Wednesday, 24 May 2023

(10.00 am)

LORD BRACADALE: Good morning, Dr Cary.

THE WITNESS: Good morning, sir.

LORD BRACADALE: Ms Grahame, senior counsel to the Inquiry, will take your evidence. Before that will you say the words of the affirmation after me please.

DR NATHANIEL CARY (affirmed)

LORD BRACADALE: Thank you. Ms Grahame.

Questions from MS GRAHAME

MS GRAHAME: Thank you very much.

Good morning, Dr Cary.

A. Good morning.

Q. You are Nathaniel Cary?

A. Yes.

Q. We have heard some people call you Nat?

A. Yes, I am more than happy with that.

Q. I won't be calling you Nat today, but if we have heard evidence of a Nat, that is you?

A. It is.

Q. We have your professional address and contact details so there is no need for you to give those today.

A. Thank you.

Q. Am I correct in saying you are a consultant forensic pathologist?

A. That is correct.
Q. I am going to deal with your experience and your qualifications in a moment, I would like to go through those with you, but can we look, first of all, just at a couple of statements that you have given to the Inquiry.
A. Yes.
Q. The first statement was taken from you on 17 November 2022.
A. Yes.
Q. If we can have that up on the screen, while you look at that, you will see that this document is appearing on the screen, and we can work through that today and we can look at that, anything -- any documents on the screen. In addition there is a blue folder in front of you, and that contains hard copies of the documents we think you may want to have regard to today.
A. Thank you.
Q. You must feel free to use any of the hard copies that you wish, or if I am referring to something it might be on the screen. But if you think there is something else that we should have on the screen, please let me know --
A. Thank you.
Q. -- and we will make sure that is up there. If we can also look at -- I will come back to the statement in

a moment, but if we can look at your CV, you provided us with a brief CV, WIT 00037. We will see that this is a brief curriculum vitae for yourself, and you are a Home Office registered consultant forensic pathologist.

A. Yes.

Q. Are there many Home Office registered consultant forensic pathologists?

A. The list is usually about 35 to 40. But that is for England and Wales, of course there are equivalent forensic pathologists in Scotland and in Northern Ireland.

Q. What sort of cases do Home Office appointed pathologists do?

A. Primarily what would be called suspicious deaths, so those are cases where there may be some form of third party involvement. That could be direct third party involvement or we get involved in things like manslaughters, involuntary manslaughter, all that sort of thing. So there is a wide range of casework to cover.

Q. Were you involved in the Litvinenko case?

A. Yes, I was, which was obviously a fairly unusual kind of a case.

Q. Am I correct in saying you had to carry out your

post mortem in a big white suit to protect you?
A. Yes, we had full protective clothing including ventilated hoods.
Q. So you had to be protected from radiation yourself?
A. Yes, and we had a radiation protection officer with us at all times to make sure the levels never went up to anything unacceptable.
Q. Am I also right in thinking you have also been involved in a number of high profile cases including the Soham murders?
A. I have but it happened to be that I was just the person on call at the time, so I don't particularly seek out such cases, they just happen.
Q. Right. Am I right in thinking you have also been involved in the Hillsborough disaster?
A. Yes, so the re-inquiry into that, a few years ago now.
Q. Where people suffered crushing injuries?
A. Yes, they had crush asphyxia in a crowd and in fact we published a paper after that which showed the range of what you might see after a crush experience.
Q. Thank you. Tell us about the work that you do as a forensic pathologist, about the experience you have?
A. So much of the work would involve carrying out a post mortem examination, and then commissioning further tests. That is not always the case, but there

might be no post mortem examination, there might be no body, so we might just give expert opinions as forensic pathologists in a whole variety of circumstances but most of it would involve carrying out a post mortem examination.

Q. So you are doing the post mortems yourself?

A. Yes, exactly.

Q. Do you do what we have heard referred to as a double-doctor post mortem? I think the position in England is slightly different?

A. It is, although I introduced that in relation to paediatric cases in England some years ago now because I think the range of complexity in a paediatric case with natural disease as well as an important factor, especially in babies, I felt, and others felt, that it would be worthwhile to have a paediatric pathologist there as well. So I am familiar with having a double-doctor system, which in my opinion is a very good system, maybe we should think about it in England.

Q. And that is something you helped to introduce in England?

A. For paediatric, yes.

Q. Paediatric being babies and children?

A. Babies and children.

Q. As well as that aspect of your work, do you also do

a lot of medicolegal reports and opinions?
A. I do, although probably less so now.
Q. Can you help the Chair understand the sort of number of reports that you have prepared, medicolegal?
A. Yes, of course. It runs into the hundreds, it is always easy to exaggerate but it probably runs into the hundreds and then of course there will be the full post mortem reports, and over the course of many years I have carried out about 30 to 40 post mortem examinations and some years I have done more.
Q. How many years have you been working in this field of forensic pathology?
A. I have been on the Home Office list since 1992.
Q. Prior to that were you also working in this field?
A. Prior to that I was working -- I continued to work for some years as a consultant pathologist, carrying out diagnostic pathology. My role was particularly as a specialist heart pathologist, and that is when I worked at Papworth Hospital in Cambridgeshire and I had responsibilities for transplant pathology and other aspects of heart and lung pathology.
Q. And all of this work as overlapped throughout your career?
A. Exactly, but no I don't -- I am not involved in transplant pathology anymore.

Q. What experience do you have of handling post mortems involving -- or suspicious deaths involving deaths arising out of restraint?
A. A lot, I would say. I have had a particular interest in that really throughout my career, and so in a whole variety of circumstances I have been referred cases often as second or even third pathologist.
Q. What sparked that particular interest?
A. I have always been interested in the interplay between physiology, so how the body systems work and how they go wrong when something goes wrong, like death during restraint, and what might be the possible causes of that.
Q. When did your interest in this actually start? When was it sparked?
A. Probably from the very outset of my time working as a Home Office pathologist, if not shortly before.
Q. So early 1990s?
A. Yes, exactly.
Q. Were you involved in a case involving a Dennis Stevens who died in Dartmoor?
A. Yes, I was.
Q. Can you tell us a little about that case?
A. Yes. So that involved restraint, it also involved him being left on a hard floor face-down for a very long

time, and he suffered from sickle cell disease I think, or sickle cell trait, I cannot remember which he had. But anyway his death was characterised by a lot of sickling, which is blockage of little blood vessels really throughout the body by the red blood cells, which should normally be round but they become long, sort of boat-shaped and that then leads to them blocking up little blood vessels.

Q. Was that something that could be seen pathologically?

A. Yes, indeed.

Q. We heard yesterday from a Professor Lucas who talked about small blood vessels, particularly those into the lungs, can get blocked through the sickle cell; is that the type of thing you are talking about?

A. Absolutely, yes. And there are other major organs where it is really very damaging to have small blood vessels blocked. Actually I have worked with him over many years, including on sickling cases.

Q. Thank you. That is Professor Lucas?

A. Professor Lucas.

Q. Can I ask, have you given evidence before in courts or inquiries?

A. Many times, yes. In a whole variety of circumstances. I have actually given evidence in Scotland before, in fatal accident inquiries.

Q. Was that involving deaths?
A. Yes.
Q. Could you tell us, are you -- you have told us about your Home Office work, are you predominantly instructed by prosecutors or defence?
A. Sometimes defence, sometimes just interested persons of one sort or another. So I get a whole range of practice.
Q. Do you become involved as an expert in civil cases as well?
A. Yes, on occasion. And the family -- because I do quite a lot of paediatric forensic pathology, I am involved in the Family Court quite a lot.
Q. So not just criminal but civil matters as well?
A. A whole range of things, you always have to keep at the back of your mind different burdens of proof in the different circumstances.
Q. I think in terms of your involvement in relation to the death of Mr Bayoh, just to be clear, you didn't carry out a post mortem --
A. No, I didn't.
Q. -- on Mr Bayoh and you weren't present at the post mortem carried out by Dr Shearer and Dr Bouhaidar?
A. No, I wasn't, although I do know them both, and I would be confident they would be very careful pathologists in

terms of evidence of fact collection, which is the first stage really, opinion comes later, but the facts need to be collected in any suspicious death.

Q. Thank you. If we could look maybe at your letter of instruction, 18 May 2015. This is AAC 00377. I don't need to go through in this detail but this was a letter of instruction to you, Dr Nat Cary on 18 May 2015 from Aamer Anwar & Co?

A. Yes, who sits behind you I think.

Q. He does usually. So this was a letter of instruction sent to you by Mr Anwar, and you accepted that instruction. Can I ask you what contact you did have with his firm round about this time, in addition to your letter of instruction?

A. I am not sure if I did, I may have done though, if there is evidence I did, then I would accept it.

Q. I am just -- I'm only asking the questions.

A. I think obviously the letter arose from a telephone call originally, I think Mr Anwar rang me personally, so the letter follows on because in any circumstance I like to work to clear instructions so that we know where we are both coming from.

Q. Did you have any meetings with the family themselves?

A. No.

Q. Did you ultimately meet with the lawyers for the family,

from Mr Anwar's firm?

A. I am not sure, we may have spoken on the telephone. I don't remember a trip to Scotland though to meet them.

Q. Thank you. I just want to deal with this very quickly. There may be some people listening who have concerns that you were instructed by Aamer Anwar, and consider that maybe this may have influenced you in some way when you were approached by them initially. Do you have any concerns yourself about --

A. No, not at all. I mean obviously in the course of my work, particularly getting involved in deaths in custody, if you are asked to act on behalf of a family -- and fortunately there is increasing support for families in such circumstances but I can tell you ten or twenty years ago there was very little support -- you expect to be instructed by specialists in human rights really, so that was not in any way a surprise.

Q. Just to be clear, do you understand that your involvement with the Inquiry we wish you to help the Chair primarily?

A. Yes, absolutely I do understand that. Which is -- actually in any circumstance my overriding duty is always to a court or of course sometimes there won't be a court appointed at the time but it is to a potential court in the future. And I am very mindful of that.

Q. Do you bear in mind that you -- it's important that you are independent and objective?
A. Yes.
Q. And that you are not biased in favour of those who give you instructions?
A. No, not at all.
Q. Do you take care to avoid extra that?
A. I do take care and it is very easy to fall into the trap of telling people what they want to hear really, so I am very mindful of that.
Q. And you avoid doing that?
A. Cognitive bias has been increasingly recognised recently in a whole range of forensic scientific and forensic pathological settings, that you might just say it is that because you are somehow involved that whole area yourself, so I am very mindful of that. The trouble is with forensic pathology in particular you have to know the history, it's no good trying to do it shut in a box because if you don't know the history, you may not make correction decisions at all.
Q. We heard evidence from Dr Shearer at the beginning of this hearing and she explained it was very important to consider the full background circumstances?
A. I agree.
Q. Do you agree with that?

A. I agree.
Q. Thank you. Can we look now, please, at a report you prepared on 23 October 2015, which is COPFS 00196. You will see your name is at the top?
A. Yes.
Q. Your qualifications and titles. And then it says:
"Statement of witness."
As I understand it, this is effectively your report presented as a statement. And further down --
A. Yes.
Q. -- if we carry on down it does say, "Expert opinion", I think?
A. Yes.
Q. On that page we have just passed, that contains your qualifications and experience.
A. Yes, exactly.
Q. Which will enhance the brief CV we looked at moment ago and the Chair can consider that later. And then if we carry on down the page you then give a declaration understanding you owe an overriding duty to the court to provide independent assistance, by way of unbiased opinion, in relation to matters within your expertise?
A. Yes.
Q. And such advice must be uninfluenced by the exigencies of the case, and you have complied with and will

continue to comply with your duty to the court?
A. Yes.
Q. And that would apply to the Chair of this Inquiry also?
A. Very much so, yes.
Q. Then it is headed up, "Expert opinion", to Aamer Anwar & Co, solicitors. Then if we move down we then see that this is really where you go through your opinion on the matter, which has been based on a number of documents and information that has been sent to you by Mr Aamer Anwar's firm?
A. Yes.
Q. Thank you. We will come back to your report in a moment. Let me turn first of all to your contact with this Inquiry. I think you were contacted last year on behalf of the Inquiry?
A. Yes, I was.
Q. And you provided us with -- you have provided with us two statements, the first SBPI 00268. This is your first statement. And it was taken on 17 November 2022, and if we look at the last page, it's 58 pages long, we can see it was signed on 23 February 2023.
A. Yes.
Q. I think, although our version on the screen is redacted, your hard copy will show your signature?
A. Yes, thank you.

Q. And it's on every page, I think you signed every page?
A. Yes, every page.
Q. The final paragraph, if we can see that on the screen, at 232 says:
"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."
A. Yes.
Q. Did you understand that to be the case?
A. I did understand that, which is very sensible, being open and transparent.
Q. Thank you. Let's look at the second statement you have provided to the Inquiry, which is 00269. This was taken on 17 November also. On the last page we will see it is also signed on 23 February 2023. It is only four pages long, and the final paragraph, 15 is in the same terms as previously?
A. Yes.
Q. And again, your signature on this document, which we can see at the bottom of the screen now redacted, was given in the knowledge that this could again form part of the evidence available to the Chair --
A. Yes, exactly.
Q. -- to consider?

A. I was wondering originally if I would be asked to get involved in these proceedings because obviously I had been involved at the beginning and I was pleased when I was contacted really because I felt I had something to add.

Q. Good. That is excellent. Can we turn back to your report then and we can go through this and have a look. COPFS 00196. Let's go back to the documents you were sent. So this was following on with your letter of instructions, so this is 18 May 2015, it was at an early stage in the events, and you were sent a large number of documents to consider --

A. Yes.

Q. -- involving correspondence, a report and post mortem report prepared by Professor Busutil, you had a copy of the post mortem report from Dr Kerryanne Shearer and Dr Ralph Bouhaidar, you had the final post mortem report and neuropathology report by Dr Smith, toxicology reports, and various other documents: radiology, photographs, stills, and information from the PIRC?

A. Yes.

Q. You were then also sent disclosure statements, you were sent civilian statements, and other statements that were copied to you?

A. Yes.

Q. Since then, I think very recently, you have also had sight of GP and medical -- hospital records of Mr Bayoh --

A. I have.

Q. -- to consider?

A. Primarily GP records. I was aware, I think from Dr Bouhaidar and Dr Shearer's reports, of some of the hospital records.

Q. Thank you. Can we look at page 3 of 6 now, please, on your report. You say:

"This opinion will be in the form of a commentary on the final report of Doctors Shearer and Bouhaidar."

So was this effectively what some may call a desktop report?

A. Yes, exactly.

Q. Where you have looked at paperwork and you have then prepared your own views on the basis of that?

A. Yes, I also think -- I mean, in my practice I try and not use huge amounts of other people's work, what -- my opinion is that people need comment on that really, it is much more helpful, otherwise you end up with a 50-page statement.

Q. So the background, that was from information provided to you?

A. Yes.

Q. And then you talk about external findings?
A. Yes.
Q. And I think is it correct to say that the information about the external findings would have come from Dr Shearer's post mortem report?
A. Yes, but I think I was able to confirm some from photographs.
Q. So a combination of looking at the photographs, and also considering the reports by Dr Shearer --
A. Yes, exactly because you have to remember the photographs are actually still original evidence, providing there is a good chain of proof from the photographer, they actually count as original evidence quite apart from what Dr Shearer says descriptively.
Q. So you are not just simply accepting what is written down in the report by Dr Shearer?
A. Exactly right.
Q. You are actually confirming, by looking at the photographs --
A. Yes.
Q. -- what is said?
A. Yes, which is very important when you do a desktop exercise, you want as much original evidence to review as possible.
Q. If we can move down the page, please. You then talk

about the internal findings, so that was during the post mortem again. Did you have photographs of internal findings as well as what was written in the post mortem report?

A. Yes.

Q. So again, did you do a cross-check and consider the internal findings in light of those?

A. Yes, fortunately, as I would expect from experienced forensic pathologists, there was good documentation in terms of photography along the way.

