Tuesday, 23 May 2023

(10.00 am)

LORD BRACADALE: Good morning Professor Lucas.

THE WITNESS: Good morning.

LORD BRACADALE: You are going to be asked questions by Ms Thomson, counsel to the Inquiry, whom I think you have met. Before that you would raise your hand and say the words of the oath after me.

PROFESSOR SEBASTIAN BRENDAN LUCAS (sworn)

LORD BRACADALE: Thank you.

Ms Thomson.

Questions from MS THOMSON

MS THOMSON: Good morning professor, what is your full name?

A. Sebastian Brendan Lucas.

Q. May I ask you your age?

A. 75.

Q. And you are a recently retired consultant histopathologist?

A. Yes, I retired, I took off my gloves last October when my GMC registration would have to be renewed at great cost.

Q. Before I ask you more about your career to date, can I ask you to open up the blue folder that's on the table in front of you. There are various documents in there that you might find it helpful to refer to when you are

giving your evidence. We will begin by having a look at what is there. There should be a letter of instruction that you were sent by the Crown Office on 29 May 2018, with the reference number COPFS 02468.

A. Hang on. (Pause).

Sorry, I am just puzzled by the phrase "letter of instruction" because I don't see it here. There obviously was one at some time or other. What am I looking for? (Pause).

Q. It may not matter because all of these documents, if we need to look at them we will bring them up on the screen in any event. You mentioned, as you flicked through the folder looking for the letter of instruction, having come across your CV; is that in there?

A. Yes.

Q. Also the report you prepared for the Crown, is that also in the folder?

A. Yes, there are two. One from 2018 and one more recent. Yes.

Q. When you say there is a more recent report, are you referring to the statement you gave to the Inquiry?

A. Yes, the one with the Sheku Bayoh Inquiry badge on the top.

Q. I see so we have your CV and the report you prepared in 2018, and the statement that you gave to the Inquiry.

A. Yes.
Q. That is grand. What we will perhaps do is bring up the Inquiry statement on the screen please. It has Inquiry reference 00314.
A. Uh-huh.
Q. This is a copy of your statement, we can see that it was taken on 8 December of last year and --
A. And modified subsequently a bit.
Q. Yes, that is part of the normal procedure. If we go to the very bottom, please, of the statement. We see that the final paragraph, paragraph 64, reads:

"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."

And in that knowledge, you went on to sign every page of the statement; is that right?

A. Yes.
Q. You will see that your signature has been blacked out on the screen but I am hoping that there will be a copy of your signature on the hard copy in your folder.
A. There is. It is here, in front of me.
Q. You can refer to any of these documents at any time, if there is anything that I want to take you to, it will come up on the screen but if you would find it helpful

to refer to your report, or your statement or indeed your CV, you can do so and if there is anything that you would like to put up on the screen, then just let me know.

A. Okay.

Q. Let's begin with your experience, your qualifications, and your career. Can we return to the beginning of your statement, please, and bring up paragraphs 2 and 3. You give your formal qualifications at paragraph 2, which include a medical degree and a fellowship of the Royal Colleges of Pathologists and Physicians?

A. Yes.

Q. And at paragraph 3 you say:

"I have been a consultant histopathologist since 1980, with wide experience in general diagnostic histopathology and autopsy pathology. I have been a Member of the Royal College of Pathologists from 1978, and Member of the Royal Colleges of Physicians since 1975. I have published widely on all aspects of histopathology and autopsy practice, particularly in infectious diseases."

A. Yes.

Q. So you explain that you have been a consultant histopathologist since 1980 until the autumn of last year when you retired. In very general terms what does

the work of a consultant histopathologist involve?

A. It is all to do with diagnostics from looking at people's tissues, therefore it is different from looking at blood or looking at cultures in infectious diseases and so on. It really divides into three forms. There is histopathology, which is samples of people taken from living or dead people; there is cytopathology, where you scrape or obtain superficial layers of cells from various organs; and then there is autopsy pathology, which is the gross examination of people who have died. That is divided into forensics, which is suspicious deaths; and everything else, which is not. And that also involves using histopathology in a proportion of cases, so there are three lines of what we call histopathology.

Q. You explain in your statement that general diagnostic histopathology and autopsy pathology were your particular areas of practice and expertise?

A. Yes, yes. Cytopathology was invented after I trained, so I missed all that.

Q. I see.

A. And, yes, I have been doing diagnostic histopathology and latterly, certainly for the last ten years or so, I have mainly focused on autopsy pathology and given up routine live patients diagnostic histopathology. That

is when I turned 65 and decided to stop doing that.

Q. So far as autopsy pathology is concerned, am I right to understand you are not a forensic pathologist?

A. No, I am not.

Q. So the deaths you would be involved in would not be suspicious deaths?

A. In principle. In actuality, it is not quite so simple because for decades I have been involved in looking at some aspects of people who are dead people who have died under, in a sense, forensic circumstances, asked by forensic pathologists or coroners or lawyers whether I can contribute anything useful to the evaluation of those deaths. But I am not on the Home Office Register as being a proper forensic pathologist so I would never be asked directly or I would never have been asked directly to look at someone who had been murdered or thought to have been murdered.

There is one big exception to that is that which is that I got very much involved in a complicated murder investigation in New Zealand, about as far away from where we are sitting as possible. The only reason I got involved in the evaluation of that is that when I looked at the material, I realised that this poor child had not been murdered and had actually died of AIDS, HIV-AIDS, in which I have been an expert, and the unfortunate

medical and pathology staff of New Zealand hadn't realised what AIDS is because they don't have any, and they thought the kid had been murdered. And I could prove in court that in fact the child died of AIDS and there was no forensic aspect at all.

Q. Where were you in practice as a consultant?

A. Where?

Q. Yes.

A. In 1980 I was actually in Nairobi, in Kenya. But thereafter University College Hospital and St Thomas's Hospital actually, those are the two main places I have been based at, with an additional year spent in West Africa working on HIV about 30 years ago. But essentially I have been London-based.

Q. Can we move to paragraph 5 of your report.

A. Yes.

Q. Sorry, your statement, I should say. You explain that you:

"... became a professor of pathology in 1995 at Guy's and St Thomas's Hospital, retiring in 2012. From 2012 to 2022, I worked part-time at Guys and St Thomas's Hospital and also local mortuaries in Milton Keynes, Bedfordshire, Luton, and Buckinghamshire. I stopped working altogether in October 2022. Since then, I teach pathology trainees on autopsy pathology interpretation

by lecture, and I lecture to medical students on general pathology. And I review autopsy pathology cases at the request of coroners, pathologists, families, and legal firms."

So you were also a professor of pathology. And I wanted to ask you to explain the difference between your work as a consultant and your work as professor?

A. They overlap. I have been a consultant and I was a consultant in those hospitals, the professor bit is -- wasn't a personal Chair, it was a substantive Chair, there were -- the medical school had a position called Professor of Pathology. In fact, it says Guy's and St Thomas's, that where I started, within three years King's College Hospital also got in on the act and then Kings College London became the lead medical school. So it is actually three medical schools over those, which are about two or three miles apart in south-east London, where I was the professor of pathology. And when I stopped being that in 2012 I have not been replaced because medical schools don't like professors anymore because they regard us as not productive on the research assessment exercise, which is generally true; we are much more practical rather than research-orientated.

Q. So from 2012 to 2022 what was the nature of your work at hospital, was it still in a consultancy role?

A. Yes, my colleagues, friends and colleagues, over many years would always show me probable cases and I would tell them what I thought the answer was. But the responsibility for the correct answers was theirs. What I was actually doing most of the time was doing coronial autopsy work. Now, you know that the England and Wales coronial system is very different from the Procurator Fiscal system here in Scotland, but in England and Wales we look at something like 14% of everyone who has died mainly because no one knows why they died. And that was what I was doing in St Thomas's, Milton Keynes, Bedford Luton and Buckinghamshire, Stoke Mandeville Hospital, because that is where I live now.

Q. Those were you autopsies that you were requested to perform by the coroners?

A. By the coroners.

Q. But were these non-suspicious deaths or --

A. Yes, by definition. I mean, if it was a prima facie homicide case I would never be approached. But interestingly -- and you may have the same problem in Scotland -- there is a sort of curious border between forensic and non-forensic and if the -- we call them coroners but it might be the Fiscals in your case are slightly worried about a case, whether it might or might

not have medicolegal implications, they would often ask -- often ask an experienced pathologist, and they would pick on me, to look at it and to stop if I thought there was any malfeasance going on, because I am cheaper than the forensic pathologists, there is a huge pay differential in autopsy work between routine medical and forensic, about a tenfold difference in remuneration, and that has quite an impact on who you ask to look at things.

Q. I see. As part of your work carrying out autopsies for coroners, would you require to reach a view as to the cause of death and issue a death certificate?

A. Oh, yes, that is what we are there for. Yes.

Q. It is simply that you wouldn't be instructed in a case that, on the face of it, arose from suspicious circumstances?

A. No.

Q. Can I ask you a little about your experience as an expert witness. You mention in paragraph 5 being asked to review cases, not only for coroners but also for pathologists, families and legal firms. How many made medicolegal cases have you been involved in over the years, would you say?

A. Well, it drops off. Having retired, I can tell you it is amazing how quickly you become yesterday's man and

people forget about you. But until that point I was regarded -- and I am not being immodest or arrogant, it is simply true -- as in Britain the person who knows most about the morbid anatomy and the pathology of three things. One was infectious diseases, which includes HIV, which -- HIV made my career I have to say because it happened at about the time that I happened as well. The second one was maternal mortality, and I made St Thomas's Hospital the maternal autopsy centre for south-east England and had a huge experience. And then I also, and relevant to this Inquiry, took on an interest in sickle cell disease morbid anatomically because most pathology of sickle is only seen, sadly, when people with sickle trait, which is very common, and sickle disease, which is much less common, it is only seen when they die. Biopsy samples in life are not normally taken from people with sickle, apart from the kidney, which is sampled for practical reasons. And I got into sickle, to anticipate your question, at the suggestion of a haematologist at King's who was a sickle physician, who was lamenting the quality of autopsies performed on his sickle patients by all the other pathologists in the area who he said had no idea what they were looking at. And I just thought that was an interesting challenge, so I took it up.

That is exactly the same reason I got involved in maternal mortality, some local obstetricians were very annoyed by the inadequate autopsy evaluations of the fortunately few people who do die in pregnancy and delivery and someone said, "Would you like to look at it and take this one on as a speciality?" And I got absolutely fascinated and did that. Actually, I still review those cases but I don't, as I say, do any more autopsies directly myself because I can't legally. I am off the register.

Q. In terms of your medicolegal practice then, how many reports have you prepared in cases where sickle cell has been relevant?

A. At the last count in my folders I had looked at more than 100 people who had died of/with sickle cell disease. I have looked obviously at thousands of people who have -- no that is unfair -- a lot more people who have had sickle cell trait because it is common, and as we are going to discuss in great detail, sickle cell trait is mostly harmless.

Q. But in terms of -- is that in terms of your medicolegal practice or is that your work more broadly as a clinician and in terms of the autopsy work?

A. No, these are autopsies I have done myself and cases that people have asked me to review because of my known

expertise.

Q. All right. So you have been involved in more than 100 cases involving --

A. That is sickle cell disease.

Q. -- sickle cell disease, either as the pathologist or --

A. Yes, mostly as the pathologist --

Q. -- as the reviewer?

A. -- and about a quarter of them will be other pathologists or coroners saying, "Will you please look at this and tell me what you think is going on".

Q. And how many cases have you been involved in that concern sickle cell trait, can you put a figure on that?

A. Sickle cell trait, pathologists occasionally sent me saying, "I can see sickle cells here, do you think this is relevant?" And once it is established it is sickle cell trait and there is a perfectly reasonable alternative cause of death I say the sickle cell trait is irrelevant, which is the general rule. And as all the sickle societies all emphasise, having sickle trait means you have a normal lifespan unless some critical things happen, which we will no doubt come to.

Q. We will discuss those in detail later this morning. But I am interested to know whether you can put a figure on the number of sickle cell trait cases that you have been involved in over the years?

A. Interestingly the important sickle cell traits cases I have been involved in, and this is directly relevant to what we are talking about today, are people who have died in custody. And that is why I -- that is why I know about them. And I produced and gave you a list of eight, I think, that I have been involved in, including Mr Bayoh.

Q. So you have been involved in eight at least eight cases --

A. Yes.

Q. -- involving sickle cell trait --

A. Yes.

Q. -- where the deceased died following contact with police or in police custody?

A. Or prison, police arrest -- yes, in cells or the prison service. Or deportation centres as well, which counts as the same thing.

Q. Returning to your medicolegal work more generally, you mention in paragraph 5 of your statement being instructed by coroners, also being asked to review cases by pathologists?

A. Yes.

Q. You mention families and legal firms. By and large who do your instructions come from; are they in civil cases, in criminal cases for the coroner?

A. They are probably equally distributed actually across that source. As I say it is drying up now, you get forgotten about.

Q. So civil work as well as criminal work?

A. Very little -- if you regard death in custody as criminal, okay then that is part of it. But most of it is not actually, most of it is medical. Yes.

Q. Have you given evidence before?

A. I have given evidence in coroners' courts enormous numbers of times, in one or two High Court trials on medical negligence, none of which involved sickle. That is about it actually, I always work on the principle, if it is a contested civil case, that it is up to the lawyers to sort it out before we go to trial and they normally do.

Q. Have you published over the years?

A. Yes, I have published less on sickle than I have on all the other things I have been interested in. What I have done is lecture a lot on it but I did produce for the College of Pathologists a set of guidelines on how pathologists should address a sickle disease -- and I also mentioned sickle trait -- autopsy. This is really to try and improve autopsy standards nationally.

And I have written one or two papers on some aspects of sickle cell pathology. All of which derived from

autopsy work, I may say, the only in-life pathology interest in sickle really that matters is kidney disease because people with sickle cell disease can have very bad kidney disease. But apart from that, sickle pathology is a sort of unknown territory to most pathologists.

Q. Can we look at paragraph 4 of your report please.

A. Paragraph sorry?

Q. 4, it will come up on screen and you have the hard copy two:

"Also in the late 1990s, I have taken a special interest in the morbid anatomy of sickle cell disease."

You mentioned that a few moment ago in your evidence, Professor:

"I have done around 100 autopsies now on individuals with sickle cell disease and autopsies on uncountable numbers of individuals with sickle cell trait, because trait is so common. I have been involved in medicolegal court work on many sickle disease deaths and, specifically, five previous deaths in custody or stress-related deaths among men with sickle cell trait."

I think you said that number is perhaps eight --

A. I think it is eight actually. I am not sure why I wrote five.

Q. -- rather than five.

You have explained to us how your interest in sickle cell disease and trait developed over the years. How have you developed that interest in practical terms? Is your autopsy work or is it your diagnostic work; what allows you to develop that interest?

