pressure at a time when the lungs were being well, artificially ventilated. The new data I and the Inquiry have identified do show that the timing I gave initially for the closest the fracture was to death (2 hours) is likely to have been affected by the steroids even if how is yet unknown, that the fracture most probably occurred during the altercation with Mr Bayoh's friend or during his different interactions with the police and is unlikely to have been caused by CPR.

135. I have been referred back to page 8 of my final report, where I state:

"I cannot explain the sound reported during CPR but do not believe it was caused by the 1st left rib fracturing, for all the reasons already given".

136. I have been asked about the reference within my first report to CPR causing a sound if the rib was already broken. I think the first report was very much a draft, and there were things I was checking up on, but it is known that if you have a broken rib, it can cause a sound. Whether you would find that in a non-displaced fracture, I don't know. In a displaced fracture, movement of the bone ends over one another can cause a noise.

Radiology

137. I have been asked if it assists me to see skeletal surveys, x-rays and CT scans, or whether I rely on the radiologist's interpretation. Radiologists, because of their training, are very good at interpreting the appearances on imaging studies and I'm not. Even so, they had difficulty identifying the first rib fracture on the initial skeletal survey. It must have been a very subtle change that was only noticed later on. The reports from this type of case are usually checked by a radiologist of similar or greater experience. A senior radiologist would have an

infinite amount more understanding and interpretive skills than I would have. So, no, I don't think it would help for me to see them.

138. I have been referred to figure 1 on page 13 of my final report, and the associated text where I state:

"Macroscopic image of the 1st rib taken at time of postmortem examination. This was one of 4 images attached to an email entitled 'rib pictures, part 2'.

I have interpreted the dark red area (arrowed) as evidence of soft tissue haemorrhage associated with the fracture."

139. I have been asked what the soft tissue haemorrhage tells me. As I explained, CPR doesn't really lead to significant haemorrhage. In this image I believe you can clearly see soft tissue haemorrhage see haemorrhage over an area that extends beyond the immediate site of fracture. There's all the caveats about tissue decomposition but, nevertheless, to me the appearances suggest that this fracture occurred before CPR was applied. This is supported by the amount and extent of Glycophorin A staining and the presence of established osteocyte apoptosis.

140. I have been referred to the images COPFS-03737(a) and COPFS-03737(b), which are images of the rib taken from the post-mortem examination. I have been asked if I recall seeing these two photographs. I don't now remember, no.

141. COPFS-03737(a) looks like it's the other side of the rib, as if the one I have got has been turned over relative to that. If you look at the overall shape of the rib within COPFS-03737(b), it is very similar shape to that in my figure 1.

142. I have been referred to the image COPFS-03737(c), which is also an image of the rib taken from the post-mortem examination. I don't remember this image. I selected the image contained within figure 1 of my final report as I thought it gave the best image of the haemorrhage.

143. I have been referred to an e-mail from Kerryanne Shearer to Alasdair Macleod, dated 17 May 2017 at 12.21, which contains the images COPFS-03737(a), COPFS-03737(b) and COPFS-03737(c).[12] I have been asked about the image contained within figure 1 of my final report, which is not contained within this email and is referred to as being part of a set of four images. I can't remember receiving the first email, but I might have done, but this sounds like a second email, with another four images on, or maybe three plus one. I can't remember that much. It does sound as if there are other photos.

144. I have been referred to figure 2 on page 14 of my final report. I have been asked if this image shows H&E staining. Yes. That's just the shorthand that we say. It's a fracture site, stained using haematoxylin and eosin, and the bone comes out as pink because it's a proteinaceous material, and the blue, dotty things in the bone are residual nuclei or smeared nuclei of cells that have died or are dying. This is seen better in the lower of the two images where you can see something that looks a bit like a lacework in the middle, like a blue lacework. The white are fat cells, and the bits around them are other cells that you find in bone marrow, including the nuclei of the fat cells the remnant nuclei of blood cell precursors.

145. If you look right down in the bottom right-hand corner of that middle image, you can see little blue dots inside the bone. Those are viable osteocyte nuclei within the holes in bone or lacunae in which they live, whereas if you look at the bottom image, you can see that whilst the top arrow points to the same blue dots, which are viable osteocytes, the bottom arrow points to where there are empty osteocyte lacunae. The nuclei have been lost, the cells have died. Empty lacunae extend right across that area. Where the deep pink interfaces with the very pale pinky white, is actually the fracture line. The empty lacunae are all next to the fracture line. One of the things I take into account when looking for osteocyte necrosis is to see how many of those lacunae are empty. I don't make an accurate measurement but here you can see nearly all the lacunae next to the fracture line are empty of nuclei, this is what I mean by established osteocyte necrosis, or strictly osteocyte apoptosis.