Q. Thank you. Then if we can look at page 4. We heard evidence from Dr Shearer that she carried out or instructed further investigations, and I see there you have detailed radiology, histology, and so she has given evidence about all of those things?

A. Excellent.

Q. Did you also consider her information about those and the reports that she received?

A. Yes, absolutely. It is all potentially relevant.

Q. So, as part of your consideration of the position, did you look through the reports by the individual specialists who had carried out tests and further investigations in relation to Mr Bayoh?

A. I did. In terms of the histology, so looking at the samples of organs under the microscope, I carried out my

own examination of the same material.

Q. So I was going to say there you say:

"I have examined material prepared for microscopy from multiple tissue samples taken at the first post mortem examination."

So were these slides that were sent to you?

A. Yes, exactly. So that is Dr Shearer accession number quoted F15-542, so that is a unique reference to those tissue blocks for that case.

Q. So you were able to not only look at her reports and the photographs but you were also able to look at the actual tissue itself?

A. Yes, exactly. It is something that obviously I have quite a lot of experience of as a -- not just a forensic pathologist but also having worked as a diagnostic pathologist, we are the sort of pathologists who make a diagnosis of cancer and you have to make sure that you get it right.

Q. So that is something you have experience of, even prior to you becoming a forensic pathologist?

A. Yes. That is -- the primary speciality I was trained in was diagnostic histopathology.

Q. You say you were also provided with a blocking diagram of samples taken from the heart?

A. Yes, which is very helpful because some bits of the

heart perhaps matter more than others, so if you know where the actual samples have been taken from, that are then processed to produce microscope slides, you can get a picture of the whole heart.

Q. And that was further items that were sent to you from Dr Shearer?

A. Yes, exactly.

Q. That you could consider yourself?

A. So that was the paperwork that came with it.

Q. Then you have indicated here:

"My own examination reveals the following ..."

A. Yes.

Q. I would like to just go through that. You have briefly identified what your views were, having carried out that examination. Tell us what you found in the heart?

A. Yes. I will try and unpack this a bit because it is in sort of histology speak really. It's not very understandable unless you are another histologist or perhaps a reporting clinician. So the left ventricle, these are the first sections that I refer to, that is the main pumping chamber of the heart. To all intents and purposes it behaves like a circular structure, so it's a complete encircling pump, and those sections of that left ventricle showed some variation in the size of the individual cells, those are called the myocytes,

and some of them were a little bit big. But that is not a very uncommon finding, to have that, and it is not really of any consequence.

I noted there was a section, there was a small area of the acute responding inflammatory cells, these are called polymorphs, but that was only one small focus which was likely to be insignificant, it might relate to the sort of final phase of resus, et cetera.

However, the section of the left ventricle there was no evidence of scarring, which would be important. If there has been any previous damage to the heart, then it will scar, in a whole variety of circumstances. There was also no evidence of what is termed the myocyte disarray, so these are the heart cells instead of being nicely packed together and organised they are all rather random in a disorderly fashion and that can give rise to a diagnosis of a condition associated with sudden death which is called hypertrophic cardiomyopathy. No evidence of that here.

Finally in relation to the heart, the right ventricle, which is the lower pressure pumping chamber on the right side of the heart, and that pumps blood to the lungs, that all appeared to be completely normal.

So this is a normal heart really and I think the naked eye description of the heart by Dr Shearer was

normal, and microscopically it is normal. So this was not a death due to heart disease.

Q. We have heard detailed evidence from Dr Shearer in relation to the examination she carried out regarding the heart, including microscopic examination, and she, if I am correct in saying, she said the heart was normal?

A. I agree with that.

Q. You agree with that?

A. Yes.

Q. And no signs of previous scarring or damage to the heart that would be indicative of any problems?

A. That is correct.

Q. Thank you. Then lungs. You talk about some congestion and patchy oedema. Could you tell us --

A. Yes, so that tends to be the final common pathway in any form of death, the heart just gets -- sorry, the lungs get gummed up with blood, and then fluid moves out into the lung spaces, that is called oedema. So it's a very non-specific finding in a whole range of circumstances so it doesn't give the cause. The only other abnormality I referred to there is the pleura, the lining covering the lungs, was slightly thickened and that is non-specific but might be indicative of a previous chest infection.

Q. So --
A. I have a feeling from the GP notes he had been treated for a chest infection previously.
Q. Thank you. But not an infection that was there at that time?
A. No, all resolved, or resolving.
Q. Would slight pleural thickening cause any ongoing problems for anyone?
A. No.
Q. Then you say:

"... sparse subpleural lymphoid infiltrate."

Can you explain what that is?
A. That is part and parcel of the thickening of the pleura, it has gone along with some residual chronic inflammatory cells so no longer the acute reaction but just left with something smouldering on from maybe months to years before.
Q. Would that cause anyone any problems?
A. No, it wouldn't.
Q. Then you say liver and kidneys, severe congestion for both. Can you explain that?
A. Yes, so that is really again entirely non-specific in a death. It's just when the heart terminally fails some organs get more gummed up with blood than others and it is very common to see it in the liver and kidneys.

Q. And then pancreas, you say autolysis. What's that?
A. So that is just the breakdown after death of the tissues, and of course the pancreas is full of digestive enzymes, it is one of the things it does, it makes digestive enzymes. So of course, once you have died they will start just digesting the pancreas itself.
Q. Is that a normal --
A. Another entirely normal finding, yes.
Q. And then adrenals: congestion?
A. Yes, so these are glands that produce adrenaline amongst other things, and again it's the same post mortem effect of being gummed up with blood.
Q. So, so far nothing out of the ordinary in relation to a post mortem?
A. Exactly, and also nothing that might account for sudden death.
Q. And then you say:

"Thyroid: Congestion. Slightly nodular architecture. No focal abnormality."

A. Yes.
Q. Explain that, please?
A. So he has a -- so again more congestion but the architecture of the thyroid is slightly abnormal in that it's nodular, which is very common as a sort of incidental autopsy finding.

Q. Again, nothing that you were able to find that would have explained his death?
A. Exactly.
Q. Bone, tell us any --
A. So I defer to the bone expert on this, who I think has already given evidence.
Q. We have heard from Professor Freemont, who is an osteoarticular pathologist.
A. Yes, so mine is the most basic of descriptions there and I would defer to him on any opinions about fracture and when it might have happened, and in what circumstances.
Q. Would you defer to Professor Freemont in terms of mechanism as well as timing?
A. Yes, I think what I would say is that the rib, I think it was the first rib ...
Q. It was the first left rib.
A. Yes, so we see resuscitation fractures very commonly when someone has been vigorously resuscitated but they are almost always ribs 2 downward, not the first rib. That is somewhat protected by the collarbone from being fractured in those circumstances, so that may well be a significant finding. He then found something quite subtle, which I'm afraid is beyond me, which is called osteocyte necrosis, which means the bone was alive at the time it was fractured, and that I think is a very

important finding.

Q. But you would defer to him in relation to his views on that?

A. Completely I would.

Q. Thank you. Can we move on, please. And then at the end you say brain congestion?

A. Yes.

Q. Can you give us a little bit of a description about the significance of that?

A. Again, it is entirely a terminal finding. I think there is full neuropathology in this case.

Q. And then you say:

"These findings are in keeping with those described by Doctors Shearer and Bouhaidar."

A. Yes.

Q. So was there anything at this stage that you found that caused you any concern or where you conflicted with anything that the pathologists who had carried out the post mortem found and reported in their final report?

A. No, so no conflict.

Q. No conflict there. Just taking that as it is, you have looked through their report and you have looked at the slides and done your own examination of what you have been sent, including the photos. Is it fair to say that on the basis of that you were able to exclude underlying

natural disease?

A. Yes. As is often the case with a post mortem examination you can say more about what isn't there as opposed to what is. But we were able to exclude a lot of things that weren't there.

Q. We've heard evidence, detailed evidence, from Dr Shearer in relation to the microbiology.

A. Yes.

Q. And she said that there was no evidence of any infective process or infection that could have caused his death?

A. Yes.

Q. You agree with that?

A. I do agree with that, although I didn't specifically reassess that.

Q. No. She talked about the neuropathology investigations that were -- further investigations that were carried out by Dr Smith --

A. Yes.

Q. -- and said there was no evidence of any brain infection.

A. Yes.

Q. She said that the findings in relation to the post mortem were consistent with cardiac arrest and prolonged resuscitation.

A. Yes, absolutely.

Q. Would you agree with that?
A. I agree with that.
Q. So you did consider whether -- Dr Shearer did consider whether there was an underlying heart condition.
A. Yes.
Q. She gave very detailed evidence about that. Was there anything that you could see that would indicate there was any underlying problems with the heart?
A. No.
Q. From what you looked at, could you see -- or did you -- were you concerned that Dr Shearer had missed anything or not done that assessment adequately in any way?
A. No, I thought it was a thorough post mortem examination, and of course it was helpfully corroborated by Dr Bouhaidar.
Q. Thank you. Then we had heard from Dr Shearer about when she looked at the external and internal findings from her post mortem, that the external and internal injuries were minor, and there was no evidence of a direct traumatic cause of death. She talked about sometimes she can see a stab wound to the heart, that is a very big explanation for someone dying.
A. Yes, absolutely.
Q. But there was nothing like that here, she said.
A. No, nothing at all.

Q. Would you agree with that?
A. I agree with that, this was not a primary traumatic cause of death in the sense of lots of fractures or letting of blood from a wound.
Q. In relation to the neuropathology again, I think Dr Shearer gave evidence there was no evidence of traumatic brain injury. Would you agree with that?
A. Yes, which is important. So no brain inflammation but no traumatic brain injury either.
Q. Then we also heard evidence from Dr Shearer that there was nothing that she found during the post mortem that would have indicated that CS or PAVA spray had any bearing on cause of death.
A. Yes, that is correct.
Q. Would you agree with that as well?
A. I would agree with that, it is used quite a lot but it really is fairly benign, otherwise I suspect it wouldn't be licensed so much around the world for use. It can cause problems, obviously it is very uncomfortable for the eyes and in vulnerable individuals it can cause what in effect is the equivalent of an asthma attack, but I don't think it is that sort of a case in any case, and there were no underlying findings for asthma in the lung to suggest he suffered from it.
Q. So we heard from Dr Shearer that there was no signs of

any pre-existing issues with his respiratory system.

A. I would agree with that.

Q. Would you agree with that?

A. Yes.

Q. And also that she wasn't able to find any reference in the background materials she had that there were any reports of Mr Bayoh wheezing or being breathless or very breathless.

A. No, quite.

Q. Would you agree that there was nothing of that sort?

A. Yes, which would be a good measure of him getting into breathing difficulties.

Q. Dr Shearer explained it is very important not just simply to look at her findings in a vacuum but to consider the background circumstances, for the Chair to look at everything and put it together. Do you agree with that?

A. I entirely agree, so everything is relevant potentially including, you know, what is going on at the time that cardiac arrest occurred. That is often the case in all sorts of deaths, what is actually happening to the person immediately before, at the time they go into cardiac arrest and afterwards, and that gives you a bit of a clue as to what has gone on.

Q. So the timing, what is happening at the time when

someone goes into cardiac arrest, would you describe that as crucial?

A. Yes.

Q. I think Dr Shearer said it was crucial.

A. I agree.

Q. Then Dr Shearer also gave evidence in relation to her post mortem report and she mentioned excited delirium.

A. Yes.

Q. She talked about the up-to-date position that has been intimated from the Royal College of pathologists.

A. Yes.

Q. You are in the Royal College of Pathologists, are you a fellow?

A. I am a fellow.

Q. That is a more senior level than member, isn't it?

A. Yes, although I think if everybody -- nowadays if they pass the final exam they become fellows, but when I did it you were a member first and then a fellow. I think I should say that I was involved in some of the advice given by the College to the Forensic Science Regulator on this issue, because we felt it was an entity that had been manufactured in North America, and was really never an appropriate cause of death. It describes a mode of behaviour. I hope this Inquiry has heard from a very senior psychiatrist on that point.

Q. We have heard -- Dr Shearer very helpfully took us through the Forensic Regulator's guidance --
A. Yes.
Q. -- from the Royal College of Pathologists, and we have also heard from Dr Lipsedge, a consultant psychiatrist who has talked to us about the history of excited delirium.
A. Yes, absolutely, it was very helpful. I was involved in advising the Forensic Science Regulator on that issue because I sat on one of the committees of the Forensic Science Regulator.
Q. So if we have heard from witnesses that excited delirium, at least in this country, is not a cause of death, pathologically it is not something that can be tested for, would you agree with that?
A. I would agree with that but I have never thought it was viable. I won't go into the complexities of North America -- and I am including the USA and Canada -- versus this country or the United Kingdom in general. But it simply in my opinion is incapable of accounting for death, it simply describes a behavioural disturbance in someone who is drug intoxicated often, you can see it with acute psychiatric illness. But the only way in which drugs can cause primary death in those circumstances, or the main way, is through what is

called hyperthermia, so their body temperature rises and all their body systems don't function properly.

Q. Thank you. I would like to move on and turn to the factors that at least Dr Shearer, at the time of her final post mortem report, highlighted and considered were significant factors in relation to an explanation of why Mr Bayoh died.

A. Yes.

Q. At the time of her final post mortem report, which was prior to the testing on sickle cell which we will leave for now, the two factors that she considered significant were toxicology --

A. Yes.

Q. -- and the restraint.

A. Yes.

Q. I would like to talk about those with you, if I may.

A. Yes, of course.

Q. So in relation to the toxicological findings, we can see from the report on the screen that you had considered the detailed findings from a number of blood samples that had been taken from Mr Bayoh. Dr Shearer had explained they came from hospital samples and also post mortem samples. You have identified there that there were positive findings of alpha-PVP and MDMA at different levels, which were noticed in the

toxicological report?

A. Yes.

Q. In addition, there was the presence of MDA, which you say either is a primary administered drug or a metabolite, and I think Dr Shearer gave evidence it was more likely to be a metabolite. Would you agree with that?

A. Yes, I would accept that. It is just that sometimes when people get hold of these drug substances they are not pure in the first place, so you can have both present in one administration.

Q. You say:

"... HDMA and HMMA as metabolites both identified in the urine."

A. Yes.

Q. "Analysis of a urine sample for androgens and synthetic anabolic steroids revealed the presence of nandrolone and metabolites consistent with recent administration of the anabolic steroid nandrolone."

A. Yes.

Q. That is something that you noted also?

A. Yes.

Q. Did you see any reason not to accept that toxicological evidence?

A. No, not at all.

Q. In relation to the nandrolone, we have heard that that is a steroid?
A. Yes.
Q. And I think in relation to the recent administration Dr Shearer gave evidence -- she was asked to explain what was meant by recent administration and she said:
"In the previous kind of few days to a week or so. Steroid use is not something that we see as acutely causing people to die, what we can see is long-term effects of it, chronic changes where we can get heart damage, which we didn't see in this case."
A. Yes, I agree with that.
Q. Do you agree with that as well?
A. Yes, and in fact these drugs, I'm afraid, are misused on a grand scale internationally and we don't -- we would -- if there were lots of deaths arising with that as the primary cause, we'd see it.
Q. So if it had been abused and caused long-term chronic changes, that would have been obvious from the post mortem?
A. Correct. That would have manifested itself through the heart, so the heart getting much too big or the heart muscle getting too big, the little cells.
Q. Dr Shearer said the heart was normal sized and you have explained to us what was found by her and you. As

I understand it, you found the heart to be normal?
A. Yes, I agree with that.
Q. Then we have also heard evidence from Professor Lipsedge, who has talked about the impact that these stimulant drugs have on a person's state of mind, that is the MDMA and alpha-PVP.
A. Yes.
Q. He described psychostimulant intoxication resulting in psychosis and thought alpha-PVP would be the most significant --
A. Yes.
Q. -- in that regard.
A. I would definitely defer to him on that one.
Q. Would you?
A. But he has a lot of -- particularly from the past he as an awful lots of hands-on experience of treating people in a very disturbed state and having to deal with them as a consultant psychiatrist, so he is very, very experienced at the coalface, if you like, of acute behavioural disturbances of one sort or another.
Q. We have also heard evidence from a Professor Eddleston, who is a toxicologist and an expert in these matters, and he talked about these alpha-PVP being more likely to cause even psychosis.
A. Yes, I would defer to him on that.