A. Yes, that is a very good point. How do you get good at anything is ... this is Lucas' very simple rule for people who want to be experts. Say that you are and then people will start sending you problem cases and you learn on the job. It really is as simple as that. And after a while you realise you have seen more than anyone else and people realise that. It sounds arrogant but it is absolutely true, people who want to be lung cancer experts simply declare, "That is what I am going to do" and then the difficult cases are sent to them and they get better and better at it in sorting them out.

So in sickle and in HIV and in maternal mortality I passed the word to the coroners in the area of south-east England to say if you have a case that -- a patient who has one of these things and requires a coronial autopsy, please -- emphasise the please -- send it to us at St Thomas and we will do the job for you. So I managed to build up a large collection of experience on these things.

Q. Are there --

A. I have to say also that the coroners are quite grateful because they realise it will be sorted out better than their local pathologist, simply because I have seen more, and the pathologists are quite grateful because it means they haven't got the problems of trying to sort out these difficult case, so they pass them on. And we never charge very much money.

Q. Are there many specialists in the UK with an interest in sickle cell?

A. Zero. I have -- succession planning is important, I have -- the HIV and the maternal work is sorted out and I approached one of my younger colleagues at St Thomas's to say would you take on the sickle cases coming for coronial autopsy and to my pleasure he said yes. I said read the guidelines, show me your first few and just get on with it and learn on the job and he is doing well, and that is how you do it.

Q. I want to ask you some questions now about sickle cell disease. Can you explain to the Chair, can you help us to understand what is sickle cell disease?

A. Sickle cell disease happens in people who have two chromosome abnormalities on the same allele, I can't even remember which chromosome it is now, and it really doesn't matter. It means that their haemoglobin A is abnormal because they have both genes wrong, one from

the mother, one from the father, and it means that the red cells being produced in the bone marrow have a very short life expectancy. Normal red cells that people who have not got sickle cell disease or thalassaemia -- but we are not worried about thalassaemia today -- should last about 100 to 120 days in the circulation before they are basically worn out and they are phagocytosed and taken away.

In sickle cell disease the red cell lifespan is something like about eight or ten days, it depends very much on the concentration of haemoglobin A, haemoglobin sickle and others because haemoglobin is quite complicated, so every patient is -- every person with sickle cell disease is different, it is not a uniform process. But you can talk about it collectively in terms of probability.

Until, really until about the 1980s, medical management of people with sickle cell disease was not very good, and most people across the world who had sickle cell disease did not make 20 years, they died within the first two decades of life. That has completely changed, we have prenatal diagnosis, we have natal diagnosis, as I am sure you know, so we know everyone who has sickle trait or sickle cell disease or thalassaemia. We have medical management processes in

Britain which I think are better than most other countries, certainly better than America, and people now live to a good age. So the average age of death of people with sickle cell disease from sickle cell disease has gone up to 40, 50, 60, 70 and we are now in the very interesting era, rather like we are with HIV, of looking at the impact of a chronic process going on, sickle, managed medically with old age. But that is not our concern here today.

Q. So how is sickle cell disease managed then?

A. It is managed by specialist haematologists, it is very focal in Britain because the epidemiology is important. Most people with sickle cell disease live in London, particularly south-east London and West London, in Birmingham and in Manchester but in many other places there is no one around at all. So that is where the expertise is concentrated, and I was very lucky being at Guy's and St Thomas's and King's to be in the epicentre of sickle in Britain.

Q. Can you help us to understand why it is that there is this particular demographic in London for sickle cell disease --

A. Oh, Christ, that is called immigration history. I don't think we need to go into that.

Q. But is sickle cell disease associated with being of

a particular background or race?

A. Yes. Most people with -- the sickle gene arose evolutionarily, if I can use that word, somewhere in West Africa and spread as people spread. And the current belief, which I have no reason to contest, is that it conveyed an advantage in defence against malaria; if one has the sickle gene and even sickle trait you have some protection against having a severe infection of malaria and dying. So in that sense it was successful. Unfortunately sickle cell disease is itself not nice.

Most people in the world with sickle cell trait or sickle cell disease are from black Africa, to use the term very generally, particularly West Africa. There are others as well that I am sure you know, that occasionally white people, blonde people, non-black people, get sickle, particularly in North Africa: Tunisia, Libya around there. India also has a lot of people who are not of African origin with the sickle gene. It stretches through the Middle East as well, but the great majority have a direct link with black Africa.

Q. Can a person with sickle cell disease who is receiving appropriate professional medical treatment lead a fairly ordinary life?

A. Yes -- yes and no. It is not a nice disease to have. The treatment is -- I don't treat people with sickle and I am therefore relying on what I know from what my colleagues told me and what I read. There is a medical treatment, you can take a drug called hydroxyurea, which has an interesting effect on the type of red cells produced in the bone marrow which is beneficial; regular blood transfusions to keep the haemoglobin level up; prophylaxis against significant infection such as pneumococcal sepsis, which is a particular predilection for people with sickle cell disease because they have lost their spleen usually and you need the spleen to protect against pneumococcal, so there is penicillin or other antibiotics and vaccination.

And in children regular examination of brain blood flow -- it sounds slightly odd to say that, but you can do that using Doppler technology, it is not invasive at all, just to predict those people who might be more likely to have a sickle-related stroke. And all these -- and general good medical attention, just being seen by a doctor and practice nurse is a very good thing. As a result, life expectancy has shot up and now it is unusual for young people to die of sickle cell disease in this country. Back in West Africa, I'm afraid it is the norm.

Q. Will a person with sickle cell disease enjoy good health?

A. Well, yes, except they are going to have to go to the clinic for a top-up transfusion, you're going to have to be seen quite often. It is a lot of regular outpatient and occasional inpatient. Then, if the patient has a sickle crisis, which is often a painful crisis, they are going to be admitted to hospital, with a standard regime of painkillers, often opiates, blood transfusion and general management and then they go out. And many sickle patients are having regular crises, you know, like monthly or bi-monthly or sometimes even weekly, and in no way can you say their life is normal.

Q. What is a sickle crisis?

A. It is an event caused by sickling in critical organs, particularly the bone and the lung. In the bone it causes the bit of bone to die, which is incredibly painful, so that is a painful sickle crisis. Affecting arm, legs, other bones as well.

In the lung, and we will be coming to this in relation to sickle cell trait later on today, it causes the blood vessels in the lung to block up for one reason or another and produces an acute chest syndrome or an obstructive problem in the lungs which means that the cardiovascular system doesn't work and they are in

great risk actually of dying.

Q. What would cause a sickle crisis?

A. Well, yes, lots of things. Dehydration is an important one, because if a person is dehydrated then the concentration of sickle abnormal cells in the blood goes up; a change in the acidity or alkalinity of the blood; a coexistent infection at the same time; operations; immobility; and probably stress. There are multiple factors. But that said, most of the sickle crises, particularly in younger people, just come out of the blue, they just happen, and the sickle doctors are well aware of this. There has been -- a great problem until I think things have got better in the last 20 years is that a sickle patient presenting to a hospital which simply doesn't see it, they won't know what to do. But now the word has spread, and haematologists are much better at sort of taking these patients and looking after them properly.

Q. You said that a sickle crisis can be a painful event?

A. It is very painful and can be fatal.

Q. And can be fatal?

A. Yes.

Q. And it would require hospitalisation?

A. Hospitalisation and specialist care. This is not something for, you know, ordinary doctors to tackle

because they won't know what to do. Although there are of course guidelines now, again everything is much more regularised and you can look it up instantly: what do you do if someone has a sickle crisis? You do A, B, C, D, and then -- as a sort of start off, and then ask for specialist help.

Q. Why might a sickle crisis prove fatal in an individual?

A. A bone crisis, where bone infarcts and is very painful of itself is not going to kill you. The main cause of death is going to be cardiovascular and it involves particularly the lung, and if you -- blood, as you know, circulates around the body, it goes from the heart, the right side of the heart to the lungs, becomes oxygenated, comes back to the heart, the left side pumps it out under a high pressure and it then supplies blood and everything else to the rest of the body.

In the lungs if that circulation starts blocking off because the sickle cells which themselves -- sorry, the red cells sickle and they sort of knot together to form an obstruction in the smaller bits, it can simply bring the lung circulation to a halt and if the lung circulation is brought to a halt, the heart stops. It is as simple as that. That is called an acute chest crisis and it has a characteristic clinical presentation, characteristic x-ray presentation, and if

you see it under the microscope, sadly if they die, a characteristic pathology appearance as well.

Q. What would you see then in the red cells when sickling occurs?

A. I am not sure how much you have had on this basic physiology of sickle but red blood cells are round, biconcave, so they have little dimples on either side, and they are sort of 10 microns across. In sickle because the haemoglobin in the sickle cell polymerises and fixes itself in a different arrangement, it actually means the cells lose their round contour and they look like sickles, bent and longer with pointy ends, and they are rigid. The point is they don't bend like ordinary red cells bend, so they are going to block up small blood vessels really quite quickly actually, and if that happens in the lungs and is not managed and sort of reversed fairly rapidly, it can be fatal. And as we will come to this can happen exceptionally in people with sickle cell trait as well as sickle cell disease but it is much, much more common in sickle disease.

Q. Let's move on now to sickle cell trait, you have explained what sickle cell disease is, what is sickle cell trait?

A. I mentioned that you have sickle cell disease if both alleles of the critical chromosome have the wrong

haemoglobin A, and you have one from the mother and one from the father. In sickle trait one of them is normal haemoglobin A and the other one is haemoglobin S, sickle, so instead of having haemoglobin SS, which is sickle cell disease, you have haemoglobin AS: A being normal and S being sickle. So it's a sort of halfway house.

Q. How does that differ then in terms of the way that a person can live their life depending on whether they have the full disease or whether they have the trait?

A. There are something like -- I looked it up once -- at least 300 million people with sickle cell trait across the world, and in this country something like 1 in 78 newborns has it because we have an ever larger immigrant population. That is the national average, obviously different places have different rates. And the huge majority, the vast majority of people would (a) not necessarily even know they had it and (b) will suffer nothing. But there will be certain circumstances where that is incorrect and there are problems.

Q. Okay, so you say that in the majority of people suffering from sickle cell trait the individual won't know that they have it?

A. Yes --

Q. And --

A. -- and they don't need to do anything different in their lives at all.
Q. And it will have no effect?
A. Correct.
Q. Are there circumstances in which a person with sickle cell trait may find themselves unwell as a result of having that trait?
A. Yes.
Q. Can it in fact in some circumstances be life limiting or have the potential to cause death?
A. Correct.
Q. In what circumstances?
A. Let's leave out the police custody business. The most important contribution to this whole area of where sickle cell trait can impact on life expectancy has come from two sources. One is from military recruits, particularly in America, and secondly from sports, in other words, high stress and high strenuous exercise.

That can take you to -- it's a very famous paper written by John Karch published in 1987 in the New England Journal, which I am sure you have seen reference of, looking at why American army recruits die, and they looked at many, many years' worth, they looked at millions, it's these huge epidemiology databases. And they looked at -- and when military recruits, a large

proportion of which are black, as we know, or a significant proportion are black, and they compared them with white people.

Now, white people have normal haemoglobin AA. The black people here, a proportion would have haemoglobin AS, none of them would have had haemoglobin SS, sickle, because if you had sickle cell disease or SS you wouldn't be admitted into the army. But AS was an admission. That is an American thing. In Britain we did it slightly differently. I will come to that later. They looked at why they died, taking clinical stories, autopsies, and everything else. And it became quite evident that when soldiers died you could weed out the ones who had a natural disease, who had been murdered, who had had accidents and drug overdoses and all that, you can take them out you and you just look at the soldiers who died who had no obvious cause of death, they just died. And statistically it was an extraordinary discrepancy in looking at the populations just in black people who are AA, normal, versus AS, sickle trait and the AS sickle trait soldiers who died with no obvious cause were, as epidemiologists say, grossly over represented in terms of the numbers. There was a relative risk of about 30.

So if you were a recruit in the American army, and

you died, then -- that is the wrong way of it putting it, if you were a recruit in the American army there was a 30 times greater risk of dying if one had sickle trait compared to having normal haemoglobin AA. This is among the black ones. The white soldiers, again there will be a small number of unexplained deaths but AS was grossly overrepresented.

Karch and colleagues and the American army were extremely interested in this and they were trying to work out why this happened. And when you drill down to what had actually happened when these people died, it was on exercises, where the soldiers were working basically in high temperatures, and dehydrated and they -- the prime recommendation coming out of those reports was not stop exercises, you can't do that, because armies are based on exercises, but please make sure that the recruits are adequately hydrated, giving them drinking water. That turned the corner and the rate of deaths, these unexplained deaths in sickle trait soldiers/trainees, dropped.

Q. You mentioned military recruits and also sports --

A. Yes.

Q. -- and in association with strenuous exercise.

A. Yes. Now, the sports is derived not from mass epidemiology but from individual case reports because

you don't get thousands of marathon runners together and do things and there were lots of case reports, and there still are occasionally, of people with sickle trait working out -- and again it comes down to heat, exhaustion, dehydration, marathon runners, things like that -- and just simply dropping dead. And the conclusion was -- and when you did the autopsy they had the acute chest syndrome. One has to say that the particular focus on the chest syndrome came from looking at individual cases of mainly sportsmen because they were going to be looked at very carefully, whereas Karch didn't go into the details of the pathology in his review of millions of people.

So it became quite evident, and this is the 1990s we are talking about, that a small proportion of athletes with sickle trait undergoing extreme stress, and in hot and dry circumstances, had a risk of dying, and they died of the acute chest syndrome. In other words, although they had sickle trait and not sickle disease they had enough sickle gene in their red cells for the red cells to sickle, block off the lungs, as a result of which they died.

Q. Is that what is meant by acute chest syndrome?

A. Acute chest syndrome, ACS. It's one of those standard acronyms in haematology.

Q. So papers were published by Karch in relation to military recruits and in the 1990s in relation to sports and strenuous exercise; where did these reports take medical understanding and medical thinking around the risks to a person who lives with sickle cell trait?

A. It had an impact on how military recruits are treated and I -- in fact in about 2005 the British Army asked me to go and talk to them about sickle trait in their recruits because we have a lot, particularly people coming from the Caribbean islands, there are a large number of recruits in the British Army -- or there were then anyway -- and should they be excluded from admission. And I think the RAF did exclude -- I don't know whether it still does -- potential pilots if they have got sickle trait.

But anyway the army didn't exclude them and they were learning from the Americans, and I just talked generally about the epidemiology, the pathology, and just the general recommendation is particularly on hydration and just be careful. I suppose also if a soldier who happens to have sickle trait is feeling very unwell on an exercise, don't make him carry on -- or her -- stop and do something. I think that was another lesson actually came out, the sort of the bullying thing, push yourself harder and harder and

harder, don't do that because it can be dangerous. And from the military and from the sports information, this sort of information, this dripped down into practice. I don't know what the current guidelines in the army are now at all, you probably are better informed than I am. But it is really just common sense stuff.