[12] COPFS-03737

146. I have been referred to figure 4 on page 15 of my final report, and the associated text that states:

"On EVG there is no pre-existing structure."

147. I have been asked to provide more detail about this. EVG stands for elastic van Gieson. Elastic fibres are found in the walls of blood vessels particularly arteries because they give elastic recoil to the arteries. The van Gieson bit itself will stain up collagen fibres, and if you look at the top left-hand corner, you can see a very dark brown piece of tissue. That's bone, which contains a lot of collagen, and the little bits of black that are in the bone marrow, and just to the right, represent bits of bone that were forced into the bone marrow when the bone was cut. In the top right-hand corner, there's another bit of bone that was present before the bone was cut. I have already explained in paragraphs 70-77 that I asked for a set of special stains because in the decomposed tissues I needed to establish the extent of haemorrhage and the nature of lamellated structures in the marrow.

148. I have been referred to figure 5 on page 16 of my final report. I have been asked to talk through the three images contained within figure 5. There's two things we need to note on these slides. The first is the brown staining. The way that we look for glycophorin A is that we buy in an antibody that will stick to the glycophorin A protein wherever it is in the tissue. Now you can't see that, but the antibody has an enzyme attached to it that will convert a special substance that you put over the slides into a brown stain wherever the antibody has bound to glycophorin A.

149. The second thing to notice is that the top picture is a picture of very nearly the whole slide, and it's a little out of focus because of the way in which I have to take photographs at very low magnification, but I think it nevertheless shows large areas of brown colouration at the site of the fracture and extending into adjacent soft tissues.

150. In the left of the three pieces of bone on the top slide, you can see an area of brown going from top-right to bottom-left and there's also some bits on the left-hand side. The middle piece again is a whole piece of bone, and you can see the brown staining looking a bit like a V-shape about a third of the way down the piece of bone. Again, in the right-hand component, which is more out of focus, you can see the distribution of the brown stain. This is along the line of the fracture. This shows that there were a lot of red blood cells along the lines of the fracture. This staining is independent of whether or not the red blood cells are intact because it's just looking for their membranes, the surface that covers the cells. Even when the cells break down, those membranes stay in the same place. We can see a lot of brown staining indicating a lot of red blood cell membranes which I believe equates to a lot of bleeding in and around the fracture site.

151. The middle picture is a higher magnification image, and it shows what you would expect in normal bone marrow. The bone is pale – there's one piece running from the middle of the top to the middle of the left-hand side, for instance, and there's another piece at the bottom running almost parallel to the piece at the top, and one or two other areas which are bone which don't show staining. Between the pieces of bone there are areas of bone marrow. Red blood cells are made in bone marrow, and near the bottom piece of bone, which runs from quite close to the bottom of the right-hand side of the image to the middle of the bottom of the image, there's also a piece of bone that curves upwards and round towards the centre of the image, and the top arrow points to relatively normal bone marrow where you can see the red blood cells and the fat cells which are small white holes. Above and below that area there are four or five much larger holes. These come as a consequence of gas-forming organisms which are part of the process of decomposition, but nevertheless we can show that the stain has worked because it is picking up where the red cells would normally be made within bone marrow.

152. The bottom picture also shows staining of bone marrow and, in particular, it shows this staining at the microscopic level through the fracture site. The bottom arrow points to what I've called the necrotic debris within the fracture gap. The fracture gap is marked by that line of brownish staining that runs from the bottom

left-hand corner to the top right-hand corner of the bottom image. You can see at a microscopic level the same features that we looked at in the top image.

Further testing

153. I have been asked if, with the passage of time, I would recommend any further testing. I don't think so. I'd asked for a set of stains to be done as part of a protocol recommended by different, largely American, authors in this setting. I attached a paper to my final report in 2017 that I have found helpful, particularly in my work in Biomedical Egyptology. I am of the opinion they gave the answers to my questions. I don't think the tissue, particularly after it has undergone this degree of degradation, is likely to yield anything more.

154. 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 20, 2023 | 11:41 AM BST

Signature of Witness ..... [redacted]