Q. He also expressed the view in evidence that nandrolone may give you higher blood pressure, and may make your heart more sensitive to having dysrhythmias. Do you agree with that?
A. Yes, although the main evidence that that was likely to have occurred to an extent would be from enlargement of the heart really, and scarring of the heart. If there wasn't any evidence of that, I would say that he might have been more vulnerable to heart rhythm disturbances but not excessively so such that you would actually see pathological changes.
Q. Professor Eddleston said there was nothing present at the post mortem --
A. Correct.
Q. -- that he could see that would be indicative of problems such as you have described.
A. Yes, absolutely.
Q. Would you agree with that?
A. I would agree with that.
Q. The Chair has a report available to him from a Professor Crane.
A. Yes, another person I know quite well.
Q. I believe he is also a forensic pathologist?
A. He is, he is very recently retired but he is still giving opinion.

Q. Thank you. He has said:

"The combined effects of MDMA and alpha-PVP would have predisposed to the development of a sudden upset in heart rhythm, including a fatal dysrhythmia such as ventricular fibrillation."

Is that the sort of thing that can happen?

A. Yes, but it is more of the background, so if that is going on in the background and you have got all this behavioural disturbance as well, then excessive restraint over a significant period becomes more relevant. So these restraint-related deaths often have stimulant drugs in the background, it is not unusual.

Q. Professor Crane went on to say:

"If restrained at the time he suffered cardiac arrest and if that restraint was such as to have impeded respiration, then it would be reasonable to conclude that such restraint played a part in the fatal outcome."

A. I agree with him.

Q. Now, Dr --

A. It is absolutely fundamental to a case like this what was going on at the time. So timings, as I think as you emphasised earlier, are highly relevant to be considered.

Q. Thank you. Dr Shearer also gave evidence that.

"These stimulant drugs, the MDMA and the alpha-PVP,

can have an effect on the cardiovascular system, so they can cause an increase in heart rate and an increase in blood pressure and they can also cause arrhythmias. They can result in a cardiac arrhythmia that is not kind of conducive to life, basically the heart can't beat properly and the heart then stops and causes a sudden cardiac arrest. So any stimulant drugs, that is one of the kind of extreme complications of that. They can both stimulate the heart in adverse ways~..."

I am reading this slightly short:

"... and can cause the heart to stop in a sudden cardiac arrest."

She agreed that it would cause the heart rate it increase, it would cause blood pressure to increase, and both could cause an arrhythmia where the heart was unable to pump oxygen around the body. And I asked her.

"Question: If the heart stops, that would be the point at which they die?"

And she said:

"Answer: Yes, they are in cardiac arrest at that point, yes."

So are these possible things that can happen once you have taken these stimulant drugs?

A. They are, although that would be much more concerning if he had been found dead sitting in a chair at home or

dead in bed and the only positive finding was the toxicology. But it is not that sort of case, it is what is going on actually at the time he went into his cardiac arrest, and everything going on is relevant.

Q. So would you recommend to the Chair that when he is considering the evidence he considers all of the evidence and not just simply one aspect in isolation?

A. Absolutely.

Q. She also talked about the fact there was no evidence of seizure. She said:

"Answer: Although it is not possible to tell at post mortem if someone had high blood pressure or their heart rate had increased, there was no evidence of seizure and there was no evidence of any damage to the heart in any way."

A. Yes, so I mean -- I think it is very likely that whatever the circumstances precisely were, his heart rate would have been high and his blood pressure would have been high because struggling against restraint is very, very energetic, and sometimes if you see no movement it is what is called isometric exercise, so the muscles are doing a huge amount of work, they are consuming oxygen, putting your blood pressure up, putting your pulse rate up, but there is not very much to see. That always has to be borne in mind really,

particularly when someone is quite physically held down.
Q. I would like to move on to the restraint, in the final post mortem report Dr Shearer had identified that as being a matter of some significance in relation to the cause of death.
A. Yes.
Q. She gave evidence about the importance of the background, what was happening at the time, she talked about injuries identified externally and internally.
A. Yes.
Q. She said those were consistent with restraint and struggle against restraint. Would you agree with that?
A. Yes, I agree with that.
Q. She talked about injuries which were consistent with the application of handcuffs.
A. Yes.
Q. And leg restraints.
A. Yes.
Q. And the use of a baton.
A. Yes.
Q. You would agree with all of that?
A. I would agree with all of that, yes.
Q. She also talked about skin -- injuries to skin, scraping, abrasions, lacerations, that type of thing, which were consistent with the body being moved along

the ground --

A. Yes.

Q. -- with clothing on. We talked about an injury to the trunk area, we have heard evidence that he was wearing a T-shirt but she also said it could be skin just on the ground itself because his T-shirt was short-sleeved.

A. Yes, but actually you can get abrasions through light fabric as well.

Q. So you agree that all of that could be consistent with --

A. I do.

Q. -- struggle and restraint? And then she also talked about injuries to his head and we spoke about the fracture to the first left rib.

A. Yes, absolutely.

Q. She also spoke about petechial haemorrhages.

A. Yes.

Q. Can you tell us a little bit about petechial haemorrhages?

A. Yes, basically they are little bleeds into the skin of the face, particularly the linings of the eyes, but they can occur more extensively in the skin of the face. They are essentially due to blood being able to get into the face but not so easily out again and that is because the venous pressure is much lower than the arterial

pressure that puts it in there. And in circumstances of restraint you may see them if someone is gripped around the neck, because that is an easy explanation for why blood can get in but can't get out again. But you may also see them if a chest is effectively pinned, so you see them in crush asphyxia-type deaths because the blood literally can't get out of the face and the head and back into the chest because the chest is squeezed so hard.

Q. So if there is something, a pressure of some description, or a weight of some description on the chest, that can prevent the blood coming back from the head down into the body?

A. Yes, and it can also force the blood back up the veins into the skin of the face.

Q. What happens under that pressure of being forced?

A. The little tiny branches of the veins rupture because they can't take that pressure.

Q. Is it the rupturing of those vessels that causes these petechial haemorrhages?

A. Little bleeds, exactly. So little tiny small vessel ruptures.

Q. Dr Shearer gave evidence that those were visible --

A. Yes.

Q. -- in the eyelids.

A. Yes, and I corroborated that, yes.
Q. Thank you. Dr Shearer indicated that the presence of these petechial haemorrhages could be indicative of a degree of asphyxia.
A. I agree with that.
Q. Can you explain what asphyxia is?
A. So asphyxia just really means lack of oxygen delivery in the tissues but in these circumstances it tends to be used to describe a term where it's due to blood being forced into the tissues in some way.
Q. The blood is being forced into the tissues?
A. Yes, so it could be a sign of crushing of some sort or squeezing the chest.
Q. If the Chair took the view that these petechial haemorrhages were consistent with asphyxia of some sort, would that be consistent with positional and mechanical asphyxia, or just one?
A. Positional alone, so just being for instance flat on your front, won't give you petechial haemorrhages, it implies a degree mechanical asphyxia. And I should say in restraint-related deaths they are quite uncommon, they are not common, so when they are present this case becomes more like a crush asphyxia where someone had been in a crowd crush.
Q. So it would be more akin to mechanical asphyxia?

A. Yes.
Q. When we hear about mechanical asphyxia, what type of mechanism would cause mechanical asphyxia which is more likely to give rise to petechial haemorrhages?
A. So the chest being squeezed very hard. In fact the trunk overall.
Q. We have heard evidence a while ago that when doctors talk about a chest, they also include the back.
A. Oh yes, it's the front and back of the chest, of course. You are absolutely right, that often causes confusion in casework.
Q. So if pressure or weight or force was applied to the front of the chest or to the rear, to your back, could either of those count as mechanical asphyxia?
A. Yes, it could.
Q. That could give rise to petechial haemorrhages?
A. Yes, exactly.
Q. In terms of the level of force that would be required or the pressure required, can you help us understand anything about what would be required?
A. The easiest model is I go back to crowd crushing really, we know the sort of levels of force developing in a crowd, so it's often substantial force but I think if you have multiple individuals applying that force, particularly if they are quite heavy, then you will

develop sufficient force to get this degree of mechanical asphyxia.

Q. When you say multiple individuals, do you have a minimum number or not?

A. I think you are very unlikely to see it with one or two, although I have seen it when people have been effectively crushed by bouncers having been evicted from a night club. But these are huge guys, applying maximum pressure to someone's trunk.

Q. When you say huge, can you give us an idea of how big?

A. Over 18 stone, that sort of amount. So over 100 or more kilos.

Q. So if there was some evidence about weight being applied by someone who was 25 stone, is that the type of level?

A. That is the type of level, yes. That is a lot of force to apply.

Q. If that was -- if we have heard evidence that that was combined with an attempt to restrain, and pressure being applied to a person, would that add to the level of force and weight and pressure?

A. Yes, it would. So the limbs, if the limbs are held down, although the trunk being compressed would be the most significant, the limbs can actually basically transmit force to the trunk if they are being held down and restrained.

Q. How does that happen?
A. I suppose the simplest explanation I could give that would be a sort of similar mechanism would be how a guy rope works and holds up the tent pole; it is not direct force, it is just the presence of something pulling.
Q. Right. That would be how the arms would also apply force?
A. Yes, also of course that sort of holding, being completely held down increases struggling which increases oxygen consumption which then makes the consequences of the restraint greater.
Q. So if, for example, someone was straddling legs?
A. Yes.
Q. And another officer was applying some weight who was about 25 stone, and another officer had a baton being held over the arm, had pinned the arm of the person on the ground, would that combine in terms of force and pressure being applied --
A. Yes, I would be more concerned about the trunkal pressure particularly, so the 25 stone, and also the pinning of the legs. The pinning of an arm I suspect would be little, if any, contribution.
Q. Thank you.
A. Unless it was very high up, around the shoulder region.
Q. Probably in the area between the crook of your elbow and

up here (indicates), so in this sort of area?

A. So not a strong contender compared to 25 stone body mass.

Q. Thank you. In terms of positional asphyxia, Dr Shearer said you really couldn't take positional asphyxia out of the equation when you have it in context of all the other things that are going on:

"... it might require less to be in that position for a reduced period of time because your heart is already under strain due to the drugs being taken, and because you have maybe run around and you have a build-up of lactic acid."

She said:

"This would be a multifactorial death. We do not know what the main factor has been. I think all the forensic pathologists that have been involved in this case would completely agree with that and have all completely agreed with that."

Do you agree with that, that it is multifactorial?

A. It is multifactorial, undoubtedly but I think we are all talking about -- when factors are proposed, we are thinking of factors at more than minimal, which is a very important test as a forensic pathologist. So these are not trivial factors, these are factors that are more than minimal.

Q. Lawyers tend to say anything that is not -- anything that is de minimis or trivial we will ignore, anything more than that --

A. Exactly, so I think for us to include it in the cause of death we would be mindful of it being more than de minimis.

Q. So anything more than trivial or de minimis would be considered of significance or material?

A. Yes, although I would agree how significant. I mean, to me the most significant factors here were the restraint but the other factors form the backdrop. And the cause of death is always phrased in this traditional manner of 1a or 1b and then 2 but that was only ever devised for statistical recording of causes of death and narrative causes in this sort of case are very helpful, when the cause is multifactorial you look at factors that are definitely acting, factors that are probably acting and then factors that are no better than possibly acting. This case lends itself to that sort of analysis really.

Q. Thank you. We heard in relation to the final post mortem report from Dr Shearer that it was sudden death in a man intoxicated by MDMA and alpha-PVP whilst being restrained. I think in your Inquiry statement you have made it clear that the suggestion you would have made would be to substitute the phrase, "Whilst being

restrained", with, "In association with struggling and restraint"?

A. Yes, I think that is an important component because that gives you your high oxygen consumption.

Q. And so --

A. You can see I have already touched on the fact that a narrative cause of death is best in those circumstances and that is exactly what we have all adopted, basically agreed upon. The only thing I have added is the struggling.

Q. So is it fair to say you agreed with the conclusions of Dr Shearer and Bouhaidar but you added in the importance of struggle?

A. Yes.

Q. And is that in line with what you said a moment ago --

A. Exactly.

Q. -- about all the different factors?

A. Yes, so the struggling is a "more likely than not" factor.

Q. Integral in the circumstances --

A. Yes, but it is very difficult to have restraint and struggling separated from one another because they are all part and parcel of the same thing. And in fact sometimes people are not getting enough oxygen in and of course they struggle more, and that may be

misinterpreted as resisting restraint. In fact, I suspect even if you held someone down who is meek and mild, they would struggle.

Q. We have heard evidence from Dr Shearer that she agreed with your suggestion and has adopted that now.

A. Yes, that is helpful when really all the experts from the same speciality agree.

Q. It is, and helpful to the Chair also.

A. Exactly.

Q. So you have talked about -- you talked earlier this morning about you need to know about how things work normally --

A. Yes.

Q. -- when things are working properly, so from a physiological point of view you understand how they are supposed to work so you can then identify if they fail or they stop working properly?

A. Yes, exactly.

Q. I think you said if they go wrong?

A. Yes.

Q. I would like to ask you some questions about how things work normally, how they are supposed to work.

A. Yes.

Q. Just so that the Chair can have a very clear picture about that. So what we have here, what we have heard

evidence about, is that an apparently healthy male, Mr Bayoh, he was in his early 30s, he was 5 feet 10 inches in height, his weight was 12 stone 10, and he had a BMI of 25.6.

A. Yes.

Q. Can I ask you to what extent does your BMI give an indication of your level of health or fitness?

A. You have to be a bit careful about this one because Olympic oarsmen will have probably quite a high BMI due to the amount of muscle mass they have, so it's a very crude sort of assessment of how people are. But you know he is not into the overweight or obese category at all.

Q. Thank you.

A. Particularly as he is quite muscular.

Q. I would like to get a picture for the Chair about how a person's respiratory system would work --

A. Yes.

Q. -- when it's not impeded in any way, so it's working normally and in a healthy way. Could you explain that to us, please?

A. Yes, so the idea is to get oxygen in and expire carbon dioxide and you do this through breathing which you do a few times a minute, so you take breaths in and you take breaths out and in your lungs air exchange

takes place and the oxygen comes from the air and gets into the blood, and at the same time really the carbon dioxide comes out and gets into the alveolar airspaces to then be expired with each breath. So it's serving two useful functions, breathing: getting oxygen in, getting carbon dioxide, the waste product of respiration, out.

Q. So when we breathe in, we are breathing in oxygen into our lungs?

A. Yes.

Q. And a process takes place within the lungs which allows you to then breathe out carbon dioxide?

A. Yes.

Q. What happens to the air once it is into your lungs?

A. So that is where the little tiny airspaces called alveoli, oxygen can actually get directly into the blood stream and carbon dioxide can get directly out.

Q. So from your lungs oxygen will go into your bloodstream?

A. Yes.

Q. Where does it go once it is in your bloodstream.

A. It is then circulated back to the heart and then pumped around the body.

Q. Does the heart pump it round all of the body, including the brain?

A. Yes. Yes, so some organs are more sensitive than

others, the brain and heart are the most sensitive and probably out of those the quickest to suffer if you are run out of oxygen is the brain.

Q. If your brain is suffering from a lack of oxygen or a deficit of oxygen, what happens to your body?

A. So ultimately you would actually irreversibly damage the brain function and that is why when someone goes two a cardiac arrest where the circulation has failed as well as the breathing, you don't have very long to get resuscitation going, essentially to keep the brain alive.

Q. We have heard evidence about something called hypoxia.

A. Yes.

Q. Can you explain to us what hypoxia is?

A. So that is a lack of oxygen delivery to the tissues. So there may be what is called hypoxaemia which is a lack of adequate oxygen in the blood, that can happen in a whole variety of ways, but failure to get oxygen into the blood is the most important.