Q. In the cases in which autopsies were performed, certainly in relation to the sports people, acute chest syndrome was found to be the cause of death --

A. Was the common factor, absolutely.

Q. The common factor. And in the military exercise were autopsies carried out too or was this a desktop review of deaths some time later?

A. Yes, they are, and I have experience of that and a case we may discuss of a soldier who died on exercises in 2002 I think, or it might have been earlier, who died of acute chest syndrome on exercises in a very hot summer up north -- actually south of here, sorry.

Q. In both studies then, the military one and the individual studies in relation to the sportspeople there were autopsy findings --

A. Yes, there were.

Q. -- that were consistent with the acute chest syndrome?

A. It's an interesting point, you might think that one would learn lessons very quickly from autopsy findings;

not so, because how is the information going to be broadcast? One of the defects of the England and Wales coronial system is the autopsy report is private, it belongs to the coroner and no one else, and in an awful lot of cases -- this is not just relating to sickle but lots and lots of other circumstances as well, where you think it would be rather a good thing if these reports were collectively published so people would read them. But, no, that is against the law.

But enough of collections. And talking about these things and meetings and looking at cases and just -- and once a pathologist has come across one of these cases they don't forget it, they will remember that, so the next time it is easier for them to evaluate. We all learn on the job frankly.

Q. Can you help me to understand the mechanisms that are at play here, and how it is that exercise, high temperature, dehydration, exhaustion in a person suffering or a person who has sickle cell trait can result in a sickle crisis, acute chest syndrome and death. What is the mechanism?

A. Let's just go back to the basic simple physiology. You have a volume of blood in which -- with red cells in and both in sickle cell disease where you have SS, and sickle trait where you have A, normal, S, in the red

cells, there will come a point if you stress that blood that the cells will sickle. What are those stresses? Temperature, high or low; dehydration, in other words the concentration of the sickle haemoglobin goes up; acidosis, that is the pH drops. I mentioned dehydration, yes. And it is not so difficult to see that you can get into a situation where these factors come together, it is multifactorial, and there is a tipping point at which the cells then suddenly sickle. In every person it will be a different tipping point, a different threshold for these various promoting factors. And sickle cell disease is quite easy to see how that can happen almost spontaneously because they have no normal haemoglobin. Sickle cell trait people are protected because they have one allele which is A and one which is S, but there will come a point or can come a point where these various factors make the blood sickle.

Q. Are you able to help us to understand what it is about these factors that causes the sickling of the blood?

A. It is the -- it's concentration, it's temperature, it's pH, acidity, just stick with those three, there will come a point in anyone or anyone's blood which contains sickle trait where you can make it sickle. The question is how much stress is required to actually make it

sickle; everybody will be different. Which is why in sickle trait these events are very, very uncommon but they can occur and it is biologically very plausible and you can make it happen experimentally.

Q. What about oxygen, does hypoxia play a part?

A. Oh yes, sorry. Thank you. Hypoxia -- lack of oxygen, temperature, concentration, acidity, oxygen concentration. Thank you. Those are the four.

Q. Will they always work in combination or could one of these factors alone take a person to tipping point?

A. I don't know.

Q. You can't say?

A. It would be sensible to say it all kind of goes together as a package but each individual person will have a different trigger level for these various factors, doing it.

Q. So when these four factors come together: concentration; temperature, high or low; pH and oxygen, in a person with sickle cell trait, they may reach tipping point and have a sickle crisis?

A. The acute chest syndrome happens and the lung circulation is compromised or stops.

Q. So should we understand that it is exactly the same course of events, as it were, as you would see in a person with sickle cell disease?

A. Yes.
Q. But the tipping point is so much higher in a person who only has the trait?
A. Very well put.
Q. But once that tipping point has been reached, they will experience the same crisis, the same acute chest --
A. Yes, they would, that's right.
Q. -- and potentially the same fatal consequences as a person with full sickle cell disease.
A. That is right, and of course once it starts happening, it is going to get worse, because if the circulation in the lung is compromised because of sickling, by definition the blood is going to get more acid because it not circulating, by definition the oxygen concentration is going to go down because it is not being re-oxygenated by the circulation through the lungs, and so on. It just makes it worse, so it becomes an irreversible process. But there will of course be a point where you could reverse it quickly.
Q. So before that tipping point that you have mentioned is reached, then the crisis -- I suppose before it gets to the crisis, the process could be reversed and would that require medical treatment or could it be as simple as having a glass of water and taking a few deep breaths?
A. Yes, and getting out of the sun and stopping exercising.

Because when you do strenuous exercise, whether it's part of a sports programme, part of an exercise, or running away from the police -- and I have seen this in one or two cases -- the muscles are extremely active and what do they do? They produce lactic acid because of anaerobic metabolism, so strenuous exercise is a sort of pro-acidotic feature simply because that is what muscles do, it's their normal physiology.

Q. You mentioned that hypoxia is one of the four contributors?

A. That is why pilots in some countries are not allowed to be sickle trait -- well, the other way around, you can't be a pilot if you are sickle trait because up in the stratosphere the oxygen concentrations are less than they are on the surface. Occasionally there do appear to be pilots who have sickle trait -- I have seen reports -- who have suffered a sickle crisis and died. But as I say, in Britain I don't think we let pilot trainees be sickle trait.

Q. If I could digress for a moment you did say earlier that a person living with sickle trait might not know they have it. Clearly pilots are screened and I think you mentioned new born babies are screened nowadays, is that right?

A. They are in England and Wales, I have no idea -- I am

pretty sure they must be screened in Scotland as it is a UK programme. It started up within the last 20 years, it's done as part of the Guthrie test. When a baby is born, heel prick blood is taken and screened for loads of things and they added on sickle, and HIV by the way, some time in the last 20 years. That therefore goes into the GP records and so on and so forth.

Q. A person over the age of 20 then, who perhaps hasn't been screened as a new born --

A. Or an immigrant coming in.

Q. Yes.

A. Absolutely. No reason why they should have been.

Q. They could be living with it and not know that they have it?

A. Oh, yes.

Q. Would it be reasonable to assume that unless they have had some form of ill-health or crisis that has resulted in them getting medical attention then there might not be anything in their medical records one way or the other?

A. Yes. That is right. We all -- a lot of us have blood tests for one reason or another but there is no particular reason why sickle screen would be part of a routine non-programmed -- well -- no, that is right~...

Q. Returning to the tipping point in sickle cell trait I would like to ask you a few more questions about that. You said that it varies from person to person, is it a very individualised thing?
A. Yes, because everyone will have a different concentration of sickle in their blood. Same as with sickle cell disease, no two sicklers are the same. They have different tipping points for these things. But their threshold is much lower because they have far more sickle haemoglobin going around.
Q. So could a set of circumstances, let's say running a marathon on a hot day, that might cause one person with sickle cell trait to reach tipping point simply not affect another person carrying the same trait?
A. Absolutely right, absolutely right. Correct.
Q. In terms of the contributors to the mix that can lead to the tipping point: concentration, temperature, pH and oxygen, can I ask you some questions about oxygen and hypoxia in particular. Is there any correlation between the degree of oxygen deficiency or hypoxia and the amount of sickling that you might see or the rapidity with which a person will reach tipping point?
A. I am really not the right person to ask this. I am sure there are some sickle biologists who can give you a really good answer. But all I can tell you is the

lower the oxygen, the more likely sickling will take place.

Anticipating, I am sure, what will come up later on, if you look at autopsy of people who have sickle cell trait you will find generally a certain amount of sickle red cells in them. Now, that doesn't mean they sickled in life but of course after you die the oxygen tension in autopsy blood in the body goes down, and the pH goes down as well because that is what happens as part of decomposition. That is why you see sickle cells in sickle cell trait, which is a clue that there is a sickle process, it doesn't obviously mean in most cases that it is anything to do with why they died, it happened after they died.

This is part of the great problem in interpreting autopsy pathology in terms of sickle cell trait, is how much happened before they died and how much happened after they died. That is really the critical question in this case and in many of the other medicolegal ones as well.

Q. I'm sure and it's a question to which we will return.

A. We will come on to that.

Q. Let's go back to paragraph 4 of your statement which is still on the screen. You mention having carried out 100 autopsies now on individuals with sickle cell disease?

A. More than 100, when I last did a count actually it is about 110. But that is it, I won't do any more.

Q. Can you explain what you would tend to see at autopsy if you carry out an autopsy on a person who has died from sickle cell -- perhaps I should -- let me reword that -- who has died as a result of the acute chest syndrome provoked by a sickle crisis; what would you expect to see at autopsy?

A. The acute chest syndrome, what you will see is in the lungs, in the circulation of the lungs you will see a lot/most/all the red cells sickled. Now, you want to be very careful here because we are not talking about the big blood vessels like the main pulmonary artery that gets blocked off by thrombosis -- deep vein thrombosis and embolism, we are talking about the smaller vessels: the arterioles, the capillaries, which is where gas exchange occurs, and the postcapillary venules, the very small vessels, the ones you can't see with the naked eye but under the microscope you can see them very clearly. In those vessels in what we accept is an acute chest syndrome case you find these small vessels are greatly dilated, so their diameter, their calibre is many times bigger than it should be and all the red cells are sickled and are sort of matted together. That is what you see. Necessarily it's

a subjective quantification, but that is what you see.

Q. During the person's life, when they go into crisis and start to develop this acute chest syndrome, what is the effect on their respiratory system of the red cells sickling and matting together; what impact does that have?

A. Okay, well subjectively they become short of breath, as the oxygen -- the whole point of the lung circulation is to get oxygen from the air into the body and this is going to be compromised when sickling of red cells starts in significant quantities. Obviously there is a huge amount of redundancy, you can sickle quite a bit and it won't matter but it will get to a point where it really does matter and the oxygen therefore in the blood starts going down. This would simply make the sickling worse and it becomes a sort of ... I have forgotten what the phrase is now but it sort of gets out of control.

So shortness of breath, fever, because the temperature goes up quite quickly as an automatic response. I am trying to think what are the other clinical syndromes. You see abnormal things on x-ray, if you do one, or MRI scans, you can see basically the circulation looking a lot more solid, it's getting more solid because it is filling up with more sickle red cells. And agitation, obviously subjectively it is not

very nice and patients who are sickle cell disease ones know very well when they are going into a chest crisis because they have done it before and they say, "Oh, this is what is happening again, help". And of course the treatment is oxygen, hydration, blood transfusion, basic supportive care, and in nearly all cases they will be rescued but not always.

Q. In those cases where rescue fails what is the actual mechanism that brings about death?

A. Lack of oxygen, via the lungs into the body, so essentially you die -- I was about to say of asphyxia, that is -- let's leave asphyxia out, that is a rather different process. Ultimately it is because the heart needs oxygen to keep beating, and the brain needs oxygen to keep you awake. And those two organs are the ones. If the kidneys or the liver fail that doesn't matter, you don't die suddenly because of kidney or liver failure, you die because the lungs, the heart and the brain don't work. And they don't work because the circulation taking oxygen to them is blocked.

Q. Again in paragraph 4, and we have spoken about this already, you have been involved in a number of medicolegal cases, and five, or in fact you think eight previous deaths in custody or stress-related deaths amongst men with sickle cell trait?

A. Notice men, it's all men. I have not seen a woman have this, interestingly.
Q. Can you explain that?
A. Hmm. There are fewer women in the army. Exercise-wise I don't suppose there is any difference at all. As to deaths in custody, I would be terribly simplistic, and could be shot down very rapidly, is that they don't get arrested as much as the men do, for lots of social reasons.
Q. In terms of sickle cell disease, is there an equal distribution of the disease between the sexes?
A. Sickle cell disease, men and women are the same. In disease.
Q. Insofar as you know, because I hear that a lot of people who have the trait won't know about it, but have you any reason to suppose that the trait is not equally distributed between men and women?
A. No, the trait is very well distributed. One interesting way you pick up people having sickle trait is that when the women deliver and you are, for one reason or another, asked to look at the placenta you can see the placenta is full of sickle red cells and you say, "Ah, that's sickle trait". Well, you say, "Ah, that is sickle", and you go to the haematology register and see where they are and they say, "No, this patient has

sickle trait" or maybe, "This patient has sickle disease and we knew that anyway". But if it is trait it will be often a new finding which may or may not get disseminated to the patient or their healthcare workers, who knows? We produce reports, we have no idea who reads them.

Q. You have mentioned five or perhaps eight cases, deaths in custody or relating to stress in men with sickle trait and there is one that you discuss in some detail in your statement and it's the case of Prince Fosu, I don't think we need to go to the paragraphs in your statement but can you tell a little bit about that case?

A. Okay. This is a young Nigerian man who was, I think, to be deported for immigration illegalities, whatever, and he didn't want to be deported, and so he indulged in the deportation centre in a dirty protest, rather like in Northern Ireland in the troubled days. He took his clothes off, he didn't drink much, he didn't eat anything, he smeared faeces all over the walls and -- basically as a protest, and said, "I do not want to be here, I want to stay in England please". And despite him obviously deteriorating, it would appear that not money was done to try and sort of bring him back. I don't know the great details, because I only got involved when it came to the inquest because he died,

and the pathologist who had done it was a forensic pathologist because normally, if there is a death in custody you wouldn't get a non-forensic pathologist to do it because the suspicion is that the authorities may have had a role in that person's death, so that is forensic. That is fine. And the forensic pathologist had said he died of ... actually, it is in this account here, isn't it? Can I actually get the phraseology correct?

Q. Absolutely, it is paragraphs 49 to 51. Perhaps we can just move to those now on the screen as well.

A. Thank you. Paragraph 51 the original pathologist said he died of cardiorespiratory collapse associated with sickle cell trait. This is in the West London coronial jurisdiction and the coroner at the time obviously -- well, I must backtrack. The inquest took place in 2020, the death happened eight years earlier, there had been eight years of legal arguments between the family, the police, the legal authorities, and so on. So finally an inquest happened and it was one of those inquest rooms where most of the people were lawyers, a bit of family, and me and the coroner. The original pathologist had said this was cardiorespiratory collapse associated with sickle cell trait because he had seen in a couple of organs some sickle cells.

Q. Can we scroll down please to 51 for the benefit of anyone who following this.

A. The coroner then said, "Look, this doesn't look bad", and because of all the problems with the police and particularly the prison authority or whoever runs detention centres -- and I am not entirely clear who actually is in charge of these things -- it was sort of prima facie case where they weren't doing their job very well otherwise he shouldn't have died. So he wanted supporting, and I was asked -- and also Rick James from Wales, from Cardiff, was also asked to look at the case.

Rick, who is very good, said the cause of death was cardiovascular collapse, dehydration, malnutrition hypothermia and mania -- if we add that one in as well -- although he was a bit dodgy on the mania because pathologists are not allowed to diagnose mania, we don't know what it looks like. He put sickle trait under a contributor in part 2 -- you are well aware of how death certificates are organised, part 1 is the main reason why they died, part 2 will have factors, often several, which made them die of the main cause when they did rather than last year or next year or something like that. In other words, a sort of contributor in terms of timing.

I looked at it and I said actually I don't think

sickle is relevant here at all. Why? Very simply because there was no acute chest syndrome histopathology at all. The sickle red cells could be seen in the kidney, I think, or something like that, and that was irrelevant. If the sickle was going to be relevant to his cause of death it would have to have blocked up the lungs a bit and a lot. And for that reason I said I don't think sickle is relevant here and the coroner believed me.

Just to backtrack two stages, it is very interesting the forensic pathologist, who I won't name but is a highly experienced person, simply doesn't know and didn't know anything about sickle. This is the main problem actually when these cases interact with death and authority, that the pathologists have no real experience because these deaths are uncommon, and they don't know how to handle them.

Q. If we can just look at the very bottom of what we see on the screen, where you repeat your opinion:

"... was that sickle trait did not play any role in death; rather it was cold, hunger, malnutrition and neglect, because on histology there were very few sickle cells. He hadn't sickled enough."

A. Particularly in the lungs.

Q. "So it wasn't relevant to his death. The inquest jury

concluded that the medical cause of Prince's death was 'A sudden death following hypothermia, dehydration and malnourishment in a man with psychotic illness, contributed to by neglect and multiple very serious failures."

A. That was a very fair and correct outcome. I just want to make one point about the word "neglect" because it is a very, very difficult concept, and the advice the coroners give pathologists is never use the word neglect in your reports, because coroners have a different view as to what neglect is compared to the general population, in which I count myself.

Q. I see.

A. In other words, it's very tricky area. For obvious reasons.

Q. I want to move on now to ask you questions about your involvement in Mr Bayoh's case.

A. Yes.

Q. Just to be clear, you did not yourself carry out a post mortem, nor were you present when Drs Shearer and Bouhaidar carried out the post mortem on 4 May of 2015.

A. I was -- in fact this morning I was trying to remember why I got involved in this in the first place, and the answer is I was approached by --

Q. Perhaps I can help with you that and we can bring up

an email that I think you received from the Crown Office on 29 May 2018 which is COPFS 02468.

A. Les Brown, that was it.

Q. If we can scroll down to the substance of the email, please. We can see 29 May 2018 from Les Brown to yourself. There are some pleasantries, there is reference to the PIRC investigators delivering histology slides to you on 4 June and also four reports that will be sent to you for your consideration, although I don't think we see a reference to what the reports are in the body of the email. If we scroll down a little bit we will see what you were asked to address:

"I would be grateful if you could address the following areas in your considerations which may lead to the instruction of a formal report."

So this was -- this wasn't a formal letter of instruction, it was the Crown approaching you to see if you could assist, and suggesting that there might be an instruction further down the line. There are four bullet points:

"To comment on the supplementary report by Liz Soilleux and in particular her conclusions on the cause and mechanism of death and the significance of sickle cell carrier status.

"Whether the apparent sickling of cells occurred

ante or post mortem.

"From your own experience conducting autopsies in persons who are sickle cell carriers the extent to which sickling of cells is noted generally.

"Whether it is possible or likely that sickle cell carrier status contributed to the cause and mechanism of death in this case having regard to all of the relevant factors set out in the reports."

Can we scroll down a little further just to be sure whether there's anything else (inaudible) includes the email. So you received that email and you would have received the histology slides on 4 June. What did the slides contain?

A. Right, let's find the right bit.

Q. I don't want to look at the slides at this point, or your findings, I am just interested to know in general terms what it was that you were being sent. What are histology slides? Help the members of the public who are perhaps listening to your evidence to understand what histology slides are?

A. Oh, sorry. Histology is the study of tissues down the microscope, it is a process that was invented in the middle of the 19th century in Berlin by Rudolf Virchow whereby you take pieces of tissue, any tissue, and it is preserved, fixed, otherwise it will disintegrate. It is

processed so that you can cut very thin slices of it, and when I say thin, I mean slices of tissue that are 4 or 5 microns thick. A micron is a thousandth of a millimetre, so this is very thin stuff indeed.

And it has chosen that because cells are of a certain size so if you look at tissues down the microscope you can actually see them properly. So you have these 5 -- 4/5 micron thick bits of tissue, they are put on a slide, you then add various vegetable dyes to it to highlight things because if you just look down the microscope at tissue you don't see anything at all, it needs to be brought out. And there is a standard combination of stains called haematoxylin and eosin, H and E. So in pathology terms we are talking about what did the H and E slide look like. H, haematoxylin, goes and highlights stuff that contains RNA and DNA, which is what the chromosomes are, so the nucleus is highlighted. And eosin sort of stains everything else, and that is red. So what you are looking at in histology is sheets of cells, stained red and blue, and you look at them and you learn, simply by experience of what they are telling you.

In sickle, for example, you are looking at red cells, you can see the cells are red, we call them red actually because they are red when you stain them with

eosin, that takes up the eosin. Red cells don't have a nucleus, that has been disposed of long before they get into the circulation, and you can look at their -- how sick they look, whether they are sickled, whether they are the normal shape and size and so on.

So it's the interpretation of cells, coloured, down the microscope taken from any organ. This is the basis of all histopathology/pathology, it's the base of all cancer diagnostics as well, you are not allowed to have cancer in most cases unless someone who is a pathologist says so because they have looked down the microscope.

Q. We heard evidence last week from Professor Freemont, an osteoarticular pathologist, and he had looked at slides down the microscope and he also told us about the H and E stains --

A. Good, so you've had all that. I hope I have said the same thing as he did.

Q. I think he did. Should we understand then that these are standard slides, that H and E is a standard slide that any histopathologist would --

A. Yes, it's a universe process, the glass slides are 3 inches by about 1 millimetre thick and this is the basis of all diagnostic histopathology globally and has been really since the end of the 19th century.

Q. Let's look at your reply to this email at COPFS 03682.

We know that you received the histology slides on 4 June, and you responded to Les Brown on the same date. If we can scroll down a little to the substance of the email, thank you. Here we are.

A. Yes.

Q. "Dear Les.

"Seen the slides.

"I think that sickle trait did play a role in the death, along with all the other possibilities (drugs, excited delirium, restraint, posture et cetera). Not possible to quantify the contribution of course.

"Do you want my short report with all the duty to the Court material plus a mini-CV, or will a straight letter to you do?"

If we scroll down a little more, please:

"I will not put this in my report, but from what I can see of the scenario: the police officers involved should not be prosecuted assuming they approached and restrained BAYOT [I think that should be Bayoh] in the normal approved fashion, appropriate for the perceived risk.

"There is a discussion to be had concerning whether a differential police approach is needed for subjects who are known to have a predilection (albeit uncommon) for sudden death under stress, even if they knew the

fact in advance. An endless moral maize there ...

"Sebastian."

I want to ask you a few questions about this, please. Can I take you at this juncture to paragraph 61 of your statement and we will look at that and then I will ask you some questions based on what is said in this email and what is said in your statement.

At paragraph 61 you say:

"I have been referred to my emails of 4 June to Les Brown of the COPFS, where I write 'I will not put this in my report'. Do you know, if I was doing it again, I certainly wouldn't write that. What I can see of the scenario has obviously changed, because, I think, when I was sent all this original material, there was very little evidence or information or documentation about restraint. The death, in a sense, was slightly more spontaneous, but as I understand from what's happened in the last seven years or so now, actually, a lot more information has come about what actually happened, and I wouldn't write that now."

Paragraph 62:

"I feel that this was an 'off-the-cuff' comment at the time."

Can you help us to understand the background and why you felt it appropriate to comment on the police not

being prosecuted?

A. I ... as I said I wouldn't do that now. If I had known all this was going to happen I would have left that out. The background to this, just for the other people who haven't had the conversation that we have had before, is that I changed -- I have changed my mind on this issue over the last seven years as to how important sickle is and I think at the moment it is less important than I thought it was in 2018 --

Q. Can I pause there, sorry to interrupt but just to say that what I would like to do is explore the opinion that you gave in 2018.

A. Okay.

Q. And then the opinion that you would give today because I do understand that you have changed your opinion, and I am keen to explore the reasons for that. If you could perhaps just bear that in mind. But I am sorry I interrupted you, please carry on.

A. No, so back in 2018 I could -- it really comes down -- ultimately it comes down to what the histopathology showed, so let me just quickly move to what I wrote then. I know it is here somewhere. (Pause). Okay, 84. If you look at the second page, it is the histopathology review.

Q. Would you like us to bring this up on the screen?

A. Yes, why not.
Q. COPFS 84, sorry we are -- if we can just orientate ourselves for a moment so having had this exchange of emails with Les Brown you were instructed to prepare a report, it is dated June of 2018. This is your report, and if we can -- we will go to the second page but for completeness if we can look at the first page, you repeat the questions that you were asked.
A. Yes.
Q. Which we have seen already in the email. And you go on to say the materials that you were -- you list the materials you were provided with, and we will perhaps come to look at that list again shortly, and you were keen that we look at page 2, Professor; is that right? Was it "Histopathology review"?
A. Yes.
Q. Here we are, it's on page 2.
A. Right. Bone marrow, no sickle cells. Brain, yes. Red neurone change -- this is essentially asphyxia-related brain damage from hypoxia-ischaemia. There is some sickling, SRBC is the standard code for sickle red blood cells. Kidney, massive accumulation of sickle cells in some of the vessels, no DIC, no casts. Liver, much sickling going on, otherwise the liver was okay. Heart ... sickling but otherwise it's okay.

I should say the heart can carry on beating really almost to the bitter end, it's often the last thing that finally gives up.

Thyroid, sickle, and adrenal, sickle. Lungs -- the crucial one is the lungs, actually, this is what it's really all about. Congested. There were six pieces. Now, the lungs have five lobes, three on the right and two on the left, so normally when one looks at lungs there should be five slides, I am not quite sure where the sixth piece came from, so I am not quite sure which bits are which lung. Six pieces sampled, alveolar oedema ... areas of sickling in veins and alveolar capillaries, I emphasise it is the small vessels that have the problem, the big vessels don't. Three out of the six have sickling, three out of six have mild or no sickling and that is it.

At that time, and it has to be put in context of what else I was told because, just stand back, in all these sorts of cases we have someone who has died and we have a putative risk for causing problem, like sickle cell trait, and it is a question of balancing all these facts out.

Let's not talk about him particularly but if someone had an obvious massive drug overdose, then a bit of sickling on the side you say is irrelevant in comparison

to that. If they had had an obvious acute heart lesion which was reasonable to cause death, sudden death, you wouldn't bring the sickle in. But the point is when I was faced with this case back in 2018, all I had was a very brief story about being arrested and then sort of dying suddenly and I didn't really get details about restraint, I had a drug toxicity report which didn't look particularly striking in terms of significant large amounts, although they may well be and I am not an expert toxicologist, and we had the sickling in the lungs and I was thinking: could this possibly have contributed to why he died because I really don't see much else? So in a sense it was -- I won't say a diagnosis of exclusion but it was a diagnosis of consideration of other factors, and I perhaps should have been more cautious and said, "I haven't had all the facts in the case, but one might consider sickling to have some relevance", and to be quite honest I think in this report at that time I overdid the importance of the sickling in terms of possibly causing lung damage, blocking up the circulation.

Later it became evident, and I think it was Nat Cary who you will be talking to tomorrow, who said, "There was a hell of a lot more restraint than that", and I am putting that phrase very loosely because that is my

memory of it. That made me think: hmm, yes, in this case I think the sickling is now less important and other factors relating to how he was managed at the time of his arrest are more important. So it's a balance. And I do really want to emphasise there is no defined case definitional cut-off point as to how much sickling matters. It can't be done. It is simply too subjective.

Liz Soilleux -- and I was possibly slightly influenced by Liz who certainly at that time I respected as a good histopathologist -- was very emphatic that sickle did play a role and I thought maybe she is right, so in a sense I slightly echoed that.

Now I wouldn't because I think other factors are more important, but I do want to emphasise there is no agreed point at which you say, "That matters and that doesn't; it's that disease, it is not that disease". It is not like many other things where it is either there or it isn't and we have agreed case definitions. This is a very subjective area, in part because of the -- because it is multifactorial, it is cumulative, there can really be no strict case definition, but also because we simply don't have enough experience. As I have said, I have seen eight cases, eight -- sorry, eight males who have died where sickle trait may or may

not have been important in causing their death, and that is a career of over 40 years, no other pathologist has seen anything like that. So no one really knows.

Q. All right. We are going very shortly to consider the information that was made available to you by the Crown and we are going to look at the opinion that you expressed in your initial report and what caused you to reach the conclusions that you did. I think we might be slightly at cross-purposes, and the fault is absolutely mine, can we please go back to the email that we were looking at a moment ago. That is COPFS 03628, this precedes your report, and this is the email that you sent to Les Brown and the Crown Office on 4 June.

A. Okay, yes.

Q. Bear with me. What I was hoping to draw your attention to, if we can scroll down a little please, was the part at the top of the main paragraph there:

"I will not put this in my report, but from what I can see of the scenario: the police officers involved should not be prosecuted assuming they approached and restrained [Bayoh] in the normal approved fashion, appropriate for the perceived risk."

I simply wanted to hear from you why you felt it was appropriate for you as a histopathologist to comment on whether or not the police should be prosecuted?

I wonder whether that was a matter that was in fact outwith your expertise?

A. As you can see, this is in a sense a private email, this is not a publishable report although it is now public. I make no apologies for that. But I have been involved in an awful lot -- as -- a significant amount of this sort of approach, and the extent to which the officers of law and order look after people, because every coronial autopsy actually starts off, usually, if it is death in the community, with the police being involved because they go and see to make sure the person is dead, so I am well aware of what might happen.

But as I say for the -- my recollection is from the information I had back in 2018 the story was that he had been restrained under what -- as I said, normal approved fashion and no more. That is what I understood at the time. And yet had suddenly died, to the surprise, I got the impression, of the police officers.

So if that is the case, I am not -- I don't know whether to say this or not, but I am not -- actually I do say it, about a differential police approach and so on. This is a very difficult area and it is really not for me to comment on, but it is very difficult when you have got someone who is behaving oddly and needs to be restrained, I assume the need is correct. And they did

what they had, I assume, been trained to do, and yet despite that suddenly he was dead. That was the version I had at the time. I learned rather later it wasn't like that and that is what made me change my mind.

Q. All right. I am conscious of the time but before we perhaps break, as we usually do, at about 11.30 am, can I just be clear that you made a number of assumptions around the need for a restraint and that officers had followed their training? You told us in the last few moments that you made certain assumptions, is that fair?