Q. So a lack of sufficient oxygen into your body?

A. Yes.

Q. And --

A. And then at the end organs as well, so all the tissues need to continuously be supplied with oxygen, so that is through oxygenation of the blood and then circulation.

Q. So if you are not getting enough oxygen, how does the body react to that initially?
A. So various organs react in various ways, but one of the consequences of a lack of oxygen in the blood may be the brain damage I have just described, but also the heart may become irritable and may be vulnerable to heart rhythm disturbances which can ultimately culminate in cardiac arrest.
Q. If your body was suffering from some sort of deficit or inadequacy in terms of the quantity of oxygen it was taking in, how would you react as a person to try and get more oxygen in?
A. Well, at the worst end of it you would go unconscious, but on the sort of run-up to that you might well struggle and panic and desperately try and get oxygen in.
Q. How does your body try desperately to get oxygen in?
A. Just struggling against whatever is stopping it getting in would be the mechanism in those circumstances.
Q. You have said at the worst end it would become unconscious, why would it become unconscious; what would that be a sign of?
A. That is a sign of the brain being affected by the lack of oxygen.
Q. So the brain shuts down in some way, if I can use a lay

person's term?

A. Yes, and in fact ultimately if the heart then stops, stops supplying blood to the brain, it means you are on a very slippery slope to basically being dead. The breathing also stops when the heart stops, whereas if breathing stops the heart won't stop immediately. So you have a good store of oxygen in your bloodstream and in your lungs which will keep you going for a while if your breathing stops.

Q. Can I just go over what you have said there. When the breathing stops what does that signify in terms of changes to your body? Did you say the heart --

A. If -- let's turn it around the other way, if your heart stops your breathing will stop very soon afterwards. But if your breathing stops your heart won't stop until it runs out of oxygen, which will usually take minutes.

Q. So if your breathing stops and you are not taking any oxygen in at that stage, your heart will stop quite soon after that?

A. No, if the breathing stops due to some other external factor, you have a while where your blood becomes more lacking in oxygen, where unless that is reversed ultimately you will have a cardiac arrest. But what I am saying is if you have a cardiac arrest because the brainstem needs a good blood supply really very soon

after a cardiac arrest your breathing will stop.

Q. If you become unconscious because of inadequate oxygen in your system --

A. Yes.

Q. -- the heart will still continue pumping --

A. It will.

Q. -- for a while to allow the --

A. Yes, because it is able to go on extracting oxygen down to when there is not very much oxygen in your blood.

Q. So there will be a window of opportunity to reverse the effects of that the lack of oxygen?

A. There will. And although we always see portrayed on television resuscitation being successful, that reveals that window of opportunity is neither very long or very likely. It is possible, which is why all efforts are made to resuscitate people.

Q. Is there a set time or is everyone different?

A. I think everyone is different but if you don't commence adequate resuscitation with oxygenation, you know, within a minute or two, you are running the grave risk of brain damage.

Q. Is that why people start CPR very quickly --

A. Exactly.

Q. -- once you stop breathing?

A. Yes.

Q. We heard evidence a while ago about a situation where it is not as simple as just breathing or not breathing, there will be a process where maybe you are not breathing normally?

A. Yes, so you can have inadequate breathing in these circumstances. I agree with that. So -- and sometimes when people have been hypoxic, so lacking oxygen in the blood, when they come around a bit more they may not breathe adequately. Probably due to the brain damage they have had from a period of severe lack of oxygen.

Q. So you can be breathing normally, not breathing normally, and then not breathing?

A. Yes. So the not breathing normally --

Q. Is that the process?

A. Exactly, so it may be shallow breaths, it may be only intermittent breaths.

Q. Is it common, is it normal, for someone to go from breathing normally to not breathing at all?

A. As a primary stoppage of breathing that is quite unusual, there are a number of medical causes that can do that, but the commonest cause of stopping breathing is your heart stopping and not supplying your brainstem with blood.

Q. So more common for there to be a process --

A. Yes.

Q. -- where the breathing changes from normal to not breathing?
A. Yes, exactly.
Q. Thank you. I would like to ask you about possible causes of hypoxia.
A. Yes.
Q. So a possible cause is where there is that inadequate supply of oxygen and that sort of deficit builds up. We have heard about positional asphyxia.
A. Yes.
Q. And we have heard evidence from a number of witnesses, Dr Shearer talked about people being slumped in a position where there is an inadequate ability of the body to take in oxygen.
A. Yes, so that would be -- the more important element there would actually be obstruction of the airway, so if you are in a slumped position, particularly if you are affected by disease or drugs, then your airway may be vulnerable and that would be the primary source of hypoxia.
Q. We heard from Professor Lucas yesterday about a case where someone had slumped over a couch, I think he said they were drunk, and their neck was leaning over the arm of the couch --
A. Yes.

Q. -- and that had caused positional asphyxia.
A. Yes. But of course the most important underlying factor there would be the alcohol intoxication.
Q. That is what caused them to be slumped?
A. Exactly, so you could lay a fit healthy person on their front, you could even slump them slightly, and they wouldn't get into any sort of difficulty that would cause their death. So it's the acute intoxication that is relevant there.
Q. Would a healthy person be able to just simply move into a more comfortable position?
A. Yes.
Q. Even if they were asleep?
A. Even if they were asleep, yes. Fortunately you wouldn't expect someone to just get into a bad position in their sleep and then die, unless they had severe chronic lung disease.
Q. Something wrong with them?
A. Something really badly wrong with them, yes.
Q. Or they had taken drugs or alcohol?
A. Or they had taken drugs or alcohol, exactly.
Q. Now, can I ask you about the use of the word asphyxia there, positional asphyxia?
A. Yes.
Q. Because I would like to be clear about the distinction

between hypoxia and asphyxia.

A. Yes.

Q. Can you help me understand that?

A. Yes. They overlap but basically asphyxial mechanisms are those involving lack of adequate delivery of tissue oxygen, so that overlaps considerably with hypoxia but we tend to use the term asphyxia for specific circumstances like compression of the neck, or smothering, suffocation, that sort of thing. And then there is what is called internal asphyxia where the problem is due to poisons of one sort or another, cyanide, carbon monoxide poisoning, that actually stops oxygen getting to the tissues.

Q. We have heard a witness a while ago now talking about chemical asphyxiation.

A. Yes.

Q. Is that the sort of thing that can stop people breathing?

A. Yes, exactly.

Q. Is it fair to say that hypoxia is where there is a deficit of oxygen but asphyxia is where that has maybe been the endpoint of that lack of oxygen?

A. I think asphyxia is one of the causes, one of the root causes, but all these chemical mechanisms also apply.

Q. When you say causes, is that cause of death?

A. Well, causes the hypoxia, the lack of oxygen. So they are asphyxial mechanisms. But of course most people would not really use the term chemical asphyxia because it is really poisoning due to whatever the chemical was.
Q. We have also heard evidence about mechanical asphyxia and you have described that to us today in terms of pressure on the trunk?
A. Yes, and that is what applies here I think. It is a mechanical type of asphyxia, and support for asphyxia here is the presence of the petechial haemorrhages identified by Dr Shearer.
Q. Then the other possible causes, would they include things like infection?
A. Not very likely to cause asphyxia in these circumstances.
Q. Or hypoxia?
A. Hypoxia can certainly be the result of infection, yes. So pneumonia, severe pneumonia.
Q. So something that affected your lungs like pneumonia could cause hypoxia in the sense you can't get oxygen into your lungs?
A. Absolutely, and everybody is probably very familiar with this now from Covid-19. So that causes a very strange effect, which is ill understood, which is caused silent hypoxaemia. The hypoxaemia reflects the fact the blood

is often severely lacking in oxygen but you wouldn't know there is anything wrong, they just go bluer and bluer because of what going on.

The reason for that is probably because the brainstem is also affected in some way so it doesn't detect the lack of oxygen in the blood, and we have some good detection mechanisms for lack of oxygen in the blood and we will do everything like breathing harder to try and cope with that but also detection of carbon dioxide building up, which is what keeps you breathing, that is the primary driver of breathing that makes you breathe because the carbon dioxide is building up, so if you were to hold your breath, the overwhelming desire to break the breath hold would come from the build-up of carbon dioxide.

Q. Is that a message from your brain saying you have to breathe?

A. Exactly, exactly. So the free divers can get round this, they can go under water for a long time because they resist that effect, but they also take in a very deep breath of air before they go so that there is plenty of space in their lungs for carbon dioxide to come out into while they are under water.

Q. That is why if you were in hospital with Covid, for example, they can monitor your oxygen levels and the

levels in your blood?

A. Yes, exactly.

Q. Whereas people at home maybe weren't able to do that?

A. No. I mean there is a -- you can look at your saturations through one of these finger measures so people are using that as a sort of screen and I know the ambulance personnel use that to see whether the person is hypoxic because that is an important determinant as to how bad things are.

Q. You said that might not even be readily visible to the person or their family?

A. No, not at all.

Q. Could a pre-existing condition perhaps cause hypoxia?

A. Yes, it could.

Q. Something like asthma?

A. Asthma, yes, an acute asthma attack could cause hypoxia, or any sort of chronic lung disease.

Q. In this particular case there was no evidence of infection or any pre-existing condition that would impact on or cause hypoxia in Mr Bayoh?

A. That is correct, yes. That is my understanding.

Q. If you have hypoxia, you have talked about the brain sending signals to the body to breathe, is it reversible, hypoxia?

A. Yes. If the cause is reversible. But obviously if it

goes to a point where your brain is damaged it may be very hard to reverse it.

Q. So for the person slumped on the couch, if they are healthy they could sit up or move position?

A. Yes.

Q. Same with positional asphyxia or mechanical, they could move out of the way or remove the obstruction or they could hopefully be healed from the infection for whatever?

A. Yes, exactly, so given antibiotics, maybe support with oxygen administration in hospital, all that sort of thing.

Q. So that is all reversible?

A. Yes.

Q. You say that up until the point of maybe once your brain is damaged. I am interested in the tipping point, if I can use that, or the point of no return. The point after which you have been hypoxic for a certain period or it is of a certain severity that it is no longer capable of being reversible.

A. Yes, and obviously I have been involved in a lot of these sorts of cases and the most important tipping point is the occurrence of cardiac arrest. Once that has happened, even with good resus your fate is sealed.

Q. What happens during a cardiac arrest?

A. So your heart goes into a completely ineffective rhythm. There are a number of rhythms that it can undergo, so it can have ventricular fibrillation, people are probably familiar with, it just wobbles in every direction at once in an co-ordinated manner and that is -- the good thing about that is reversible with a defibrillator which gets it back to work. But in many circumstances the final common pathway may be asystole, where there is an absolutely flat line on the ECG and then you have to really get that heart going again, which would take some doing and drugs administration et cetera.

Q. Can you explain the difference to us between respiratory arrest and cardiac arrest?

A. So that is just breathing stopping.

Q. All right. So if --

A. But as I mentioned earlier, a cardiac arrest will stop your breathing very rapidly because the brain needs blood.

Q. So respiratory arrest is breathing stopping?

A. Yes.

Q. And is this the moment in time between -- that you talked about a moment ago where a person could be unconscious, they have stopped breathing, is that them in respiratory arrest but their heart continues beating --

A. If the heart is continuing to function, yes. The most important thing there would then be to support their airway, rescue breaths, administer oxygen; all those sorts of things would be helpful.

Q. So is it a very short period of time between respiratory arrest and cardiac arrest?

A. Yes, it might be a minute or two but if you have been struggling and you have been very active and you have a very high oxygen demand, the time gets shorter.

Q. We will talk about oxygen demand in a moment. So this -- if someone is unconscious, and they have stopped breathing, would that be an indication of respiratory arrest?

A. Yes, or it may be cardiac arrest because the cardiac arrest may have been the cause of the stoppage of the breathing. It is often -- it is very difficult in casework in general, indeed it is very difficult for emergency services to know what the first event has been, which is why you really want to monitor everything to see whether the heartbeat is actually working and whether they are breathing or not.

Q. If there is a pulse detected at some point, does that indicate that the heart is still working --

A. Still functioning.

Q. -- to some extent?

A. Absolutely, but the trouble with that is that if there is an evidential issue about feeling a pulse, it is really difficult in fraught circumstances to be sure that it is that person's pulse and not your own fingertips.
Q. Oh, really?
A. So it can be quite difficult and so I think most of the advice is if you think someone might be in cardiac arrest you should probably start cardiopulmonary resuscitation. It is certainly important to give the rescue breaths if they are not breathing.
Q. So as soon as they are seen to be in respiratory arrest or cardiac arrest, it is important to start the CPR straightaway?
A. Yes, absolutely.
Q. Is that immediately effectively?
A. Yes, you should -- that is a very grave state of affairs whether someone is in respiratory arrest or primary cardiac arrest.
Q. What if a person is just simply unconscious but breathing or noticed to be breathing?
A. That is obviously more perilous but they shouldn't be unconscious so you want to very quickly know what the cause of the unconsciousness was and then they would be vulnerable to airway obstruction which would keep them

unconscious.

Q. So if the person was simply unconscious but noticed to be breathing, would you start CPR at that stage or not?

A. You would start rescue breaths at first and then you would want to make sure that you had a good way of determining whether they had a pulse or not.

MS GRAHAME: Right. I am conscious of the time. We often have a break at this stage.

LORD BRACADALE: We will take a 20-minute break at this point.

(11.30 am)

(A short break)

(11.50 am)

LORD BRACADALE: Ms Grahame.

MS GRAHAME: Thank you. Hello again. Just before the break you -- I asked you about a scenario where a person was unconscious, but noticed to be breathing.

A. Yes.

Q. And I asked about when you would start CPR. Your answer was you would start rescue breaths at first and then you would want to make sure you had a good way of determining whether they had a pulse or not?

A. Yes.

Q. I am interested in this idea of rescue breaths, and I wonder if you could just give me a little bit more

explanation?

A. Yes, of course. So at its simplest level you could give mouth to mouth resuscitation, which is not as good as using some sort of device like a bag and mask because when you breathe out your own breathed out air is lacking in oxygen and is higher in carbon dioxide, so it's not the best breath to give someone to rescue them but it is better than nothing. So if they are not breathing adequately or are in full-blown respiratory arrest, it has a potentially useful effect.

Q. Is that a situation where they are not breathing adequately?

A. If they are not breathing adequately you would obviously need full emergency support, but it is actually quite hard to recognise, especially if you are a lay person, whether someone is breathing adequately or not.

Q. So in terms of the way of managing that situation, if they are unconscious, that to you would result in these rescue breaths or mouth-to-mouth from that point?

A. If they are not breathing as well, yes. If they are not breathing it is an acute medical emergency.

Q. If they are breathing or are seen by a lay person to be breathing, would you start rescue breaths then?

A. No, not if they are breathing. But if they are unconscious you would want to know what the cause of the

unconsciousness was, and obviously in these particular circumstances if they have just been heavily restrained it would rather speak for itself that that might be -- might be relevant.

Q. In terms of deciding, for a lay person it might be difficult deciding whether they are breathing or not breathing normally; is that a difficult assessment for a lay person to make?

A. It could be. People hold a mirror under the nose, or you look for chest movements.

Q. So if they are breathing normally, you wouldn't -- you would want to find out why they were unconscious?

A. Yes.

Q. If they are not breathing normally, you would start mouth-to-mouth or rescue breaths?

A. You would consider it. The trouble is it will be degrees, so if you were in full-blown respiratory arrest, so not breathing at all, that would be very obvious. If they are not breathing adequately it would be a much more difficult decision for a lay person make as to what exactly to do.

Q. And all of this is being done within a short period of time?

A. Exactly.

Q. I would like to go over your own conclusions about the

circumstances that we have heard evidence about.

A. Yes.

Q. You have addressed many of these -- all of these points in your Inquiry statement in different parts and also in your report. If you don't mind, I would like to go through things in a certain order with you if I may.