A. The version -- I don't know what I was sent. I am not even sure I have kept it and I certainly don't have it in front of me but it would have been written information -- actually it must have been -- maybe derived from the original PM report --

Q. We will look at the report shortly because your report sets out the information that was made available to you. But perhaps what it comes down to, Professor, is this: would you accept today -- and I appreciate this was a private email and not one you that ever expected to be before a Public Inquiry --

A. No, no, quite.

Q. -- but would you accept today that a comment on the police and their actions, their use of force, whether or

not they should have been prosecuted, was not really one for you to make? That goes beyond the bounds of your expertise as a histopathologist and is ultimately a matter for the Chair?

A. Yes. Full stop.

Q. All right. And indeed that prosecution is a matter not so much for the Chair but for the Crown. But the facts of what happened are a matter for the Chair and any future prosecution would be a matter for the Crown?

A. Yes. I had had several conversations on the phone I think with Mr Brown, and I have a vague feeling that this aspect may have come up, which might have been one of the reasons I put something in. But to say it is unforgivable, I don't know if that is the right word, but I wouldn't do that now. It's not right.

Q. Do you understand that in giving evidence here today you are here to assist the Chair in understanding the significance of sickle cell trait in the death of Sheku Bayoh?

A. Absolutely.

Q. Do you understand the importance of your evidence being independent and unbiased and objective?

A. Correct.

Q. And you understand the importance of staying within the boundaries of your expertise?

A. Correct.

MS THOMSON: Sir, I wonder if this would be a convenient point to break?

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

(11.31 am)

(A short break)

(11.50 am)

LORD BRACADALE: Ms Thomson.

MS THOMSON: Thank you. Professor, I would like to take you now to the report that you prepared for the Crown. You took us to it yourself a few moments before the break, if we can bring that up on screen, COPFS 84. We looked at this very briefly to get our bearings, if we can scroll to the bottom of this page, we noted earlier that you repeat there the questions that were in the earlier email. And then beneath that:

"I have been presented with~..."

And you list the documents that were made available to you to allow you to prepare your report. They are the original autopsy report; a supplementary report, from 2017, confirming that Mr Bayoh had sickle cell trait; Dr Soilleux's report and her supplementary report; and the histology slides.

If we can just scroll a little further I think that

is the end of the list of material made available to you. If we can just scroll into the second page. Yes, it is.

Did you feel that you had everything that you needed to express a view on the significance or otherwise of sickle cell trait?

A. Presumably I did at the time but in retrospect, no.

Q. In retrospect what additional information might you have found to be of benefit?

A. A more detailed account of what actually happened in the confrontation between Mr Bayoh and the police.

Q. You were not sent reports by other forensic pathologists?

A. Not at the time, I don't think. Otherwise I would have listed them.

Q. Yes. At that point in time, a report had already been prepared by Dr Cary, whom you have mentioned, by a professor Jack Crane, and several reports had been prepared by a Dr Lawler, all forensic pathologists and all of their reports pre-dated the instruction of yours.

Were you aware that other pathologists had been instructed to prepare reports?

A. No.

Q. Would you have expected to have had sight of them?

A. I can't remember. This is five years ago. But in

principle, yes.

Q. In principle, in a general sense --

A. In a general sense because this is a death in custody, yes, there would be others, because Liz Soilleux is not a forensic pathologist. So I would have expected others but I wasn't shown them until after I wrote this.

Q. You say you weren't shown them until after you wrote this, have you seen them subsequently?

A. I think I have seen Nat Cary's but actually I spoke to him because we met and he said -- he knew what I had written, and he said, "It's not so simple. There was an awful lot of restraint processes, and so on going on". And then I probably have seen his report since then. At least I know his -- I have seen his conclusions because Ms Wade presented them to me.

Q. So you were shown some material by the Inquiry as part of the process of taking your statement.

A. Yes, this was much more recently, in the last year, within the last year, not in 2018.

Q. So the reports that were not available to you at the time that you prepared your report were not subsequently sent to you by the Crown for comment?

A. Does it say so in my final statement? I don't want to contradict myself.

Q. So I don't believe it does, I think the only reference

in your statement is at paragraph 8.

A. Yes.

Q. If we can quickly pull up paragraph 8 I'd be grateful:

"I have been asked whether there would be any other material that I would have expected to see in the instruction from [Crown Office]. No, and the circumstances of the death, the autopsy report, versions of it, someone else's opinion and I was sent the histology sides also. I must have sent them back a long time ago. I have been asked if there were reports by forensic pathologists in a case like this, would I expect to see such reports. Yes, I would."

I think that is the only comment on what you had seen?

A. Hang on just a moment. I actually brought with me all the paperwork I have in relationship to this case.

Q. Is that what you have in front of you now?

A. That is what I have brought out of my briefcase.

Q. I see. Well, in that case I wonder whether we can simply park this issue for now and revisit it and you can look through your own file at your leisure over an appropriate break.

A. Okay.

Q. Thank you. But certainly so far as your report is concerned, you state in your report, if we can return to

the report, please, at the bottom of the first page that you were provided with the autopsy report, a supplementary report, and two reports by Dr Soilleux --

A. Soilleux.

Q. Soilleux, thank you.

A. And her supplementary. Yes, this is correct because I always, always put in all the documents I have seen when I am making a report. So the omission means that I didn't have them.

Q. You didn't have them, and just as a cross-check on that, if we might return very briefly to the email that you were sent by Les Brown, that is COPFS 02468. We see he says in the middle of the first paragraph:

"I will forward a total of four reports for your consideration in relation to this case separately."

Do you see that Professor?

A. I do see that, but I am not sure that I got them.

Q. There were four reports listed in your report: the two post mortem reports and the two reports by Dr Soilleux.

A. Yes, that is right. There's two from the original pathologist and two from Liz Soilleux, correct. That is the four.

Q. So les Brown refers to sending you four reports, he says he will send you four reports and you have narrated in

your report the receipt of four reports: two post mortem reports, and two by Dr Soilleux?

A. Yes.

Q. Would it have been of benefit to you to have seen the other pathologists' reports?

A. Yes.

Q. What would the benefit have been to you?

A. More detail about the confrontation between the decedent and the police.

Q. From a medical perspective would there have been a benefit to you in reading the pathologists' reports?

A. It would indeed because, as I said earlier before the break, the definition of whether sickle plays a role in this sort of death is subjective. There is no defined hard point. And the more information you have about other alternative explanations for causing a death, then the less important the sickle will be. It is really as simple as that actually, it's a balance.

Q. You weren't sent statements by the Crown Office, statements of the police officers involved in the restraint or civilian witnesses?

A. Well, I don't normally get sent that sort of stuff. But it might have been helpful in this case, and if I had had more information available I would presumably have written my report differently back in 2018. Correct.

It's certainly the sort of information that comes out in inquests, where you realise that the whole thing was actually rather different from what you were told when you did the autopsy and you change your mind, and in coronial inquest courts that is perfectly satisfactory; when different information arises you alter your perspective.

Q. Do you feel to any extent at a disadvantage not having had sight of the other pathologists' reports or statements or a summary of the events that transpired at Hayfield Road?

A. Sorry, I didn't quite get what the question was.

Q. To what extent, if at all, do you feel put at a disadvantage by not having had the reports?

A. Well, I do in retrospect, but at the time obviously I didn't feel disadvantaged.

Q. Let's return to your report, please. If we can look at the second page, at the histopathological review.

A. Yes.

Q. You took us through this very quickly before the break. I wonder if we can simply look at the significance of your findings in turn. So the bone marrow, no sickle cells; is that a matter of any significance from your perspective?

A. It is a piece of information. It's a datum.

Q. Does that information tell you anything, the absence of sickling in the bone marrow?

A. In people with -- even with people with sickle cell disease if they die suddenly, they do not necessarily show sickle cells in their bone marrow. But they might, and similarly with sickle trait. That is not the critical point. It's variable. The whole business about what happens to sickle -- red cells that contain the sickle gene and abnormal haemoglobin after death is it fraught, as I am sure you are going to bring up in more detail, and you will have seen many accounts from people who say you can place no trust whatsoever in whether you see sickle cells or not, it doesn't mean anything because they sickled after death.

To an extent that is true, I am talking from my personal experience, but I do believe that when particularly the lung vessels are extremely abnormally distended with sickled red cells, then you can take that as being meaningful because they wouldn't be so distended at the time of death if that process hadn't happened before death.

I explained earlier what the acute chest syndrome pathology is, it's distention, widening, dilatation of these blood vessels with large numbers of sickle red cells, and I do not believe that that process happens in

normal sickle trait people not under stress or anything else as a post mortem event. I believe it is premortem pathology and I will stick with that. But it is an opinion, you know. As I say there is no hard-and-fast rules, and you will find other pathologists who might say different.

Q. Before we discuss the lung findings in more detail, can we perhaps look at the other slides that you considered. We have spoken about the bone marrow. The next is the brain:

"Early red neurone change only (from hypoxia-ischaemia); some vessels have sickled red blood cells."

What does that tell you?

A. It means he has died because his brain neurones have suffered from lack of oxygen, lack of blood, and he is brain dead or is becoming brain dead.

Q. "Kidney - very congested; some parts just normal red blood cells ... but much massive SRBC accumulations in arteries, capillaries ... and veins. No disseminated intravascular coagulation ... no casts in tubules."

What does this tell you?

A. Right, this is -- most of my kidney histopathology reports read a bit like that because these are things that you look for. DIC means there is no blood clots,

there is no thrombosis going on inside the glomerulae which you can see under certain circumstances in sickle. The casts means that there is no muscle necrosis that has gone on for long enough for muscle protein to come out into the blood, go to the kidneys, and deposit in the tubules, and we call them myoglobin, myoglobin being the muscle protein casts. That is very commonly seen in people who have crush injury of muscle, like someone who has been -- lying on a concrete floor, get crush injury of the buttock muscles and thigh muscles and that injured muscle ends up in the kidney, you can see it. So I just simply put that in as relevant negative. I believe in stuffing my histology reports with lots of significant negatives because they answer questions.

Q. Should we understand from what you have just said that when you were looking at the histology slides and commenting on them, you are not limiting your analysis to the presence or absence of sickling, you are looking at the tissue more widely?

A. Yes, but particularly I am putting other things that I have seen in the context of sickle in general deaths, just to say these are there and these are not there. Also, to be quite honest, to make it look more detailed than the original pathologist to show that I know more than they do.

Q. Returning to the kidney, where you say:

"... much massive [sickled red blood cells] accumulations in arteries, capillaries ... and veins."

What does that tell you?

A. It means that around the time of death he sickled a lot in the kidneys. The obvious question is how much happened before death and how much happened after. Can you tell? No.

Q. When you say much massive SRBC, is that technical language?

A. No, it's --

Q. What is massive?

A. No, no, it is just subjective. It means lots.

Q. Lots. Okay. Moving on to the liver:

"Severe congestion of vessels and sinusoids; much SRBC present ... architecture normal, no steatosis~..."

A. Fatty change indicating chronic -- no, recent alcohol or other drugs:

"... no necrosis. Bile ducts normal."

Q. In relation to sickling, again you have recorded much SRBC present?

A. There's lots, yes, that is right. I don't think I photographed this case, which perhaps I should have done, it might have been helpful actually, so I can only go from the words, I haven't reviewed the slides since

2018.

Q. I see.

A. I don't know if they are still available.

Q. Moving on to the heart. You observe:

"Right ventricle normal; left ventricle normal apart from acute contraction bands scattered (from resuscitation)~..."

A. Yes.

Q. "... no scars, no acute ischaemia or myocarditis; much SRBC in vessels and capillaries."

So again, we have more sickling in the heart?

A. That is another variable thing. Some sickle trait and sickle disease people have lots inform the muscles when you see them when they are dead, some don't. It is variable. Again, it is not a critical thing because sickle trait or disease does not damage the heart acutely. So it doesn't really matter what is there.

Q. If we can scroll down a little please, after the heart is thyroid:

"... normal apart from much SRBC."

And you say the same in relation to the adrenal -- is that the adrenal glands? What conclusions again do you draw from that, simply that sicking is present?

A. It just means that all through the body is sickling.

Q. Okay.

A. These are the organs of a sample so they have a separate paragraph, one doesn't normally necessarily take samples of thyroid or adrenal if to the naked eye they look perfectly normal, but they were there so you comment on them.

Q. Finally the lungs:

"... congested with distended vessels; 6 pieces sampled~..."

You explained earlier ordinarily it would be five:

"... areas of alveolar oedema and no associated sickling; areas of sickling in veins and alveolar capillaries; 3/6 pieces have this sickling, 3/6 have mild or no SRBC in the non-oedema areas. No thrombi, no alveolar haemorrhage."

Help us to understand the import of your findings in relation to the lungs?

A. It is a description -- it's a semi-quantitative description of how much sickling is going on in the relevant areas of the veins and the alveolar capillaries, and half the samples taken did have it and the other half didn't.

Q. What does that tell you?

A. Well, this is the problem actually, I remember thinking about this at the time, thinking well, we don't have another good reason really as to why this person has

died the way he did. Remember, I didn't have the complete story. So I thought in that context, and also possibly biased a little bit by Liz Soilleux's previous report, who emphasised that a lot, I thought perhaps the sickling did have a role here.

Q. Can we turn to your statement for a moment please and look at what you say there about your findings in relation to the lungs. Paragraphs 15 through to 18. 15, this is in relation to the lung, you say:

"This is the crucial one in people who have died under restraint and exertion and you can say that sickle really is very important here."

Why is the lung the crucial sample?

A. Sorry, I didn't get the question.

Q. Here if we scroll up just a little bit we see we are talking about the lung tissue?

A. Yes.

Q. And then at paragraph 15 you say this is the crucial one --

A. Yes.

Q. -- in people who have died under restraint --

A. I have explained that several times already.

Q. Well, do indulge me. Why is it that the lung tissue is the crucial sample?

A. From my experience and from the literature of people who

have died under restraint and/or exertion, and there is no other obvious cause of death, it is because the sickling in the lungs is why they are dead.

Q. Okay. Sorry did you want to add to that?

A. No, not particularly.

Q. Paragraph 16:

"Now, in this particular case, you will notice I have been quite precise, that whoever did the autopsy took six samples of lung, I assume from six different parts of the lung.

"The lung has five lobes, so probably it's one piece from each lobe, plus one extra, but I don't think it was stated exactly where they come from. Some bits don't have any sickling and some bits do, which is why it's not quite so clear-cut as some of the other more obvious ones, where all the lungs are very sickled, and you can say this is a classic example of the scenario called the Acute Chest Syndrome, and this is well known in sickle disease."