A. I would be delighted, no problem.

Q. So we are dealing with a male in his early 30s, we spoke before about his height and his weight and his BMI.

A. Yes.

Q. Can I just be clear that there are some references in your statement, you have been asked about seasonal rhinitis or hay fever?

A. Yes.

Q. You have said in your statement that has -- it is not relevant to the case at all?

A. Absolutely.

Q. So if others have also given statements saying hay fever, insofar as that might be something that Mr Bayoh suffered from, that has absolutely nothing to do with this?

A. That is right.

Q. Thank you. Insofar as he may have been checked for sleep apnoea --

A. Yes.

Q. -- again, I think in your Inquiry statement, 230, and in your supplementary statement at paragraph 24 you again say that is not relevant at all to this situation?
A. Yes, I think I did see some of the original notes now that I have seen the GP notes in relation to that problem, and it seems that he had noisy snoring, which is actually quite common. But I don't think the sleep experts were wholly convinced that he had fully established sleep apnoea. Because what happens then, you have to have significant periods of not breathing and very disturbed sleep, and you wake up very drowsy in the morning, you just don't you feel like you have had a good night's sleep. There may be other experts before the Inquiry who can give more relevant evidence than me on that point, but I didn't think it was a strong contender.
Q. Nothing we have seen so far indicates that that played a part in the events at Hayfield Road.
A. No.
Q. Would you agree with that?
A. I would agree with that.
Q. We talked briefly earlier about sickle cell trait.
A. Yes.
Q. And we have heard evidence from Dr Shearer about this, that that wasn't a part of her final post mortem report

but then subsequent tests were carried out at a later stage, and there was a further supplementary post mortem report where she noted that the results of the tests were that Mr Bayoh was a carrier of sickle cell trait.

A. Yes.

Q. Have you seen that?

A. Yes. And by and large I think I mentioned earlier that that could get into problems if you became hypoxic, which is why they tend to check it before anyone has a general anaesthetic.

Q. We have heard yesterday from Professor Lucas about the implications of sickle cell trait --

A. Yes.

Q. -- in relation to Mr Bayoh. We also heard evidence from Professor Lucas in connection with reports from a Dr Soilleux.

A. Yes.

Q. Sorry, my pronunciation will no doubt be appalling.

A. It is fine, I can understand it perfectly, so it's absolutely fine.

Q. It is Dr Elizabeth Soilleux and I am very interested if a paragraph in your Inquiry statement. I wonder if we could begin, just to put it into context, so it is between paragraphs 142 and 150. Let's start with 142 just so we can put all of this into context but it

relates to the sickle cell trait.

A. Yes.

Q. You will see that there was reference just in the page above to Dr Soilleux's report, if we just go down slightly. You were referred to her supplementary report, this relates to sickle cell, and that is from May 2018. If we can look at 142:

"There is a significant body of other evidence, summarised ... that sickle cell trait ... can cause sudden cardiac death, usually in very demanding physiological settings, such as intense physical exercise."

So as a primary position, if you may, she recognises that sickle cell trait can cause sudden cardiac death?

A. Yes.

Q. I think Dr Shearer also gave evidence to the Inquiry about army recruits --

A. Exactly.

Q. -- who have died.

A. Exactly, there were publications, a publication called Military Medicine and when they had army recruits, often who are not very fit but they had to do the usual thing you have to do in the army of running absolutely miles, sometimes in hot conditions, they had some sudden collapses, and they had this condition called -- it is

called exertional rhabdomyolysis. So normally with sickle cell trait you won't sickle unless the conditions are really difficult such as hypoxia, but when you have very vigorous exercise you can get this condition. And when you have rhabdomyolysis your heart muscle -- sorry, not your heart muscle, your peripheral muscles break down and release potassium into the blood and that can be lethal because potassium is potentially a heart poison.

Q. So that can have a detrimental effect on the heart?

A. Yes.

Q. We will come on to that in a moment.

A. So it is a factor to have in mind for one of the factors.

Q. Dr Shearer also spoke about a situation such as someone trying to run a marathon without proper training.

A. Yes, that would be very similar except that you -- in a military setting you tend to get forced to run the equivalent of a marathon, but it has the same effect.

Q. Then if we carry on. 144, please. Again, referring to Dr Soilleux's report. She notes that there was genetic testing carried out, Mr Bayoh was a carrier of sickle cell, ie had sickle cell trait, and she reconsidered the likely causes of death:

"A combination of exertion (including both

walking/running around over several hours preceding the restraint by police and his struggle against restraint) and alcohol consumption would have led to dehydration. The exertion would also have led to hypoxia (decreased blood oxygen levels). During hypoxia, anaerobic respiration occurs, producing lactic acid, leading to acidosis~..."

I would like to go through this. This is from Dr Soilleux's own report in 2018.

A. Yes.

Q. She talks about:

"... exertion (including both walking/running around over several hours preceding the restraint by police and his struggle against restraint)."

We will come on to the evidence we have heard in a moment, at the moment the Inquiry doesn't have evidence that Mr Bayoh was running around over several hours preceding the restraint.

A. No, so that is a false premise.

Q. So that is false premise. And then:

"... alcohol consumption would have led to dehydration."

What are your comments about that in light of the toxicology you have seen?

A. There wasn't any positive finding of alcohol, so I am

not sure quite where she has got that from. It may be historical in relation to these events.

Q. We did hear some evidence, we have had -- the Chair has an Inquiry statement available to him indicating that a friend had maybe seen Mr Bayoh buying alcohol, maybe taking some alcohol in the hours prior. But we have heard from Professor Eddleston that he had no alcohol in his system.

A. Yes.

Q. So if there had been alcohol consumed, it would have been out of his system?

A. Yes.

Q. And that is from the samples that were taken, both in the hospital and post mortem?

A. Yes, absolutely.

Q. Then she says:

"The exertion would also have led to hypoxia (decreased blood oxygen levels)."

Will you agree with the idea that exertion can cause decreased blood oxygen levels?

A. Not really, no.

Q. What do you mean by --

A. Our systems are designed to maintain normal oxygen if we exert ourselves. You won't tend to go hypoxic simply with exertion.

Q. So would walking around cause hypoxia?
A. Not unless you had some underlying significant chronic disease.
Q. We heard from Professor Lucas yesterday about normal sort of daily events wouldn't -- you wouldn't expect to have issues with sickling?
A. I would agree with that, yes. The real thing you want to try to detect in people who have sickle cell trait is to do anything to stop them getting hypoxic, and of course most of us, as I say, wouldn't get hypoxic with ordinary daily activities or even quite extreme exercise.
Q. So if exercise caused hypoxia, what sort of level of exercise are we looking at?
A. I think -- I wouldn't expect exercise to cause hypoxia at all. But in other circumstances, such as in combination with restraint or squeezing of the chest, that is when it might cause hypoxia. But exercise of itself in a healthy person shouldn't cause hypoxia.
Q. I think Professor Lucas yesterday talked about temperature could have an impact. We have heard about the example of the recruits in the desert from Dr Shearer.
A. Yes. That is more an effect of, you know, basically hyperthermia. So getting very, very overheated.

Q. Professor Lucas talked about the impact of that combined with dehydration can also cause issues with sickling.
A. That is true.
Q. Then it says at the end of this paragraph:
"During hypoxia, anaerobic respiration occurs, producing lactic acid leading to acidosis."
Do you agree with that?
A. Yes.
Q. Can you tell us a little more about that process please?
A. It is what stops you exercising too hard if you are not very fit, you get cramp, pain in your muscles, and you may get a lot of cramp the following day. It tends to be locally produced lactic acid and that is because it is the wrong sort of metabolism, the metabolism you want going on in your muscles when active is called aerobic, a lot of people who train in the gym know about these different aspects of exercise.
So aerobic metabolism where you are breathing adequately will not produce lactic acidosis, but anaerobic, where you are not getting enough oxygen to the muscle, so they are still capable of functioning but without much oxygen, that is what gives you lactic acidosis. It is very likely -- it's why, for instance, I thought in this case the struggling is a very important component here because you may well have

evidence of lactic acidosis from extreme muscle exertion.

Q. That production of lactic acid, is that reversible?

A. Yes.

Q. How is it reversible?

A. A number of means. It may be just the passage of time, so you may breathe off carbon dioxide which is a source of acid in your bloodstream, or your kidneys may be able to help reverse it. But it is not going to be instantly reversible, it needs things to go back to normal.

Q. It says during hypoxia the production of lactic acid leads to acidosis. Once that process has started, where the lactic acid has built up and you have developed acidosis, how does that -- in terms of -- we talked earlier about the tipping point or the point of no return, does this have an impact on hypoxia?

A. Yes, it does. So the heart doesn't like to work in an acid environment. If the blood is acid, whether it's due to lactic acid or some other acid forming in the blood, that makes heart function less effective.

Q. Dr Shearer said she can't do a test at post mortem for lactic acid.

A. Yes, I think you may be able to but it is very difficult, in someone who has a prolonged cardiac arrest it would build up anyway so it becomes uninterpretable

what the result is.

Q. Would it be difficult to distinguish between lactic acid prior to the cardiac arrest and after?

A. Exactly right, yes.

Q. Then see at paragraph 145, I think you mention there that the issue about the alcohol consumption, and you comment that toxicology was negative. So it's not an appropriate thing to include in the factors?

A. Not really, I don't think any Inquiry is assisted by possibilities. You want either probabilities or actualities.

Q. Then moving on, 146. You comment that "she dismisses the importance of restraint", but note that she is not a forensic pathologist.

A. Yes. Which is important, we each have to stick to our own speciality and I am a forensic pathologist, Dr Bouhaidar and Dr Shearer are forensic pathologists, so they probably not infrequently have to deal with cases of significant restraint and it is important to be familiar with your speciality.

Q. I think both you and Dr Shearer recognised the importance of considering all the circumstances, not simply the scientific or medical findings?

A. Yes, and of course a lot of that is entirely hypothetical: it could have been this, it could have

been that.

Q. Thank you. Then the doctor continues, is quoted in paragraph 147:

"... This markedly increased acidity, presumably becoming very severe during the restraint process, coupled with dehydration, would have caused sickling of red blood cells in Mr Bayoh, despite him only having [sickle cell trait]~..."

Subject to the comment you have already made about the alcohol and the dehydration, would you agree that increased acidity would become severe during the restraint process?

A. It may well have done, yes, if there's a lot of anaerobic exercise, so this sort of -- it is called isometric exercise where you see no movement but there is an awful lot going on in the muscles.

Q. Then 148:

"The dehydration as a factor in causing the lactic acidosis would feature here. The most important factor is the totally inappropriate levels of exercise. People who suddenly run a marathon without being trained properly would get lactic acidosis quite early on."

This is your own comments in your Inquiry statement.

A. Yes.

Q. Could you just give a little further explanation here?

A. So if you are not trained you would very likely slip into that anaerobic metabolism if you tried to run a marathon. I suspect if most people who weren't very fit tried, they would just fail. They would give up. So all these factors would operate on your body to make you stop.

Q. That is if you are healthy and you are not under the influence of alcohol or drugs?

A. Yes, particularly drugs. You would tend to be driven by stimulant drugs.

Q. Then 149. Again we go back to looking at the doctor's report, and she said in 2018:

"... This would have begun to block small capillaries, including those supplying the myocardium (heart muscle)."

So this is her reference to the effect of sickling?

A. Yes.

Q. You talked about small capillaries in the body being blocked earlier she talks here about the drugs being taken, the MDMA and the alpha-PVP:

"... and the exertion would both have substantially increased the heart rate and strength of the heart's pumping (known in medical terms as the stroke volume). This in turn would have increased the heart's muscle's requirements for blood, due to increased demand for

delivery of oxygen. This increased demand would have been difficult to meet because of red blood cell sickling, due to the acidic, highly concentrated (dehydrated) blood."

You say:

"I agree with this opinion."

I am interested in your views on this explanation of the process of sickling.

A. Yes, so that would be how it could cause problems in the heart but I think she has -- I think I go on to say the problem is that Dr -- sorry I can't remember how to pronounce her name properly.

Q. I have been told it is Soilleux.

A. Soilleux, I say she:

"... has not realised that the presence of sickling in someone with sickle cell trait means they have been pushed to the limit physiologically."

I think that is the important point here.

Q. So this is in paragraph 150. Let's have that whole paragraph on the page, please. Let's start at the beginning:

"The problem is that Dr Soilleux has not realised that the presence of sickling in someone with sickle cell trait means they have been pushed to the limit physiologically."

Can you help the Chair understand what you mean by "pushed to the limit physiologically"?

A. Yes, so something that is totally abnormal for any sort of individual in any circumstances, they have been pushed to the limit physiologically. So what it means is if they get hypoxia, I think that is what I go on to say, that will be bad because that could then precipitate sickling. But as I mentioned earlier, we wouldn't expect ordinary individuals to just become hypoxic. The only way you would become hypoxic is if you go up a very high mountain.

Q. So going to the gym might create lactic acid in your system but it is reversible and it wouldn't cause you to become hypoxic?

A. Yes, exactly.

Q. But the examples that we have heard of going up a mountain or running a marathon or doing army training in the desert, these are the sorts of exertions that would cause an issue?

A. Could cause an issue.

Q. Could cause an issue?

A. Yes.

Q. They wouldn't cause an issue in everyone?

A. No, only in people with sickle cell trait. And even then, not every time because they -- I mean, the

Sickle Cell Society would make it very clear that you live a normal life with sickle cell trait.

Q. So it would just be some people depending on the circumstances?

A. Yes, exactly.

Q. You say:

"I can equally argue that the presence of sickling actually provides strong evidence for hypoxia and lactic acidosis as a result of restraint. I noticed this in Professor Lucas's report too. We don't disagree on the findings, but the interpretation is very one sided in terms of what she says."

That is Dr Soilleux.

A. Yes.

Q. So can we just look at the line above, please. Back to page 37, paragraph 150. You say:

"I can equally argue that the presence of sickling actually provides strong evidence for hypoxia and lactic acidosis as a result of restraint."

I think you have commented on this earlier, I am interested in this line.

A. I think what it means is that he had an independent marker within his system for hypoxia, which was the fact he carried sickle cell trait, so if he was hypoxic that might cause sickling. So in a way to me this argues, or

helps to support any argument that he has been rendered hypoxic and that has caused sickling.

Q. So he has been rendered hypoxic, that process has resulted in sickling?

A. Yes.

Q. That has been tested and Professor Lucas gave evidence that there were -- about the quantity of sickling?

A. Yes, indeed I think he felt -- he sees quite a lot of cases of sickle cell disease of one sort or another and sickle cell trait, he felt that there was quite an exceptionally large number of sickling cells at post mortem. The problem is anyone who even carries sickle cell trait you may see sickling in their post mortem organs anyway because of course at the time of death the person becomes hypoxic, so that causes cells to sickle in any case. So you have to be careful it is just not a false positive.

Q. I think the Chair herd evidence yesterday from Professor Lucas and as I understand it, he did talk about there being levels of sickling for someone with sickle cell trait after death, and that would be apparent.

A. Yes, and he felt this was way in excess of that, was my understanding.

Q. He did. I think the word he used in evidence yesterday

was "massive".

A. Yes.

Q. And he talked about the different organs which had sickling in, and in the lungs it was in three out of the six samples he checked.

A. Yes.

Q. So in your mind when you are thinking about the impact of sickling, am I right to say you think that process having been found as a part of the investigations for post mortem is evidence itself of hypoxia having taken place?

A. Yes.

Q. Thank you. I think in your Inquiry statement you had looked at Professor Lucas' original report which was from I think 2018, and you've talked to us about the significance of sickling and his report. Can I ask you to look at something that Professor Lucas spoke about yesterday?

A. Yes, of course.

Q. He has reviewed his opinion since 2018, and I would like his Inquiry statement to be up on the screen. SBPI 00314 and it's paragraph 25. He gave evidence about this yesterday as well, so he has confirmed that this is his current view on the matter. So he said in his Inquiry statement:

"reflection on my report four years on, I don't think sickle cell trait is quite as important as I thought it was perhaps back in 2018. That's partly influenced by lots of discussions I had with coroners in England about how we should be phrasing causes of death; what is important and what is less important, what goes into part one, which is the main thing, and what goes into part two as a contributor. If I was doing this case again now, I would move the reference to sickle cell trait [to] part 2 of the death certificate. I would not frame the entire story of his death around sickle cell trait. I would just simply put that in on the end as a small extra factor; as explained earlier, that it may well have shortened his life expectancy by a couple of minutes, given the stresses he was under at the time."

Paragraph 26:

"Again, that's guesswork. I can't quantify that seriously. No one can do that. This is a very recondite field. I have been involved in a number of inquests over deaths involving sickle cell disease generally in patients with sickle cell disease and trait. On many occasions I have provided opinions that sickling play no role whatsoever; but conversely I have seen ones where it absolutely definitely did and, if

they hadn't had sickle, they probably wouldn't have died when they did. I don't think this case comes into that category."

So you can see -- we can have maybe paragraph 25 back on the screen, please.

A. I think that is a very helpful re-evaluation, and if you were to look at the General Medical Council who regulate all doctors you would find they are very keen on people reflecting and that is precisely what Professor Lucas has done, he has reflected and we are strongly encouraged to do reflective practice.

Q. He mentioned yesterday that he had spoken to you at some point, he couldn't remember when, but he had had a conversation with you. Do you remember speaking to Professor Lucas about sickle cell?

A. I might have done, the trouble is I have talked to him quite a lot about sickle cell because he is one of the people to go to for sickle cell disease. But I may well have talked to him about this.

Q. How often do you speak to him?

A. Less so now because he used to come into St Thomas's mortuary once a week, often when I was there, and so we would talk then.

Q. He spoke about you talking to him about this -- his view in this case, and let me just see ... that you had had

a conversation, and he couldn't really remember the context of that conversation, and he thought that it had some impact on him being caused to reflect on his opinion. So he was asked about a conversation that he had with you, he says:

"We always talk about mutual cases."

A. Yes, absolutely.

Q. He said that you had said to him:

"I've seen what you said about this and I think you're wrong."

So I said --

A. Somewhat blunt, yes.

Q. So I said --

A. Perhaps I should reflect on that.

Q. So:

"Answer: He said, 'From the way you have written the report actually I can tell you this is' -- I am trying to remember what Nat actually said, 'There was an awful lot more restraint than you have given credence to', it was words to that effect. Which made me think~..."

He says that he then reflected on that, and did think about the restraint and the extent of the restraint. Do you remember that conversation?

A. I do vaguely, yes.

Q. Can you tell us any more about it?

A. I don't doubt his recollection of it actually, that sounds about right. Because I was pretty surprised at the time he gave that opinion, this was suddenly -- everything is explained by sickle cell trait, which rides roughshod over the concept of restraint being potentially dangerous.

So I don't doubt his recollection of that. What I am pleased to say is that for this Inquiry he has obviously moderated a bit and I would agree with him that it might well be appropriate to put it under part 2 of the cause of death; it may well be a factor but not the main factor.

Q. Again, does this come back to your approach as a forensic pathologist to consider all of the circumstances --

A. Yes.

Q. -- rather than simply just the scientific elements?

A. Yes, very much so, I mean circumstantial evidence is often crucial for forensic causes of death. After all, from time to time we all have to give evidence in court about cases where there is no body found or the body is so decomposed, so in those sorts of cases the circumstantial evidence is vital.

Q. As I understand Dr Shearer's evidence, she considers that an appropriate finding in relation to sickle cell

would be that it go into part 2 --

A. Yes, I would support that.

Q. -- of the certificate?

A. Yes.

Q. So would you agree with Dr Shearer on that?

A. I would.

Q. Professor Lucas also gave evidence that:

"The quantity of sickling here tells me that this is much more than just post mortem sickling, it happened perimortem as part of the death processes."

And yesterday in evidence he explained that sickling occurred antemortem in life, he was asked about when sickling was likely to commence, and he said he was sure it developed a bit during the struggle and restraint.

A. Yes, and I would support that.

Q. You would support that. I would like to, if I may, go through with you the circumstances that we've heard evidence about in this Inquiry, and identify areas where there may be hypoxia, perhaps caused by, well, different causes and what they might be. If we look first of all at sort of consumption of substances we have heard evidence and we have touched briefly on the nandrolone, the steroids --

A. Yes.

Q. -- that had been taken, and we talked earlier about

Dr Shearer's evidence about recent administration. Dr Shearer gave evidence that there was no evidence of any heart disease and took the view that nandrolone was unlikely to have played a role in the death.

A. I agree with that.

Q. You agree with that. I think we talked about Professor Eddleston also expressing that view?

A. Yes.

Q. And you agree with him?

A. So you are getting it a from a toxicologist's primary angle but also two forensic pathologists.

Q. Yes. We have also heard some evidence from Professor Freemont, the osteoarticular pathologist, about the possible implications of steroids but I think earlier you said you would defer to him in relation to the bone issues and the fracture?

A. I do.

Q. In relation to MDMA we have spoken about this and I think again you said you would defer to Professor Eddleston on toxicology and Professor Lipsedge as a consultant psychiatrist --

A. Yes, I do.

Q. -- on the impact of those. Alpha-PVP --

A. I mean, the difficulty is, and I hope -- it hasn't has had yet but it is when they then say they defer to the

forensic pathologist and it all goes round in a circle, every defers to everyone else.

Q. They are not deferring to you on that.

A. That is good. I am pleased to hear that.

Q. That is good. With alpha-PVP I would like to ask you about some other information that is available to the Inquiry, we have obviously heard from Professors Eddleston and Lipsedge about these matters but I think as part of your Inquiry statement you were asked to consider Professor Crane's views, we could maybe look at your Inquiry statement, at paragraph 174. This is Professor Crane we mentioned earlier:

"I acknowledge that in this context, Professor Crane is discussing excited delirium in the context of Mr Bayoh's behaviour as opposed to suggesting that it led to Mr Bayoh's death. I think that is entirely fair. There's no doubt that people who have taken stimulant drugs may experience all sorts of bizarre behaviours. As a primary cause of death, I am of the opinion that unless the drugs are actually directly affecting the heart (which some drugs like cocaine can) they didn't have a primary role in causing death."

I am interested in that idea, the idea that these drugs could maybe just cause you to die suddenly just out of the blue.

A. Yes, they can. It is particularly associated with cocaine, I'm not sure about alpha-PVP but I suspect anything cocaine can do, it can do. If you go to A&E units, maybe here in Edinburgh as well as in London, you will find people who have taken cocaine who get chest pain and they get feverish because that is entirely a bad effect of the cocaine on their metabolism and on their heart and they can get heart rhythm disturbances, so there are potential roles there.

Q. In this case what makes you conclude that the drugs did not simply directly affect his heart and cause a sudden death?

A. Timing. So you can't ignore the fact he has been restrained and was struggling at the time, but also he probably had the drugs a significant amount of time before, so you would expect the maximum effect of the drugs to be you know at the sort of time after he took them, whatever time that was, for the particular drugs concerned and however he took them. So he might still have them as a background effect on his heart and other organs but not as a primary effect. Also we would normally associate death due to drugs with overdose rather than just a normal abuse-type dose.

Q. In terms of what you have said previously today, would that also mean ignoring any other factors like the

restraint?

A. It would.

Q. Which is not the approach you would recommend?

A. And you have to remember that I mean sadly cocaine and other stimulant drugs are abused on a grand scale, every weekend. And obviously we don't see many deaths directly due to them, that is part of the epidemiology in the background to all this. You know, you may get complications but they are quite rare.

Q. Thank you. In terms of consumption of alcohol we have already discussed that today.

A. Yes, thank you.

Q. I would like to talk about evidence we have heard about an altercation that Mr Bayoh had with his friend, a fight to use an easier word. The Chair has evidence available to him by way of a written statement from a Mr Saeed and he has also heard oral evidence from a neighbour of Mr Bayoh called Naomi Rhodes and this was a while ago, it was last year, but essentially if I may, I would like to read out a description.

A. Thank you.

Q. And I would like to get your comments on this. So in terms of the written statement, the description available to the Chair is that in Mr Bayoh's home that he, as Mr Saeed puts it:

"... sucker punched me from behind. He punched me on the head. He started charging towards me, I seen him starting running towards me. He picked up the washing line pole, he literally chased me all the way round the back of the house with the washing line pole, a wooden one. He threw the washing line pole but it missed me. He pushed me on to the floor in a neighbour's garden, I fell over a wall. He was on top of me, he was throwing punches into my head. I tried to protect my head. He did throw a good few punches."

So that is the description of the person who was involved.

A. Yes.

Q. Then Naomi Rhodes, who heard a noise and viewed it, she is the neighbour:

"It occurred approximately between 6.30 and 6.45."

She heard them she:

"... saw Mr Bayoh delivering five to six punches to Mr Saeed. The altercation lasted approximately two minutes."

From what she saw.

So from that description I am interested in your views about whether that would place increased demands on the body for oxygen?

A. It would be physical exertion with punching, but in

a way it is evidence of how irrational his mental state was really, which of course we know is primarily driven by drugs.

Q. And --

A. But I don't think it has any bearing on his immediate cause of death.

Q. Could that description be the sort of exertion that runs the risk of developing hypoxia?

A. No, not really, you would have to go a lot more than that. Again, I'm afraid with the abuse of stimulant drugs on a grand scale these sorts of altercations happen very frequently.

Q. So from that description is there anything that I have read out to you that would give you any cause for concern it could lead to asphyxia in any way?

A. No. It does show his behaviour, illustrates that, but we know that from the PM, post mortem examination, we don't have evidence of significant head impact either in the scalp or in the skull or in the detailed analysis of the brain. So it was with no consequences.

Q. We heard evidence from Professor Lucas yesterday that this as described would not be likely to cause sickling. Would you agree with that?

A. Yes, I mean he would be the better person on that but I agree with him. Basically if it doesn't cause

hypoxia, then it doesn't cause sickling.

Q. Thank you. We have talked earlier about things that could cause a detrimental impact on a person's respiratory system. Even if there was any detrimental impact at all, would it be reversible at that stage?

A. Yes, yes it would.

Q. So we have heard evidence from a Neil Morgan, who is also a neighbour of Mr Bayoh, who talks about shortly after this altercation having a conversation with Mr Bayoh where he is standing engaged in conversation. Is that the type of behaviour that could allow that process to reverse, even if had been created in the first place?

A. It is slightly what if. I find difficulty with that question. All I can say is that I was aware of what had happened before and I don't think that has any bearing on what happened subsequently other than illustrating how his mind was altered.

Q. Thank you. Then we have heard other evidence about Mr Bayoh's behaviour where he is -- witnesses have seen him walking. It's a period of around -- I think just under a mile, around a mile, from his home address to Hayfield Road.

A. Yes.

Q. Professor Lipsedge gave evidence that he retrospectively

diagnosed psychostimulant intoxication at that stage.

A. Yes, and I would defer to him on that.

Q. If I can again just read some descriptions of evidence that we have heard.

A. Of course.

Q. That Mr Bayoh was chasing a complainer's car, so the person who had phoned up to complain to the police, chasing a car, jumping in front of other cars, and stopping them. He was walking along the street, he was chasing cars, causing them to stop in the street, striking cars with a knife, perhaps hitting or kicking at a car.

In terms of that sort of level of exertion, any risk of causing hypoxia?

A. No risk of causing hypoxia but it does illustrate how he was, and probably his elevated level of arousal, and the catecholamines, the adrenaline, noradrenaline, dopamine; all those substances in his body elevated as part and parcel of that.

Q. Any risk from that description I have given you, or many descriptions, of any type of asphyxia?

A. No, not in my opinion.

Q. Again, we heard from Professor Lucas yesterday it would not be likely to cause sickling but you defer to him on that?

A. Yes, I defer to him. But if it is not likely it cause sickling, it is not likely to cause anything else either.

Q. Again, even if there was any risk of any sort in relation to hypoxia, would it still be reversible at that stage?

A. Yes.

Q. I would like to move on to ask you about some evidence that we have heard about when the officers arrived at the scene in Hayfield Road.

A. Yes, of course.

Q. The description of his walking at around that time is:

"... walking briskly, walking with purpose, eyes bulging out of his head, being high on something."

And the first officer arriving, first two officers arriving, coming out of the first police van which has arrived, it's a large police van, they are in full uniform, they have equipment with them, and they shout commands. They draw their sprays, and during that process he is -- attempts are made to discharge a CS and PAVA spray at Mr Bayoh.

A. Yes, the problem is in this mind altered state people often don't obey commands in any case, so it's a difficult problem.

Q. We've heard evidence from Dr Lipsedge on that.

A. Good.
Q. So in relation to that, particularly shouting commands and spraying with CS and PAVA, we have your evidence about the chemical element of that. Is there a risk at that stage of hypoxia?
A. No. The difficulty is it's done quite commonly in these sorts of circumstances, it often has no effect in terms of bringing a chemical incapacity to the person at all. What it does mean is that then subsequent dealing with him means he is all contaminated by things that affect other people, so it's ill-advised really and I think the forensic medical examiners -- sorry, the forensic physicians have made some quite useful recommendations on this, people tend to be resistant to those sorts of sprays when they are in an excited state, and not only that they don't do any good either.
Q. We have heard some other evidence about sprays, and these things.
A. Yes.
Q. So given the evidence you have just given, if no hypoxia, then no asphyxia, and Professor Lucas said no sickling?
A. Exactly.
Q. You would agree with that?
A. I do agree with that.

Q. Then we have heard about a further attempt at spraying?
A. Yes.
Q. That is -- presumably your evidence on that would be the same?
A. Yes, it would.
Q. We then heard evidence about Mr Bayoh possibly chasing another female police officer for a short distance --
A. Yes.
Q. -- and striking her to the back of the head. I am interested in relation to whether that strike to the back of the head was of sufficient force to cause her to fall forward onto the ground.
A. Sure.
Q. Is that the type of event or exercise that could cause a risk of hypoxia?
A. No. It is just -- it's evidence of excessive exertion.
Q. But --
A. But I don't know how fit Mr Bayoh was, so it would very much depend on your fitness what any risk of exertion would have, but there wouldn't be a risk of hypoxia in anyone.
Q. Not the risk of exertion that would overwhelm him physiologically?
A. No, exactly.
Q. Professor Lucas certainly said no likelihood of

sickling.

A. No.

Q. And, from what you have said earlier, no risk of asphyxia?

A. Exactly.

Q. Again, would that be reversible at that stage?

A. Yes.

Q. Then the Chair has heard evidence, that is disputed, about having struck the female officer and her having fallen, that he may have then used full body force to stamp, allegedly stamp, and there is different evidence about that that the Chair will have to consider. But again would someone stamping with their full body force on to a person on the ground cause a risk of hypoxia?

A. Not in him, no.

Q. It's him I am interested in.

A. Yes. So, you know, what that means is he is just -- to me, it shows he is capable of a lot of physical activity. But I'm not touching on the factual basis for it in any case.

Q. No, no, but that sort of level of activity wouldn't render someone at risk of hypoxia?

A. No. On the contrary, it shows that actually he is still functioning rather well.

Q. We then heard evidence from officers about an officer

striking Mr Bayoh with a baton --

A. Yes.

Q. -- on multiple occasions on his head and his body. I wonder whether being struck by a baton with considerable force on multiple occasions would put you at risk of developing hypoxia?

A. Only if you are rendered unconscious by the strike.

Q. There is no evidence to suggest that.

A. No. So I wouldn't expect a delayed effect of such a head injury. And, of course, the hair can be quite cushioning against the effects of a strike, the neck may move on the body and so may diminish the effectiveness of the strike in terms of force applied to the actual head.

Q. We have heard from Dr Shearer about injuries, external and internal, in relation to that.

A. Yes.

Q. And the Chair has that evidence in front of him.

A. Yes. Good. I think there was one main injury to the scalp, which might be the result of that.

Q. But nothing that you are aware of from looking at the post mortem or any of the results that would indicate there was a risk of hypoxia from the baton strike?