A. The word "this" here applies to a classic one, where it is all sickled. Not "this", as in this particular case.

Q. I see. I see. So with acute chest syndrome you would expect sickling throughout the lung?

A. Yes.

Q. Whereas here you have found --

A. Partial.
Q. -- sickling in three out of the six --
A. Exactly.
Q. -- samples taken?
A. Exactly.
Q. Can we look at the following paragraph please again in relation to the chest:

"It obviously happens much more commonly in people with sickle disease, and what it means is that in the lung blood vessels, the cells sickle, and they simply can't move. If you can't have blood moving through the lung, the lung stops, the heart stops, and you die. It is called a crisis due to Acute Chest Syndrome. Mr Bayoh has some aspects of the Acute Chest Syndrome, which is why I think sickle contributed. Having thought this through, I would now say my bottom line is that I think we should probably move sickle trait down in the cause of death list from being in part 1 into part 2, as a contributor but not the main cause."

We will come in the fullness of your evidence to look at the change in your opinion, but focusing on the first part of that paragraph, you say that Mr Bayoh has some aspects of the acute chest syndrome which is why you think sickle contributed. I take that to mean contributed to death. What are the aspects of acute

chest syndrome, is it simply the presence of sickling in the lungs?

A. Yes. That is the pathology.

Q. That is the pathology?

A. Yes.

Q. I see. Let's return to your report, please, and if we can move on to the second page. We've looked at all of the individual tissue samples and what you found on examination, if we can now go to the commentary. You say:

"I do not need to repeat the chronology of the circumstances of death of this man who had sickle cell trait."

And then in brackets HbAS. What is HbAS?

A. Haemoglobin AS it means you have one -- half of the haemoglobin in the cells is normal A and half is sickled. HbAS is this sort of code for sickle trait status.

Q. I see. You continue:

"From my review of the gross and histology features of the deceased's tissues, and based on my previous experience of such deaths in HbAS, and knowing the literature as it has been presented by Dr Soilleux, I believe that HbAS trait did contribute to the death of BAYOH."

When you say that you reviewed the gross and histological features, what do you mean by the gross features?

A. The gross features actually really means there's no other cause of death evident. It wasn't a straightforward heart attract, stroke, pulmonary thromboembolism et cetera, that is what I meant by that. There is no other gross pathology. All the pathology here is subtle histopathological.

Q. When you refer to the histology features, are those the features we have been discussing under the section --

A. Yes, that is right.

Q. -- headed, "Histological review". You make reference there to drawing on your previous experience. To what extent did you require to rely on previous experience and how did that inform your analysis?

A. Totally. From looking at the cases as in that table, which no doubt you will come to anyway, of the eight cases, this being number five I think on that table, it comes down to quantitation, it ultimately comes down to the balance of quantitation of how much sickling is going on in the lungs consistent with classic cases of acute chest syndrome where there is no argument at all, versus alternative possible explanations for the death.

The fewer there are on the alternative explanations,

like obvious gross pathology, then the more emphasis you are probably going to put on the sickling there in the lungs. But on the other hand if it turns out that the other features in the case of which one was not so much aware at the time are actually rather more emphatic than you believed, then in a sense you downplay the sickling. Putting it fairly crudely but that is what it is about.

Q. You mention literature, you say:

"... knowing the literature as it has been presented by Dr Soilleux~..."

Did you refer to literature to inform your decision-making?

A. I looked through her literature list and said I have read that, I have read that, I have seen that, I have got the book and so on. I didn't go into any more details.

Q. Was it literature that was familiar to you?

A. Oh yes.

Q. Returning to your conclusions or your commentary section you say:

"There is no doubt that HbAS persons have died in custody or under stress (heat, dehydration, exercise) where the main pathogenesis a sickle crisis affecting the lungs (particularly), the kidneys, skeletal muscle, and the heart, and this has led to death. There is

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

I would like it ask you some questions about that paragraph. You refer to HbAS persons dying in custody under stress and we have discussed already the military paper and the papers relating to sportspeople. When you refer to stress there you put in parenthesis heat, dehydration, exercise; are these the key stressors, or are there any other stressors that could potentially be relevant?

A. Those are the main ones. We went through it before, didn't we? Hydration status; concentration of sickle in the red cells, which of course is in-built; oxygenation; and acidity, which relates to muscle activity. Drugs and alcohol of course relate in a sense to the other side of the scale, as are they sufficiently bad, abnormal to have caused death in their own right.

Q. At the bottom of that paragraph, the one that is now at the top of the screen, you refer to the critical aspects being the level of stress and accompanying elements being dehydration -- sorry, accompanying elements such

as dehydration, drugs, alcohol intake, muscle activity and body temperature. For your information, Mr Bayoh's blood and urine were subjected to a screen and were negative for alcohol.

A. Yes.

Q. Does that change anything? Is that particularly relevant here?

A. Well, alcohol is not relevant.

Q. So alcohol is not relevant?

A. Yes.

Q. All right, but would that have an impact on any of the conclusions you reached back in 2018?

A. No, not really, no.

Q. So far as temperature is concerned, we heard evidence that in hospital Mr Bayoh's temperature was recorded at 35.8 degrees?

A. 35?

Q. 35.8?

A. That is low.

Q. But that it was a cold wet morning and he was outdoors only wearing a T-shirt. Do either of those piece of information change anything?

A. How was the body -- it gets very -- how was the body temperature actually measured, because you can get any number you want depending on where you put the

thermometer or the device, I don't know. And I don't know if I did know either.

Q. But otherwise in that paragraph -- if we can scroll up so we can see the full paragraph again, please -- you make clear that some HbAS persons have died in custody or under stress as a result of the sickle crisis, that you have explained to us in detail in your evidence but you also observe, and again you've told us about this today, that the vast majority of people with sickle cell trait who undergo life's daily stresses do not suffer any such syndrome?

A. Yes.

Q. Continuing to the next paragraph of your report, please. You say:

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

the death process."

I would like it ask you some questions around that. You say --

A. I stick with that paragraph.

Q. That is helpful to know. You say that the critical aspects are the amount of sickling, can you comment on the significance of the amount of sickling in Mr Bayoh's case?

A. Well, we have already been through this, I put "massive" in the kidney, I can't remember quite what it did look like but it was obviously far more than any sickle trait person who died -- more in Mr Bayoh's kidney than in a comparable sickle trait kidney from someone else. It must have been more, otherwise I wouldn't have written that.

Q. So far as the number of critical organs are concerned, it appeared that with the exception of the bone marrow you found sickling --

A. It is everywhere, isn't it?

Q. -- everywhere?

A. Absolutely, yes, and that is certainly what impressed Dr Soilleux as well, that it was everywhere. But she won't have seen as much sickle pathology as I have, but that impressed her.

Q. What conclusions would you draw from both the amount

of sickling present and the number of organs affected?

A. As I said, it is a little sort of line, arrow, moving along the scale, and the more you see the more you think: ah, sickling might be relevant here. And when it comes to -- the crucial one is the lung, how much is going on in the lung, as I have said several times before. Because if something is going to stop the body function such that the person dies, it is going to be in the lung. That is the critical bit that is affected. Because, as I have said already, you can damage the kidney, you can damage the liver and so on but that is not going to kill you. Not acutely anyway.

Q. You have explained to us already that of the six lung samples there was sickling present in three --

A. I know, only in three, yes. Perhaps I should have hedged a bit more. But for some reason or another back in 2018 I didn't.

Q. Returning to that paragraph, you say:

"There is no rigid morphological case definition that separates harmless sickling from harmful sickling."

So some sickling can be harmless then?

A. Some of it can happen simply after death and it is nothing to do with anything, it is part of the decomposition process. That is what I meant by that actually.

Q. I see. So it's not that we are talking about sickling in lifetime that hasn't taken a person suffering from sickle cell trait to tipping point --

A. It could include that but the point is it is very difficult at autopsy work to know which one is which.

Q. You do on to say the quantity of sickling here tells you this is much more than just post mortem sickling?

A. Yes, that is what I thought at the time.

Q. Is that drawing on your experience?

A. Absolutely, because there is nothing else to draw on. Some of the papers produce pictures and so on but they are not much help, in the end it's what you have seen before and how you have interpreted it, and how in a way those deaths have been -- if they have been re-evaluated and tested again and what the final conclusions were. In a sense this particular case is another example of learning, that you readjust where your needle is on the dial according to what you have seen and what you have learned subsequently. That is how we all work.

Q. Can we move to your statement briefly please, because there are a couple of paragraphs there in which you deal with the difficulty in determining whether sickling is antemortem or post mortem. Could we look please at paragraphs 30 and 31:

"I am asked how I can tell that the sickling happen

prior to death and contributed to the death, as opposed to it was sickling that occurred around the time of death as part of the process of dying. When people who have sickle trait, who have died of something else, die, you can get after death sickling because the body cools down and it becomes slightly acid, and that can turn red cells sitting harmlessly in blood vessels into sickle cells, and we see that. There is a difference, actually. The sickle happening before as opposed to after death; this is partly based on the organ distribution of the sickling, and quantification of the process, which is subjective histopathological evaluation."

Again, you are drawing on your experience in making this assessment?

A. Everything there is true, yes.

Q. If we can move to paragraph 31:

"Almost certainly in Mr Bayoh, like in all the other cases, there's an element of both, because you don't autopsy people the moment they die or very rarely. So there is always going to be a little of additional post mortem sickle trait, 'it happens anyway sickling'."

You have called it:

"I was making a comparison between the average sickle trait person who's died and this kind of case,

like Mr Bayoh's, where there is more sickling, and it struck me it probably could have contributed to death. In other words, it happened in the time leading up to the point when his heart stopped. However, to emphasise~... again, in terms of importance and significance of the sickle cell trait, it's a lot less than the recreational drugs and the struggling and the restraint. Those are the important things here."

Can we return to your report, please, and you say the quantity of sickling in Mr Bayoh's case -- sorry, this is on page 3. You say the quantity of sickling here tells that you this is much more than just post mortem sickling. Can you help us to understand your thought processes when you reached the conclusion that what you saw histologically was more than post mortem sickling?

A. That is -- yes, that is right. I think I still think that is correct in this case. But it is balanced against other factors which have become more evident since I wrote that.

Q. What led you to the conclusion that it was more than just post mortem sickling?

A. Oh, because the lung vessels, where they were affected, were very dilate. They were very distended. That was the crucial thing.

Q. And you said earlier in your evidence that is something that happens in life and not after death?
A. I thought that happened in life because it is not what one sees in people with sickle trait who have died of other reasons.
Q. I see. You say it happens perimortem as part of the death process, that is at the very end of that paragraph. What do you mean by perimortem here?
A. In the period just before, during and after death.
Q. Okay, so that is different from antemortem?
A. It will include a bit of antemortem, but it is leading to death, leading towards death.
Q. I see, so perimortem is around the time of death?
A. Yes, around, that is right. Like peripartum is around the time of delivery, not necessarily at the moment when the baby comes out.
Q. So can I just ask you to clarify please, in your opinion when did the sickling that you saw in Mr Bayoh's case take place. If perimortem encompasses before, during and after death, to what extent was the sickling that you saw in your opinion antemortem?
A. I thought it was antemortem because the lung vessels were particularly dilated and that doesn't happen, as I have said several times, in someone who has sickle trait and just dies for some other reason. You don't

quite see -- you don't see -- the lungs look different.

Q. Okay. So although your report says that the sickling happened perimortem as part of the death processes, we should understand that there was evidence to you as a histopathologist that at least some of this sickling was antemortem?

A. Yes. That is what I inferred from the tissues, yes.

Q. Let's move on to the next paragraph of your report, please:

"In the BAYOH case, I am impressed by the quantity of sickling in the organs such as the heart, kidneys, liver, thyroid and adrenal - much more than I expect to see in the organs of those with HbAS who died of unrelated causes. The lung tissues show more variable amounts of sickling, but where it is present, it is again more pronounced (ie the blood vessels are more distended)~..."

You have told us about that already:

"... than one would normally see in HbAS persons dying of other causes."

A. I stand by every phrase there.

Q. Moving through your report, please, to the next paragraph:

"Dr Soilleux present a good diagram of the potential clinical pathology of death in this case, with which

I do not significantly disagree apart from suggesting a more prominent component from the lung pathology. She concludes with the cause of death as:

"1a. Sudden cardiac death.

"1b. Sickle cell trait, recreational drug use, struggle against restraint.

"I would suggest an alternative:

"1a. Sudden cardio-pulmonary failure.

"1b. Sickle cell trait, recreational drug use, struggle against restraint.

"The last feature - struggle against restraint - can include positional asphyxia, but as a non-forensic pathologist, I do not wish to be drawn into a more detailed discussion in that area."

Scrolling down:

"Importantly I don't think we can quantify the contribution of the three factors presented in 1b and state, with rigour, that one is more or less important than the others. It is multifactorial."

I appreciate this was the opinion you expressed in 2018, and I understand that your opinion has perhaps moved away to an extent but if we can stay with the text of your report for now, can you help us to understand the difference between 1a and 1b in the cause of death?

A. Sorry, what is the question again?

Q. The difference between 1a and 1b --
A. 1b --
Q. -- on a certificate death certificate in general, not with reference to this case?
A. The bottom line of any cause of death sequence, which here happens to be 1b -- it could be 1c, it could be 1a -- is the pathology, the clinical pathology, what goes into the national statistics. 1a, if there is one, is the mode by which that pathology caused the end of the life of that person, and often one leaves it out.

In fact statisticians don't like that because it gets in the way, they just want the bottom line. I just refined the bottom line because it made it seem as though the sickle cell trait had stopped the heart; it hadn't. The sickle cell trait had blocked off the lungs or contributed to blocking off the lungs, stopping the circulation and as an end result stopped the heart. It is just being picky and nerdy about the precise pathology going on.

Q. We have heard that death certificates also have a part 2?
A. Well, I don't think the original version had a part 2. I mean, no one was put in --
Q. Sorry, death certificates in general. Let's move away from Mr Bayoh's case.

A. Oh, right, yes. They have the option to fill in part 2, one doesn't necessarily use it.
Q. What would one use part 2 for?
A. What you put in part 2 is other conditions, acute or chronic, that contributed to the death not so much in actually causing the death but in influencing when it happened, and how it happened. I can tell you that every certifier of death certificates, every pathologist, every coroner -- and remember, coroners write death certificates on advice -- has a different view of what goes into part 1 and part 2, there is no consistency just as there is actually -- and it's a separate debate -- no consistency in what is a natural or an unnatural cause of death. This is simply an inevitable conflict between medicine and law, where there is no general agreement on details as to what should go where.
Q. So, back in 2018 the only difference between yourself and Dr Soilleux was that you would have preferred sudden cardiopulmonary failure over sudden cardiac death. For members of the public who are perhaps listening to your evidence, can you explain please the difference between sudden cardiac death and sudden cardiopulmonary failure?
A. Cardiopulmonary failure here means the acute chest syndrome. It is shorthand for that.

Q. In the paragraph beneath the cause of death, you say:

"The last feature - struggle against restraint - can include positional asphyxia, but as a non-forensic pathologist, I do not wish to be drawn into a more detailed discussion in that area."

What did you mean by that?

A. What I was really meaning is that -- I wasn't told very much about what actually happened in the struggle against restraint, and I had the impression from the original autopsy report that that aspect was not particularly important. Positional asphyxia is -- actually, can I ask you, have you had forensic pathologists talking about positional asphyxia in this Inquiry up to this moment?

Q. Yes, we have heard evidence from the Dr who performed the autopsy and we will be hearing evidence from another forensic pathologist in the fullness of time.

A. Good. The practical point is as a non-forensic pathologist I don't have much street cred going in for positional asphyxia if there is third party involvement, I just put it as crudely as that. I have seen deaths from positional asphyxia where people have, because they have been drunk, flopped over a sofa and basically their head and neck is resting on the arm of a sofa and have asphyxiated because they were drunk. Were that to be in

relation to a possible criminal offence and third party involvement, I don't get involved because that is forensic stuff. And I am simply being cautious there, perhaps unnecessarily so, that I don't have -- because the death occurred in confrontation with the forces of law and order, I couldn't really make any comment because I didn't know enough and I don't have any street cred as a forensic pathologist.

Q. Thank you. I want to move on now to ask you questions about the extent to which you perhaps departed from the opinion that you expressed in 2018, and the reasons for that departure. A good place to start might be by going back to your statement, where you give an explanation at paragraph 25:

"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 light of the expectancy by a couple of minutes, given the stresses he was under at the time."

A. The only thing I would change is I don't quite know why I put -- I think I was slightly pressed by Ms Wade about time and I said, "Maybe a couple of minutes or something like that", but it is not something I want to be taken precisely. But the rest of it is true. Particularly I remember now back in those times that the coroner for Southwark was pressing me very hard to join him as co-author on two big papers on what is a natural and unnatural cause of death and the point is we couldn't agree, which is why I took my name off it and it got published by other means. It is actually a very complicated issue.

Q. I think in fairness when you say at the very end of paragraph 25, "a couple of minutes", you go on in paragraph 26 to say:

"Again, that is guesswork. I can't quantify that~..."

A. Yes.

Q. "Seriously no one can do that." Continuing to read through paragraph 26, please:

"On many occasions I have provided opinions that sickling played 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 [that is Sheku Bayoh's case] comes into that category."

A. Yes.

Q. Can you help us to understand then why it is that you feel now in 2023 that sickle cell trait perhaps played a less important or less significant role than you had thought in 2018?

A. Two reasons, or two related reasons. One, I was informally given summaries and some evidence that a lot more restraint than I had appreciated back in 2018 had occurred in this case. Also, and this will be a fairly obvious bit of information to everyone in this courtroom, there has been a huge amount of public information about police restraint and death, particularly, but not only, in black people. The George Floyd case is the calling one, where this poor man in Minnesota certainly did not have sickle trait, as far as I know he didn't anyway, but he was photographed doing what -- I can't say this really. I was going to say one would not like the police to be doing things like that. The point is that everyone saw that

restraint kills; restraint pure and simple, prolonged, kills. And it was with that sort of thing in mind when I heard that, in this particular case, Mr Sheku Bayoh, that there was more restraint and struggle going on than I had appreciated back in 2018, I thought that actually I am going to change my mind and downgrade the role that sickle played in his death. It's as simple as that.

Q. Can we look at paragraph 35 of your statement, please, where you explain a little bit more about your change of opinion. You say:

"I think I would modify that statement now ..."

Sorry, for context we should look at what came above that. It's simply a quote from the final paragraph of the commentary section of your report with the cause of death, your preferred cause of death, back in 2018. At paragraph 35 you say:

"I think I would modify that statement now and make it a little less generous. Since I produced my report in 2018, I have been given much more information regarding the circumstances of Mr Bayoh's death information (ie more about the degree of restraint ...) and more forensic opinion."

At paragraph 36 you go on to explain the difference that this additional information has made to your thinking. You say:

"I would now actually put recreational drug abuse struggle against restraint. I'm not going to sort out which of those two is more important but definitely put sickle trait in a different line as it is a less important phenomenon."

A. A different line being part 2, not part 1.

Q. I was about to ask. And I think you explained elsewhere in your statement that it would go under part 2 of the death certificate part 1. For your information, when Dr Shearer gave her evidence, she was taken to a relevant part within your statement where you said that you would include sickle cell within part 2 of the death certificate today and asked to comment, and she said, yes, that is very sensible, that is -- "I would agree with that completely, yes", was what she said. So it would appear that you are on the same page in terms of the role played by sickle cell trait and the status it should be afforded on the death certificate.

Can we look at paragraph 37:

"I am asked whether but for sickle cell trait Sheku Bayoh would have died. Yes. Now, I say that as a non-forensic pathologist. Just from what I've learned over 45 years of looking at dead people and quite a lot of time looking at sickle patients particularly, it seems to be a very minor component here."

1 And at 38 --
2 A. That is a comparative statement; compared to ... other
3 events.
4 Q. Compared to other events?
5 A. Yes.
6 Q. I see. The other events that you know about now?
7 Meaning the restraint and the events surrounding that?
8 Sorry, professor, by "comparative" what other events are
9 you comparing --
10 A. More information about the degree of restraint applied
11 and, again, in the context of much public knowledge
12 recently from America about restraint-related deaths in
13 custody.
14 Q. And at paragraph 38:
15 "I am asked whether in my view sickle cell trait
16 made a material contribution to Sheku Bayoh's death.
17 Yes, but a small amount."
18 A. Yes.
19 Q. I wonder if I can just ask you about the language used
20 here. In paragraph 37 you say "a very minor component".
21 In paragraph 38 you say "a material contribution ... but
22 a small amount".
23 A. Sure. But, as you know very well, these are transcripts
24 of conversations on a video conference. So this is
25 speech, not me writing prime text.

Q. Absolutely. All I wanted to clarify with you is --
A. But they mean the same thing.
Q. They mean the same thing. Certainly from a legal perspective, any contribution to a harm that is more than de minimis will be a material contribution. So should we understand that it is your view that sickle cell trait made a material contribution within the definition that I have just read to you, but, all the same, you consider it was a minor component or a small amount?
A. Yes, and I would say -- I would stick with this; that I think probably the sickle problem in his lungs accelerated the moment when he died but it would have happened anyway.
Q. Can we look at paragraph 33, please:

"I am asked whether the sickling of cells I saw in the histology fits with respiratory arrest and then a cardiac arrest. Oh, yes. To the extent that one can pathologically distinguish, I think that is right."

A. This is all about how sickling causes a major organ to fail. Okay? And what I am really saying here is, if it contributed at all, which I believe it did, then it did it through respiratory arrest or respiratory malfunction, not primarily cardiac malfunction, which would have followed on later from respiratory

malfunction. As I say, and I have said several times, even in sickle cell disease the heart is not acutely affected, not like that.

Q. We have heard evidence, professor, that the police officers following Mr Bayoh's collapse couldn't find a pulse and started CPR but, when the ambulance arrived and applied the ECG pads, they got a trace?

A. That is normal in resus, yes.

Q. And when Mr Bayoh was taken to the hospital, it was a very short journey to the hospital, he was found to be in respiratory arrest but very quickly went into cardiac arrest. Would that sequence fit with what you saw in the pathology; the respiratory arrest followed by a cardiac arrest?

A. I think so. The whole business about acute events going -- around time of death and in resuscitation is actually very confusing. You made it sound very simple. It actually is much more difficult, I think, necessarily to tell the difference between these things going on, respiratory versus cardiac. And even in A&E medicine it is often rather unclear which one is primary. But they are certainly linked.

Q. I would like to move away from your report, and indeed from the statement, and return to something that you said in your report, which was that:

"There is no doubt that the vast majority of persons with HbAS who undergo life's daily stresses do not suffer sickle cell crisis."

What I would like you to do is to help the Chair to understand the impact that the trait may have on a person who is subjected to a level of stress that goes beyond the daily norm but is perhaps not at the level of military training in a desert or extreme exertion under marathon conditions or similar. I would like to tell you about some of the evidence we have had about the events of the morning of 3 May of 2015, which I will do in short chapters and then simply invite your comment.

So we have heard evidence that Mr Bayoh was a black man in his early 30s, he was 5 feet, 10 inches tall, he was 12 stone 10 in weight, his BMI was 25.6 --

A. Sorry, 25.6?

Q. 25.6.

A. Normal.

Q. He was in good health. He had sickle cell trait, although there was no mention of it in his medical records. He did not have the disease. Absent any source of stress, would you have expected Mr Bayoh to develop sickling within his organs?

A. No. Not at all.

Q. We have heard evidence that in the early hours of the morning -- overnight or in the early hours of the morning he had taken MDMA and alpha-PVP. He also used the bodybuilding steroid nandrolone and nandrolone, a level consistent with recent administration, was found in his urine. At the time of his contact with the police, which was shortly after 7 o'clock in the morning, we have heard evidence from a psychiatrist that he was in a state of drug-induced intoxication. So can I ask in your opinion whether you would expect the intoxication alone as a result of these drugs to have caused sickling?

A. No. No. And the drugs he was taking, these are difficult ones to -- you have had all this, I am sure, from people far more expert than me. There are easy drug intakes where you can measure things in terms of levels and concentrations in body fluids and draw a reasonably robust conclusion, but with the sort of medications we are talking about here there are so such sharp cut-offs. It is subjective in terms of interpretation.

Q. Yes.

A. Yes.

Q. We heard evidence that, at a point in time after he had taken these drugs but before he met the police on

Hayfield Road, perhaps between half past 6 and quarter to 7 in the morning, he had a fight with his best friend in the course of which he punched his friend, chased him with a washing line pole, pushed him to the ground, got on top of him, and I quote "threw a good few punches". A witness to this attack said it lasted about two minutes. Would you expect a fight such as I have described to have caused sickling?

A. Not a fight lasting only two minutes in someone with sickle trait. No.

Q. We have heard evidence that after the fight but, again, before he met the police on Hayfield Road Mr Bayoh was chasing cars, walking briskly, walking with purpose. He had a knife, and a number of calls were made to the emergency services. Would you expect that level of activity to cause sickling?

A. No, no.

Q. We have heard evidence that the police arrived at Hayfield Road at about twenty past 7 in the morning, at which time Mr Bayoh was walking along the road. He no longer had a knife. He was approached by the police. They shouted commands and they sprayed him with CS and PAVA spray, which apparently had no effect on him. Would you expect that to cause sickling?

A. No.

Q. We have heard evidence that Mr Bayoh then chased a female police officer across a roadway and struck her to the back of the head, causing her to fall. He may then have stamped on her back with full force as she lay on the ground. There is a dispute as to whether or not that happened. But would you have expected those actions, a short run, striking an officer and possibly stamping on their back, to have caused sickling?

A. I don't think so. But you are getting beyond the level of what I actually know, as I am sure you appreciate.

Q. We then heard evidence about the restraint. This involved Mr Bayoh being struck to the head with a baton two or three times, struck to the body or the arms another two or three times. He was then taken to the ground in what has been described as both a shoulder charge and a bear hug. He was brought to the ground quickly, where he was restrained by several officers. He struggled against the restraint, did a press up to get the officers off him. The weight of one officer -- we have heard some evidence that one officer was lying across his upper back. The officer in question was 25 stone in weight. He was, in the course of the restraint, handcuffed to the front and leg restraints were applied to stop him from kicking out. He then became unconscious and subsequently stopped breathing.

Would you expect a restraint such as the one that I have described to you to cause sickling?

A. It could --

Q. Are you able --

A. -- because it was impeding his respiration. It gets down to the oxygen business.

Q. So of the four considerations that you told us were relevant, hydration, concentration, oxygen and acidity --

A. Temperature; his temperature must have been raised at that point from all that physical exercise.

Q. And you said the restraint -- and we have heard much about the struggle against the restraint being perhaps as important as the restraint itself -- may have had an impact on respiration and that could cause sickling?

A. And also the muscular activity would presumably produce some lactic acidosis. Tell me, was he ever categorised as having excited delirium?

Q. That is not a question that I would want to be drawn into. I think I would prefer to --

A. No, I don't either. Because its definition is very difficult.

Q. There has been evidence led in the Inquiry around excited delirium, but I think it is best that we stick with --

A. I am with you on that one.
Q. -- your area of expertise. I am interested in how the restraint impeding respiration might have or "could" have I think is the word you used, caused sickling. Is it to do with the oxygen and the hypoxia that you referenced as being one of the four things --
A. Yes, it is multifactorial. It would be hypoxia plus acidosis from lactic acid coming out of the muscle from inappropriate metabolism. Yes.
Q. So hypoxia and acidosis, and these could have caused sickling?
A. Yes, but the other thing -- I don't know whether you made reference to it, and I can't remember any details, is whether his airway was obstructed.
Q. If there had been any obstruction to the airway, what would the impact of that have been from the perspective of sickling?
A. It could have precipitated but, more importantly, it kills you faster.
Q. From the description of the restraint that I provided to you, are you able to say at all at what point during that sequence of events sickling would likely have commenced?
A. No, you can't. I am sure in this case it developed a bit during that. (Pause).

MS THOMSON: Thank you, professor.

Sir, I am about to move on to an entirely different chapter. It is a little early, but I wonder if this might be a convenient point to break.

LORD BRACADALE: We will sit at 1.55 pm.

(12.57 pm)

(The short adjournment)

(1.55 pm)

LORD BRACADALE: Ms Thomson.

MS THOMSON: Thank you. Professor, earlier today you were looking through a pile of personal papers relating to the case that you are brought with you and I said if you wanted to take the opportunity at lunchtime to ascertain whether there was anything in there of significance that wasn't in the folder ...

A. All I can say is that in this pile of papers, which is all I had in my folder back home, there is no print-out of any of the other forensic pathologists' reports, so what I heard from them was snippets given me by other people, or hearsay, but I certainly didn't have them to print out. But I do remember having a conversation with Nat Cary some time ago.

Q. In that case then it would seem fair to assume that there were four reports available to you at the time that you prepared your report: there was the post mortem

report, the supplementary post mortem report and two reports prepared by Dr Soilleux.

The post mortem report contained a summary of the circumstances of the restraint and Dr Soilleux's first report contained a detailed summary of the circumstances around the restraint that was based on her reading of the witness statements, and those reports and the summaries contained within those reports were available to you at the time that you prepared your report, is that correct?

A. Uh-huh.

Q. Yes. Can you help me to understand then what information about the circumstances of the restraint that you didn't have at the time but you have now that has caused you to change your view as to the significance of sickle cell trait in the context of this death?

A. To be absolutely honest, I can't remember.

Q. All right. I want to ask you one or two questions about the literature around sickle cell. Can we perhaps bring up your statement again, paragraph 63.