A. No. If the evidence is that he wasn't rendered unconscious at the time, then, yes.

Q. Thank you. Then there is evidence before the Chair that Mr Bayoh was brought to the ground at that stage --
A. Yes.
Q. -- by another officer, and it has been described as either a shoulder charge or as a bear hug type manoeuvre.
A. Yes.
Q. This is an officer who was around 25 stone. That was done in a very quick period of time. Is that the sort of thing of itself that could give rise to a risk of hypoxia?
A. It would depend how sustained it was. So if the chest was compressed through such a bear hug for more than a few seconds then that might be relevant. It may also be relevant to the development of the first rib fracture.
Q. We have heard evidence about that.
A. Good. Okay.
Q. So in terms of asphyxia, if the person was held for a few seconds in that bear hug or in the shoulder charge, could that cause asphyxia?
A. No, I would expect that to be recoverable. Very unlikely to cause anything other than loss of -- being toppled over really.
Q. Then the Chair has heard evidence -- he has heard

evidence that Mr Bayoh was in a prone position.

A. Yes.

Q. He has also heard evidence that he was supine. I am interested in the position and whether that of itself could cause hypoxia?

A. No. So -- and this has been some of the misunderstanding that has been in this whole area, really. Prone of itself is not dangerous, it is just that in a prone position you are actually quite vulnerable to people then overdoing restraint and, of course, you are vulnerable to the abdomen being forced into the ground as well with pressure from the back. So I wouldn't say that the prone position is fundamentally dangerous, it is just in combination with excessive restraint it is dangerous. You don't see cases of supine restraint I suspect because it is much more difficult to get control of somebody when they are supine.

Q. So again is important when the Chair is considering all these factors to consider the evidence that he has heard about the circumstances?

A. Yes.

Q. We then heard evidence about the actual restraint itself.

A. Yes.

Q. The Chair has evidence about the number of officers who were involved and what they were doing individually. But he has heard evidence about at one point Mr Bayoh performing a push up-type manoeuvre, seeking to lift officers from him. If you could just give me a moment, I would like to read out a description of this.

(Pause).

So:

"His arms and legs were flying ..."

This is a PC Good:

"His arms and legs were flying, kicking out and arms flailing, and he was trying to force himself up using his arms like a press up movement."

A. Yes. Well, that tends to imply he was on his front then.

Q. Yes:

"Several officers were trying to restrain him by pushing him to the ground."

And:

"PC Walker was lying across the top of the man's back towards the upper half in an effort to stop him from forcing himself to his feet. This was effectively to assist in pushing him to the ground."

Then another officer, Tomlinson, talked about:

"Mr Bayoh was able to take ..."

He has described it in evidence as a "bench press type" position but he then went on to explain it was a press up and he demonstrated a press up type position:

"... and lift himself from the ground whilst attempts were being made to restrain him by PC Walker and I."

He then said it would be a press up type position.

A. Yes. Again, that tends to imply he is on his front then.

Q. And he said:

"Mr Bayoh was making continued efforts to break free from this restraint and using his strength to lift myself and PC Walker upwards, upwards from the ground."

So that is the sort of descriptions that we have -- some of the descriptions we've heard about this bench press -- sorry, this press up that Mr Bayoh was apparently attempting during the restraint. I am interested in that element of the restraint and whether that puts a person at risk of hypoxia?

A. Yes, it does. I think the other thing is that is a huge amount of effort. If you think about from your description, that is an enormous amount of effort to lift, just not one person but lifting more than one. And of course getting your own body weight off the ground in a press up type manoeuvre. So that is good

evidence for why the struggling should be part of the cause of death. Also, it is stating the obvious, but it shows that he was not in any way unconscious in cardiac or respiratory arrest at that point in time, because he simply wouldn't have been able to do that.

Q. So not unconscious but at risk of hypoxia?

A. At risk hypoxia.

Q. I think as I said earlier, Professor Lucas gave evidence yesterday that it was during this that he was sure that that would be when sickling would start.

A. Yes, I think he is right.

Q. So you think at risk of hypoxia. Professor Lucas said risk of sickling starting.

A. Yes. Of course, sickling is a reflection of the hypoxia developing.

Q. I am interested in asphyxia then. So we have talked about hypoxia and asphyxia and that sort of process with sickling starting. Can you help the Chair understand the sort of mechanism that is going on at this stage?

A. Yes. So lack of oxygen in small blood vessels will -- so capillaries perhaps and smaller veins, those cells in there and that lack of oxygen, the red blood cells in there will then start to sickle, because it is a lack of oxygen that causes them to sickle.

Q. So you are talking about the risk of hypoxia and the

extreme exertion?

A. Yes.

Q. Because of the struggling?

A. Yes.

Q. In terms of the restraint element, what the police are doing, what are the risks at that stage from this effort being applied to restraint?

A. They are contributing a huge amount to the use of energy, and -- but there may well be some restriction of breathing through the amount of restraint to the trunk.

Q. You talked earlier about mechanical asphyxiation?

A. Exactly. So that is what it is.

Q. So the weight of pressure or force being applied to the trunk?

A. Yes.

Q. That is what you meant. Then if sickling is starting at this stage in the process, what impact is that also having?

A. That will be -- it depends obviously where it is, but if it is in vital organs like the heart or the brain, it could be a precipitating factor for sudden death.

Q. When you are talking about a precipitating factor, could you explain that to the members of the public that are listening?

A. Yes, of course. That means it's an immediate factor

that, once you have been rendered hypoxic, sickling may be one of the mechanisms that makes that particularly dangerous. Obviously the hypoxia itself is dangerous to the brain and the heart and, as I think I mentioned earlier, the sickling, the development of sickling, to me is more a reflection of the fact that this man has been rendered hypoxic. It is a marker within his system to hypoxia.

Q. So he has developed the hypoxia. That has triggered the sickling?

A. Yes.

Q. And then that makes the situation worse?

A. Exactly. Yes.

Q. And, as this is happening, there is the element of the mechanical asphyxiation that you have described --

A. Yes, and of course that continues up to the time he has collapsed.

Q. And that also make the situation worse?

A. Yes. Exactly.

Q. Are there any other factors during that moment or that time that we've not discussed?

A. I don't think so. Obviously the drugs in the background, the physical exertion and the potential for lactic acidosis, they are all relevant to that --

Q. I wondered about the lactic acidosis or the lactic acid

building up to acidosis. How is that factoring into this --

A. That affects the heart negatively, and I think other opinions have actually suggested that the lactic acidosis could also be relevant to precipitation of sickling.

Q. So the levels of exertion that I have -- the descriptions, for example of the press up type movement, that level of exertion, that level of struggling, is that the type of exertion that could trigger lactic acid to build up?

A. I think it is very likely, and I think that was already going on to an extent.

Q. Thank you. At this point when all of this is happening, is it reversible?

A. Yes. Providing he is not in cardiac arrest, it is reversible.

Q. Sorry, I didnt hear?

A. Providing he is not in cardiac arrest. Obviously the sickling would have taken further targeted therapy, so -- but just stoppage of what is going on is probably the most important thing in stopping the whole thing in its tracks. But then he would be in need of oxygenation as well, and whatever the treatment is for an acute sickling crisis.

Q. So if the element of mechanical asphyxiation had been removed, just stopped, what impact would that have thereafter if he is suffering from hypoxia --

A. It would stop it getting any worse. But also, assuming that advanced paramedics then attended and that he was assessed, they might not have been aware of the fact he was sickle cell trait positive, but that, you hope, would be discovered by the time he transferred to hospital. And of course he would be in a much better state to be transferred to hospital, even if he is unconscious but still with a cardiac output and breathing.

Q. We have heard other evidence in this Inquiry about the benefits of calling for an ambulance.

A. Absolutely.

Q. To have medical assistance available, preferably prior to any sort of restraint or certainly at a very early stage. Is that something that you would --

A. I think that is right and actually, I think I mentioned before, I regard these cases as acute medical emergencies, and so you need to have something on standby for that. I think the forensic examiners actually, they are part of the Royal College of Physicians and they have made a position statement, I think they made it before the Inquiry, about the

emergency nature of this sort of behaviour. So it's not just irrational behaviour or behaving badly or drug intoxication, they actually constitute emergencies, and sometimes they are dealt with very effectively in the A&E department.

Q. We have heard some evidence that really an ambulance should be called if a restraint is being contemplated effectively.

A. In these circumstances I think that is right, but obviously I am not a physician. So that would be sensible.

Q. You would defer to physicians on that matter?

A. I would really. The trouble is there are only so many ambulances around, as we know. But that sounds like it is quite an unusual situation to be presented with, so there is a risk of death.

Q. I would like to go back to something we spoke about earlier, but perhaps you would prefer a break? I can see you are maybe bothered by the lighting, are you?

A. No, I get hay fever. At the moment -- it's quite bad at the moment, so my eyes are just watering. It's not the stress of the whole thing.

LORD BRACADALE: Would this be a convenient point to stop for lunch?

MS GRAHAME: Yes, sir.

LORD BRACADALE: We will stop for lunch and sit at 2 o'clock.

(12.58 pm)

(The short adjournment)

(2.00 pm)

LORD BRACADALE: Ms Grahame.

MS GRAHAME: Thank you. We had been talking about the restraint before lunch.

A. Yes.

Q. And in particular about the press up. There are other elements of the restraint we have heard evidence about and I would just like to check with you that none of these elements cause you to change your view or maybe make you want to alter anything you have said?

A. Yes, of course.

Q. We've heard evidence from Dr Shearer that injuries she found at the post mortem were consistent with the application of handcuffs.

A. Yes.

Q. And we've heard evidence from officers about their attempts to apply, successful ultimately, to apply handcuffs to Mr Bayoh.

A. Yes.

Q. In relation to risks of hypoxia or any of that sort of process, would the handcuffs themselves be a significant

factor?

A. No, they are entirely benign from that point of view. They can cause local injury but not any risk centrally.

Q. So not in relation to hypoxia or any of that?

A. Correct.

Q. What about leg restraints? We have heard about attempts to apply leg restraints or fast-tracks as they sometimes are called, and an officer straddling the legs of Mr Bayoh and then rolling down, and leg restraints being applied by multiple officers.

A. The principal risk from that could be that you get this localised lactic acid build up in the muscles, because they are not able to wash themselves out. So when you take the leg restraints off, there may be a surge of lactic acid into the blood. I don't know whether any of this coincided with restraints coming off, they were probably still on at the time he collapsed.

Q. We have heard evidence that they endeavoured to put leg restraints on and they were ultimately successful in doing so and they remained on until Mr Bayoh was taken to the hospital.

A. Yes.

Q. So once they were applied I don't think they were removed. We have not heard any evidence they were removed at the scene.

A. No, quite.
Q. So do you think that would have had an impact on the risk of hypoxia?
A. No, not directly.
Q. Then I think I described earlier we had heard evidence actually that an officer had used a baton in a manoeuvre on the upper arm area of Mr Bayoh in an attempt, as I understand it, to control his arms so they could apply handcuffs. You talked about mechanical asphyxiation and you talked about the impact of that on the trunk --
A. Yes.
Q. -- even if it came from limbs. Do you think that use of a baton in that way would have had an impact on hypoxia?
A. I think it is unlikely. Any contribution would be minimal.
Q. We've heard -- the Chair has heard a lot of evidence from people in relation to pressure being applied, where it was applied to, force being applied, and weight to be applied. I don't want to put all of that evidence to you. Let me just put some of the evidence to you.
A. Yes.
Q. This comes from a PC Walker:

"Sheku Bayoh was actively resisting with extreme force, and was himself throwing punches at this point."

This is an at an early stage. He was asked if at

any stage did he lie on Sheku Bayoh and the answer was:

"Answer: I think as part of having to reach across him I had -- the upper part of my body was on his shoulder."

I said.

"Question: You are pointing to your right shoulder?"

And he said:

"Answer: Yes, his right shoulder."

And I asked what did he mean by his upper body, and he said:

"Answer: Yes, it was just from the waist up. I was on my knees at that point reaching across him.

"Question: And that weight was placed on his right shoulder?"

His answer was:

"Answer: Shoulder and his hands, because obviously I am reaching across to put my hands on his hands and I'm reaching across him so I could reach that far. He was lying on the ground at that point."

So that was just one example of much evidence the Chair has heard about weight being applied, pressure being applied, and the level of resistance --

A. Yes.

Q. -- being demonstrated by Mr Bayoh.

A. I think I touched on that before, that the resistance

might be more a manifestation of all the physiology going wrong in his system rather than resistance in the traditional sense.

Q. In terms of that evidence about weight being applied, we have also heard other evidence about weight being applied, someone lying over or being seen to lie over Mr Bayoh. Would that evidence be significant in terms of the risk of hypoxia?

A. Yes, it would because it is trunkal compression, so those two aspects of breathing I mentioned much earlier of the intercostal breathing, and the diaphragm --

Q. Remind me what that was?

A. I think I mentioned it in my reports rather than live. So you have two types of breathing basically, your intercostal breathing are the muscles between your ribs which when they contract they expand the overall volume of your rib cage. So that is one type of breathing.

The other is called diaphragmatic breathing. In that the diaphragm, like a large web of muscles on both sides between the chest and abdomen, contracts and when it contracts it again increases the inside volume of the chest. In doing so it also has to compress the contents of the abdomen, the liver and other structures. And all that together means that if you squeeze someone from behind you may diminish both intercostal breathing from

compression of the chest but also if you compress the abdomen from behind by compressing the back, that can reduce diaphragmatic breathing.

Q. So the movement of the diaphragm increases the volume in the lungs?

A. Yes, exactly.

Q. And the movement outwards from the front of the chest increases the volume available in the lungs?

A. Through the intercostal muscles.

Q. Through the intercostal muscles, so that increases the capacity of the body to take in oxygen?

A. Yes.

Q. And if a person is prone against the ground and pressure is applied to the back?

A. Yes, you potentially take out both of those, so intercostal breathing and diaphragmatic breathing.

Q. And that would reduce the capacity of the lungs in terms of capacity to take in oxygen?

A. Yes, and also something that people often forget is it reduces the store of oxygen in the lungs, so if you make the lungs low volume, by compression, then they can't store so much oxygen for so long.

Q. Why is that?

A. Literally the lungs act as one your best stores of oxygen, so that is why instinctively if you are going to

hold your breath you will take a deep breath in first to increase your lung volume.

Q. That then allows that oxygen to be circulating around your system?

A. Correct, and that touches on a piece of experimental work I was involved in where we asked people to hold their breath after some exercise but they had to breathe right out and then hold their breath. And we proved if you hold your breath when your chest is effectively empty you run out of oxygen much more quickly than you might anticipate, and that could be a very rapid effect.

Q. So you have no reserves?

A. No reserve, exactly. And your haemoglobin, although it's an oxygen carrier, that does not carry nearly so much in physiological terms as your lungs with air in them.

Q. So if your lungs don't have the same capacity, and they are not as efficient or storing as much oxygen, how quickly can that cause problems for your system in terms of the amount of oxygen available to your system?

A. With the experimental work I was involved in where we carried out that manoeuvre of getting people to hold their breath after breathing out we rendered people hypoxic within as little as 15 to 20 seconds.

Q. Goodness.

A. Most physiologists would tell you it is very difficult to make a human hypoxic by any means really. So it's a very potent effect and it just shows that these processes can develop very rapidly.
Q. And at the moment they become hypoxic, remind us what happens at that moment?
A. They are vulnerable to their hearts getting into a rhythm disturbance particularly with all the other background factors there. A little bit of hypoxia is fine. If anyone goes on an airline flight and they pressurise the cockpit to say 6,000 to 8,000 feet, that will make everybody hypoxic but not much. It is when you go beyond a certain level really that it matters.
Q. Is this like people who climb Mount Everest?
A. Absolutely.
Q. There is less oxygen in the air?
A. Less oxygen in the air because the air is effectively thinner.
Q. But they can still function for a while anyway?
A. They can, and there are some physiological mysteries as to how on earth people can climb to the top of Everest without oxygen and that doesn't really make much sense.
Q. Thank you. The other elements that I wanted to ask you about were the period of time -- we've heard evidence that events took place over a very short period of time,

and I wondered if that made any difference in terms of risks of hypoxia? You have obviously just described something that can happen in 15 seconds.

A. Yes, absolutely. It is very fair to say that this is a pretty rapid collapse following restraint. But it may simply reflect the nature and extent of restraint, and the background nature of Mr Bayoh.

Q. So the nature of the substances he had taken?

A. Yes.

Q. And also the extent of his perhaps struggle?

A. Yes, absolutely. We don't really see data published about risk and what people do but I do know that in the prison service they have been very cautious about prolonged restraint because they have a lot of experience of that. Without wishing to put a figure on what sort of time would matter, they have obviously realised through casework that the less time you do it for the better because it really is dangerous.

Q. Can I ask you to look again at your Inquiry statement, please.

A. Of course.

Q. I am interested in paragraphs 221 and 222. If we start with 221, this is an area of your statement where you talk about causation and restraint.

A. Yes.

Q. "I consider that the restraint and struggle did materially contribute which is why it has appeared in the cause of death. You couldn't ignore the presence of drugs in the background particularly because of their effect on the heart when there is a lack of oxygen and lactic acidosis. The difficulty is, these things are all intertwined with one another."

Is that really what you have been saying earlier today --

A. Yes, absolutely.

Q. -- in evidence?

A. You can't look at each factor in isolation. So why did it happen on that particular day? Probably because all these things combined on that particular day. But I think as I mention, those that appear in the cause of death are more than minimal, and at least on the balance of probability contributory.

Q. Thank you. At paragraph 222 you said:

"In my opinion the most significant aspects of the restraint would be the time duration of the restraint and the number of persons restraining. There is an unwritten rule in the prison service where staff are not allowed to go beyond a period of time restraining an inmate. I don't know what this length of time is. Prison staff are aware of the dangers of prolonged

restraint. Mr Bayoh's case is not the most prolonged that I have seen, but he is heavily overwhelmed and using up a lot of energy by the struggle."

I am interested in this paragraph. You say the most significant aspects would be the time duration and the number of persons restraining.

A. That is a measure of the amount of force being applied really. So you won't tend to see this sort of a case with just one restrainer, unless they are absolutely massive.

Q. What was the significance in your mind about the time duration?

A. It is fair to say that most restraint-related deaths are after a longer period of restraint than this. But that is not to say that there aren't dangerous mechanisms which I have just touched on that could operate really quite quickly.

Q. In --

A. Yes, I think duration and levels of force, which is what I am trying to express through the number of people, are often the relevant factors.

Q. Thank you. Then at 223 you say:

"If it is proved that PC Walker did lie across Sheku Bayoh's back this would be a significant aspect to the restraint. This weight applied is what forces the

abdomen into the hard ground and limits diaphragmatic breathing."

That's what you have explained.

A. Yes, and I have explained that in a bit more detail now, yes.

Q. But you have said in addition that would also cause this restriction on the intercostal breathing?

A. Yes, obviously the back would mainly affect the diaphragm because it would force the abdomen into the ground but anything happening higher up would affect intercostal breathing.

Q. And those muscles are in your rib cage --

A. Yes.

Q. -- between your ribs.

A. Yes.

Q. Can I move on in terms of the evidence we've heard. Just to give you some idea of the evidence, we've heard that police arrive at Hayfield Road at 7.20 in the morning.

A. Yes.

Q. That by the time -- we heard evidence from PC Tomlinson that by the time his emergency button was activated Mr Bayoh was on the ground. That was 7.21?

A. Yes.

Q. I am giving you approximate times.

A. Sure.
Q. By 7.25 he was unconscious but breathing, so noticed to be breathing and I think at that stage there was calls for an ambulance. So I am interested in -- this is a change obviously in the state of Mr Bayoh that we have heard evidence about, where from a position where there is a restraint and struggle against that restraint and I have talked to you about the press up and the active struggling and resistance, that he is turned on to his side and he is noticed to be unconscious?
A. Yes.
Q. Could you help the Chair understand what physiologically is happening during this part of the process?
A. Providing he is still breathing he is not in cardiac arrest at that point in time, but he has been rendered so affected by the restraint that it has rendered him unconscious. Now, many, many years ago -- fortunately things are a lot better than that now -- they used to restrain people, before the good antipsychotic drugs they used to restrain them and how they talked about it was restrain them until they go limp. That perhaps would take 30 minutes or so. So you can see that prolonged time and that then renders somebody unconscious. But you are in a very perilous situation where you are unconscious anyway, but particularly when

it's through that, and hypoxia.

Q. Can you explain why a person is in a perilous situation at that point?

A. All unconscious people are highly vulnerable unless their airway is protected and supported. But if the cause of it is hypoxia then you may need rescue breaths and you may need to get their oxygen level back up again. Because they my not be breathing adequately either after an event like that.

Q. If a person is unconscious because they are hypoxic, what is happening internally at that moment in time?

A. So their brain is not functioning properly and that then in turn will -- unless things are corrected by some means, you will end up with a cardiac arrest, which of course is what happened here.

Q. So the hypoxia itself can render a person unconscious?

A. Yes.

Q. Then we have heard that at 7.29, so this is around four minutes later, he is not only noticed to be unconscious but he is noticed to be not breathing?

A. Yes.

Q. Now, we've heard evidence of Airwave messages and there is no evidence between that period of not breathing normally. I know you have talked about that earlier today.

A. Yes, quite.
Q. So the evidence we've heard is at 7.29 he was noticed to be unconscious and not breathing.
A. Yes, and in fact he may have been in cardiac arrest at that time but to be fair to everybody involved it can be very hard to recognise cardiac arrest, which is why if someone's condition suddenly changes and they stop breathing, it is time to instigate cardiopulmonary resuscitation.
Q. So CPR?
A. CPR, but after rescue breaths to make sure it is not just breathing.
Q. Can you help the Chair understand that at that moment in time: unconscious, not breathing, in terms of physiology what is happening at that stage?
A. So their brain has been starved of oxygen, and including the lower part of the brain where breathing is controlled, and so that is quite dangerous, as you can imagine, that you've been rendered hypoxic enough to go unconscious, the state of unconsciousness may not necessarily resolve itself unless the hypoxia is corrected.
Q. So in terms of reversing the effects of hypoxia, the CPR can -- can that help?
A. Yes, and particularly if that has gone to cardiac arrest

but I would be at pains to point out that once you are in cardiac arrest, your chances -- this would count as an out of hospital cardiac arrest, your chances of long-term survival are quite low. There is a chance, but out of hospital makes a difference because of the lack of specialist support in the background. You are much better to have a cardiac arrest in the Accident & Emergency department or intensive care or recovery because there will be specialists available very quickly, drugs, machines. But of course when you are out of hospital there is not much additional that can be done.

Q. Just to be clear about this situation, if it was respiratory arrest at that stage and not cardiac arrest, what difference would that make?

A. It would still be potentially reversible. So you could get respiratory arrest from restraint from trunkal compression in particular.

Q. Would that be reversible by means of CPR?

A. Well, stopping the restraint, and particularly supporting the breathing. But sometimes they may be in quite a low heart output state and so a bit of help along the way for the heart might be helpful as well.

Q. When you say "help", what do you mean?

A. So chest compressions. The sort of cardiac arrest they

tend to have -- go into -- at the worst end of it they go into a condition called pulseless electrical activity where the heart has a beat but you don't -- sorry, it has an electrical beat but you don't actually get any useful output from the heart, or you are getting very low output from the heart. Particularly in those sort of circumstances chest compressions may well help to restore a bit more cardiac function. And of course the lack of whatever caused it in the first place still ongoing really helps.

Q. So there could be a beat, a heartbeat, but the blood is not pumping as efficiently --

A. Yes, exactly. It could be an electrical beat with the blood not pumping or the blood could be pumping very inefficiently.

Q. So in terms of this restraint and the struggle against restraint, and the impact on Mr Bayoh and what is happening physiologically at this stage, would you be able to summarise to the Chair what your views are about what was happening physiologically during that time?

A. Yes. So he was obviously very alert because of his underlying mental problem, principally triggered by the stimulant drugs and he was likely -- by the time restraint was applied he was likely to be exhausted from everything else that had gone on. But he became much

more exhausted through the struggling and at that time he was then quite forcefully restrained in such a way that it may well have triggered hypoxia quite early on, and it becomes a vicious cycle.

Q. Can you describe that vicious cycle again, just in summary for the Chair, to help him?

A. So lack of oxygen initially producing struggling against restraint, so appearing to be resistant but then the restraint continues and the hypoxia develops, or becomes worse, and then you have the likelihood of unconsciousness developing and/or respiratory arrest. And once you have got one or both of those, you are then more vulnerable to go into cardiac arrest through the hypoxia, so the lack of oxygen in the blood will cause cardiac arrest.

Q. And the hypoxia may also promote sickling?

A. Yes, exactly. That is why I felt rather than being a primary cause, it is a really important physiological marker of what has happened to him in the period leading up to death.

Q. And the hypoxia may be -- the petechial haemorrhages may be consistent with --

A. They are more to do with circulation, so they actually tell you something about how that developed as part of restraint, so he was squeezed so hard that these

petechiae developed.

Q. In terms of the evidence earlier about lactic acid build up and acidosis, tell us about how that factors in?

A. So all that would be a neglect influence in the background. So the heart doesn't like potassium, so if anything leaked out, potassium into the blood, you had a very elevated potassium, that could affect heart function. Likewise the lactic acid. But to be fair we don't have a measurement of either in this case so we are having to say if this had happened then that would be likely.

Q. Thank you. Then we have heard that CPR was performed initially by officers at the scene.

A. Yes.

Q. And then continued during his time at the hospital.

A. Yes, including with a mechanical device, I think.

Q. Attempts were made to use the thumper device.

A. Yes.

Q. Can you explain to the Chair the impact of that CPR continuing? He left -- the ambulance arrived at the scene at around 7.33, and then the paramedics arrived and they dealt with Mr Bayoh and then took him to Victoria, which was within a very short distance away. His life wasn't pronounced extinct until 9.04, so there was a prolonged period.

Can you explain to the Chair what was happening physiologically during that period?

A. I would think to all intents and purposes he was dead at the beginning of that really, or very close to. Because as I say the likelihood of out of hospital cardiac arrest successful resuscitation is slim, particularly in these sorts of circumstances. So what was going on, the prolonged CPR would really stand a diminishing chance of working. So you know you really are not going to walk out of hospital if you have had to have CPR ongoing for half an hour. It's incredibly unlikely.

Q. Thank you. We've heard evidence from one of the doctors at A&E whose original statement at the time in 2015 noticed a pulse when he arrived at the hospital but very quickly then took the view he had gone into cardiac arrest.

A. Yes.

Q. So thought perhaps in retrospect he was maybe in respiratory arrest when he arrived --

A. Yes.

Q. -- but very quickly had gone into cardiac arrest. I think her recollection of the precise details in evidence wasn't quite as good. That was a Dr Pickering --

A. No, that would depend on how she determined he had

a pulse. If it was electrically, in other words, having an active ECG, then it might be he was actually in this state of pulseless electrical activity, which would be a common consequence of hypoxia.

Q. So that in itself would be consequence of hypoxia?

A. Yes, absolutely.

Q. Thank you. Can I ask you about some hypothetical scenarios that we have heard about --

A. Yes, of course.

Q. -- and the risks, if any, of hypoxia being caused. Now, two of them relate to officers attending at a remote viewing position, so not approaching Mr Bayoh at all when he is in Hayfield Road, or being closer to him, observing, watching, feeding back to ACR. So not actually engaging with him at all. Could that put him at risk in any way, the mere presence of police officers in the vicinity, of hypoxia?

A. Theoretically can contribute to fear, but I don't think otherwise.

Q. What impact would fear have in relation to risks of hypoxia?

A. More the risk of culminating in cardiac arrest, so the fear would be part of the background of all these other factors on his heart.

Q. Then we've also heard some evidence from a witness

regarding a process we've called "de-escalation"?

A. Yes. I did actually -- years ago I was a police surgeon in London with the Met Police for nearly three years, so I have dealt with this sort of thing many, many years ago myself.

Q. And just to read out a short description of that for your purposes:

"Engage and negotiate and de-escalate, try to understand what's going on, allow them to inform the decision-making about the process, provide additional updates, opportunity to communicate key to building rapport, attempt to de-escalate, engage and negotiate."

So it involves actual engagement --

A. Yes.

Q. -- with the person. I am interested, in terms of risks of hypoxia, whether that process of de-escalation would incur any risks at all?

A. No risk. It would reduce the risk. That is the aim of the whole thing. The only problem is, which to be fair to everyone involved, you may not be able to negotiate with them at all. Although a calm atmosphere is good even when people have these sorts of psychotic problems. Dr Lipsedge would be the better person to address that.

Q. The Chair has heard evidence from him --

A. Yes. So a calm atmosphere, even if you've got mental

illness, is potentially more useful than a really fraught atmosphere.

Q. Does a calm atmosphere help protect against hypoxia?

A. Could do. But obviously it is not a primary factor.

Q. No, and then --

A. Sometimes you have lone police officers or a couple where the only thing available to them is de-escalation, and it works rather well quite often.

Q. Then the fourth scenario is called "verbal dominance". It has been described as a hard stop:

"An authoritarian approach, wishing to try and control the individual, verbal dominance, approach of communication, a methodology of trying to dominate the individual by getting them to comply with your instructions to minimise the risk or minimise the requirement to possibly use other force."

A. Yes. All this potentially works if you're bad, but if there is something wrong with you mentally, it may not have any potential to work at all. I think people are increasingly recognising this sort of condition is, as I said before, an acute medical emergency and there would be no amount of de-escalation or domination that will help, and some of it might even make it worse.

Q. Would that apply to the verbal dominance hard stop version as well?

A. I think so. Yes, absolutely. These people are very frightened often, these acute psychotic states that are drug-induced, and so the hard stop approach is hardly going to help really. I have actually had cases of my own where somebody felt -- he was paranoid in his own flat and he felt that the police were going to get him with guns. It was a sort of self-fulfilling prophecy because of course they did come with an armed squad round. Again, that didn't exactly help.

Q. Could you give me one moment, please.

A. Yes, of course.

MS GRAHAME: Thank you very much, Dr Cary. I have no further questions at the moment.

LORD BRACADALE: Are there any Rule 9 applications? No.

Thank you very much Dr Cary for coming to give evidence to the Inquiry. I am very grateful for all the work you have done on this case from the very beginning. If you can now go back to the witness room, a member of staff of the Inquiry will look after you.

A. Thank you, sir.

(The witness withdrew)

LORD BRACADALE: We have now reached the end of this hearing on the cause of death and I wish to say something about the progress of the Inquiry.

In total we have now heard 59 days of evidence. In

addition, hundreds of documents have been published on the website, including extensive statements taken by members of the Inquiry team.

Next month I shall hear oral submissions from core participants. These submissions will be focused on the evidence about the events leading up to and including the death of Mr Bayoh and the cause of death. There is more evidence to be heard after these submissions. Earlier this year the Inquiry heard evidence on the post-incident management by Police Scotland. There will be further evidence in relation to that, which will conclude after the summer.

Later we will hear evidence on training and the adequacy of training. This will be followed by a hearing on the post-incident management by PIRC and the Crown Office. Finally, there will be a hearing dedicated to exploring whether Mr Bayoh's race had any role in how he was approached and restrained by police officers and the post-incident management.

As there is a considerable amount of evidence still to be heard, particularly in relation to race, it would be premature to reach conclusions at this stage. For that reason I will not be issuing an interim report. After I have heard and reviewed all the evidence, I will produce a final report. The Inquiry will now adjourn.

(2.38 pm)

(The Inquiry adjourned until 10.00 am on Friday, 26 May 2023)

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