A. Ah, yes.

Q. "I would like to recommend a book to the Inquiry which may be helpful 'Sickle cell and deaths in custody' by Simon Dyson and Gwyneth Boswell. It is basically

looking at North America and Britain and the extent to which people who are black, who have sickle or sickle trait and died in custody of one sort or another, to what extent the system, which includes the pathologists and the investigators and the police, blame sickle for the deaths when actually the deaths were caused by restraint or other things related to being in custody."

Why did you recommend this book to the Inquiry team?

A. Because this is a death in custody.

Q. And to what extent did you -- or why did you consider that this book might be helpful to the Inquiry?

A. For background information that you, your teams and the other experts may not be aware of.

Q. Is it a book that you have read?

A. Yes, I read it -- I bought it in 2009 when it came out.

Q. The authors are Simon Dyson, who I believe is a sociologist, and Gwyneth Boswell, who is a criminologist. So neither of them are medical people.

A. Correct.

Q. But from your perspective as a pathologist, as a histopathologist, did you find the book to be relevant and accurate?

A. Absolutely. Relevant in that they are dealing with what we are dealing with here, and they cite a lot of cases, at least four of which are mine.

Q. So they cite at least four cases in which you have been personally involved?
A. Yes.
Q. Are they cases involving the deaths of individuals in custody or similar circumstances?
A. Custody and one -- three were in custody and one was on army exercises, yes. British Army exercises.
Q. Is there anything in the book that from your perspective as a medical man seems to be incorrect or inaccurate?
A. They get the details wrong. I know that because two of them, I did the autopsies myself.
Q. To what extent do the authors getting the details wrong impact on their overall conclusions or what you see is the relevance of their work?
A. Not that much. I think the overall thrust of that book, and also the article which came out a little earlier in 2006, which you kindly sent me last night, which is basically a summary without all the case details of what is in the book, I think is broadly correct, in that, when people who have sickle trait have died in custody, there have been many cases documented, and we have one in this country, which I am sure we will come to, where the cause of death had been put down as sickle cell trait and, in their view and in mine, that is not justified.

Q. Are these the sorts of issues that are explored in the book?
A. It is sort of, but they are not medical, as you said, so they don't really know the precise details of what goes on. But they explore it primarily from a criminological and a sociological and historical actually -- historical perspective. They are not terribly interested in clinical pathology.
Q. And to what extent, if at all, have you been influenced by the book?
A. I think it reinforced what I already suspected. Bearing in mind the very first sickle cell trait death in custody that I was involved in looking at was back in 1995, so I had had that experience under my belt, so to speak, already and made me wary of what the police and prison guards do, or don't do.
Q. You have mentioned the article that the Inquiry team sent to you. I think we have a copy of that. It is publicly available, albeit it has not as yet been given an Inquiry reference number. But it is referred to by Dr Soilleux in her report.
A. Yes.
Q. So I wonder -- just to orientate ourselves, let's bring up the article. Thank you. So it's by the same authors, Simon Dyson and Gwyneth Boswell. We see

the article is entitled "Sickle cell anaemia and deaths in custody in the UK and the USA" and it was published in the Howard Journal of Crime and Justice. So it has not been published in a medical journal, it has been published in a journal of crime and justice in February of 2006. And you said a moment ago that this was a precursor to the book?

A. Yes. Well, it must be --

Q. Or preceded the book.

A. -- because large chunks of the text are the same thing. If you compare the book with the article, you can see big paragraphs that are just transferred. But the book is a great expansion. It is ten times longer than the article.

Q. So it appears that the article was written first and then this has lead a few years later --

A. So I would presume, yes.

Q. -- to the publication of the book. The point has been made that this article hasn't been published on a database called PubMed. I think that was an observation by Dr Soilleux. What sort of articles would you expect to find on PubMed?

A. That is interesting. PubMed is the biggest collection of all medical-related articles across all the disciplines of medicine, not just pathology obviously

but everything else, and it has the titles -- and these are articles rather than books. PubMed doesn't do books so much, it does journal articles. Articles are coming out all the time and they are constantly scanned and they are put in. Not all journals get into PubMed even if they are medical. Some of the exceptions include journals that are essentially unreferee'd, that is to say not peer-reviewed, and promoted by billionaires wanting to make their name. But it does have the core of the big journals that are, in a sense, publicly-owned. They come from national public funds. The big medical journals in America and the British ones, they will all be in there, but there are certain fringe ones that are not there.

Q. Is this a database of medical journals?

A. It is a database of medical journals and, although the title looks medical, the journal they put it in, the Howard Journal of Crime and Justice, obviously doesn't. Universities that run big databases would have a different set. If you went into a legal -- a law department or criminology department in the university, they would know about this. It is not a journal I have ever looked at before. But, in fact, if you are linked into journals and went through JSTOR, you get this. Because the University of London will have it.

Q. But it is not a journal that you would expect necessarily to find within PubMed because it is not a medical journal?

A. No. And, to be honest, I don't know how Liz Soilleux came across it either. I never asked her. I could have done.

Q. If we could perhaps look very briefly at the abstract. If we just scroll down a little bit to the first page of the article. This is all we will look at. If we pause there we see the authors are Simon Dyson, Director, Unit for the Social Study of Thalassaemia and Sickle cell, School of Allied Health Sciences, at De Montfort University and Professor Gwyneth Boswell, Principal Research Fellow based in London. So we see that neither profess to be medically qualified. We have a sociologist and I think a criminologist and, if we scroll down a little, please. A little further, just to the abstract. This gives an introduction to the article. It says:

"An unexplained death in custody represents an important focal point for public scrutiny of the criminal justice system, especially when excess deaths occur in those of minority ethnic descent. Sickle cell anaemia is a serious inherited blood disorder disproportionately affecting minority ethnic groups.

Sickle cell trait is the genetic carrier state and is not an illness. The evidence suggests that the treatment of sickle cell in the criminal justice system is twofold. Justice authorities have misused sickle cell trait to explain away ten sudden deaths, often associated with forced restraint of African-Caribbean people in custody. Meanwhile seven death have been attributable to lack of provision of healthcare for those prisoners suffering from the illness sickle cell anaemia."

So that abstract simply gives a flavour of what the article is about. Have you read the article, professor? Have you had the opportunity to cast your eye over it?

A. I read it quickly last night on my mobile phone. My computer is not working at the moment. But I have read the book.

Q. And from your perspective again as a consultant histopathologist did you have any concerns about the medical content insofar as there was medical content in the --

A. Yes, I am concerned because I think they get some of the details wrong, but the overall thrust of what they say is true.

MS THOMSON: Bear with me a second, please.

Thank you, professor. There are no further

questions from me. Thank you.

LORD BRACADALE: Any Rule 9 applications? Professor Lucas, would you withdraw to the witness room, please, while I hear submissions.

(In the absence of the witness)

Rule 9 Application by Ms MITCHELL

LORD BRACADALE: Can I just check, Ms Mitchell, did you put any written application in?

MS MITCHELL: A Rule 9? I don't know, but this arises, my Lord, from something the witness said in evidence, which isn't contained within his statement. So I can get that answer to you shortly.

LORD BRACADALE: I don't think you did.

MS MITCHELL: Yes. The situation is this witness explained that he had had several conversations on the phone with Mr Brown in relation to the sentence which he put into his report which he said he wouldn't do now, that about the officers involved should not be prosecuted assuming that they were approached and restrained. What I would like to do is with this witness explore the circumstances of the several conversations with Mr Brown, whether or not he has any records of those conversations, when they took place and how it might be that they would have conversations about such matters.

LORD BRACADALE: Is that the matter?

MS MITCHELL: Yes.

LORD BRACADALE: Thank you. If you could go back to your seat, I will hear from Ms McCall.

Rule 9 Application by MS MCCALL

MS MCCALL: I am sorry, I didn't have a Rule 9 application in but the matter arises out of the witness's oral testimony, and it relates to the basis for his ultimate conclusion, his conclusion having changed. The context is that junior counsel to the Inquiry spent some time exploring what material the witness had and why his opinion changed, and, in my submission, it is important the Inquiry understands, if it can be understood, what the possible foundation is for his ultimate view.

So at page 95 of the [draft] transcript, line 18, he was asked why his opinion has changed, and he said there were two reasons. The second of those, which I am not concerned with, was about public information about police restraint and deaths, but the first reason he gave was he was informally given summaries and some evidence that a lot more restraint had occurred. Then at page 108 he was asked what information he had now that he didn't have before that resulted in his change of view, and he replied "I can't remember".

The first question I want to ask is: what was the source of the information and what form did the

summaries and evidence take, the sources of these summaries and evidence not being recorded in any of the statements or reports he has prepared.

The second matter that arises on the same theme is that the witness said at page 107, line 16, [draft] that he had heard snippets and hearsay and had spoken to Nat Cary. So the second thing I wanted to ask was, if it is possible that the summaries and evidence was conveyed in a conversation with another or others, who was the conversation with, when was it and in what context did that conversation occur.

Those are the matters.

LORD BRACADALE: Thank you. I will rise to consider these various references.

(2.17 pm)

(A short break)

(2.26 pm)

LORD BRACADALE: I shall allow Ms Mitchell and Ms McCall to ask the questions which they indicated they wished to.

Can we have the witness back, and Ms Mitchell.

(In the presence of the witness)

Questions from MS MITCHELL

LORD BRACADALE: Professor Lucas, Ms Mitchell KC, who acts for the Sheku Bayoh families, has some questions for you.

Ms Mitchell.

MS MITCHELL: Thank you. I would like to revisit something that you said in your communications with Les Brown, and that was said earlier today at page 59, lines 6 to 10 [draft]. I will just read those out. In your note you put:

"I will not put this in my report but what I can see of the scenario, the police officers involved should not be prosecuted assuming they approached and restrained Mr Bayoh in the normal approved fashion appropriate for the perceived risk."

And we have heard your evidence about that. Now, if we look at that and just highlighting the word "assuming", the assumption in what you said there related to the way they were approached and restrained, and in relation to the fact that they had done everything in the approved manner commensurate with the perceived risk. So there was an assumption being made, and the reader of that particular document would be clear that you would be making an assumption in that regard?

A. Yes.

Q. In your evidence you were asked about where that might have come from, that piece that was written in the document. Because, I think as you accepted yourself, it

is not something that would normally appear in a report that you would do. Your response was this, and this is at 61/22 [draft]:

"I had several conversations on the phone, I think with Mr Brown, and I have a vague feeling that this aspect may have come up, which might have been one of the reasons I put something in. But to say it is ..."

Then you go on to talk about why you put it in. What I would like to do is ask you about your communications with Mr Brown.

A. I really don't think I can remember any more than you have just reiterated, to be honest.

Q. Okay. If I can just try and test the edges of that and see. You clearly said in your evidence you had several conversations with Mr Brown. Do you recall for example what way you would have chatted with him? Would it have been via telephone?

A. Telephone I suppose, yes.

Q. Yes. And your evidence to the Inquiry was several conversations.

A. I think so. This is a long time ago.

Q. Indeed it is, and I am sure the Inquiry will take that into consideration. But we are keen to try and get as much information with regards to the communication with experts as possible. In having these telephone calls,

would you keep any records of them?
A. No.
Q. Would it be your habit to keep records and you simply didn't in this case, or are telephone calls something that you don't habitually record?
A. I can't remember -- it depends where I would have been in the house -- I think he called me, rather than I called him. And I'm afraid I can't remember.
Q. Is it your habit to record conversations that you have from people who are instructing you on reports?
A. Not necessarily, no.
Q. These conversations, from the way that you have answered the question, were presumably all before you had written your report?
A. I suppose so, yes.
Q. Was there any --
A. I should think so. You can probably tell from the date on the emails.
Q. Well, yes. The date that we are looking at and focusing in on here is the date by which you had written the report which has the comment in it that I referred you to. You say that you might have a vague feeling that this aspect may have come up. Can you help us with why you think that?
A. Otherwise I can't think why I wrote what I did. It is

not something I would spontaneously have done. There must have been some prompt somewhere in and around this that made me think -- in an email, not in the report, of course, as you say, that I make some reference to this, what might happen next. Maybe I asked what might happen next to the police, something like that.

Q. And you believe that that might have been a prompt for you?

A. Yes, and what I said, as you can see, is that if they behaved properly and restrained someone in the appropriate manner which is laid down in various training manuals and so on, then it is a very unfortunate consequence, but if they overstepped that is different.

Q. Can I just be clear, was the prompt a prompt by you or by the person you were speaking to, Mr Brown?

A. I am sorry, I cannot remember.

Q. Can you think of any reason why the Crown might want to speak to an expert such as yourself in your area about this given it is not your area of expertise?

A. They wanted to know whether sickle cell trait could have -- could -- have played a role in the death of Mr Sheku Bayoh and my conclusion was yes. Now, exactly how much, yes, we discussed all morning, so we don't need to reiterate that. That is the only reason I was

brought in, because -- and one of the other people must have said, "Ask Lucas, he knows about sickle", or something like that.

Q. And that opinion was given to the Crown in circumstances where the Crown knew that you were having to make assumptions about how Mr Bayoh was approached and restrained, and the interaction with him in the approved fashion appropriate for the perceived risk?

A. I didn't put that in report. So I didn't frame my report with that particular thing in mind. But, yes, that is in a sense correct.

Q. You sent that information to the Crown, so the Crown had it?

A. Yes, okay.

Q. Do you accept that?

A. Yes.

MS MITCHELL: No further questions.

Questions from MS MCCALL

LORD BRACADALE: Professor Lucas, Ms McCall KC represents a number of the police officers who attended.

Ms McCall.

MS MCCALL: Professor Lucas, this morning some time was spent exploring the basis for your opinion, and the information that you had, and you were asked about the change in your opinion and you were asked about why that

happened. What you said this morning -- this is at [draft] page 95 -- was:

"Answer: There were two reasons: I was informally given summaries and some evidence that a lot more restraint than I had appreciated back in 2018 had occurred in this case."

So that was the first reason. The second reason was about public information about police restraint and deaths.

A. Yes.

Q. I am interested in the first of those. So the summaries and some evidence that you were given informally. The question I have is what was the source of that information and what form did the summaries and evidence you were given take?

A. I can recall being shown conclusions, or extracts from some of the other forensic pathologists who were involved in consultation, but I don't think I ever had their full reports, and I had a conversation with Dr Nat Cary as well.

Q. Can you remember who it was that showed you the conclusions of other people's reports?

A. No. Not at the moment I can't.

Q. You also mentioned this morning, and you have just alluded to it there, that you had heard snippets and

hearsay and that you had spoken to Nat Cary.

A. Uh-huh.

Q. Can I ask if it is possible that the information that you got, sight of the conclusions and other hearsay or snippets, was in conversations? You have mentioned Nat Cary, were there conversations with anyone else?

A. I don't think so.

Q. When was your conversation with Nat Cary? Can you give us an idea?

A. Some time since 2018, which is quite a long period.

Q. In what context did that conversation come about?

A. Oh, we always talk about mutual cases, and I think he said something to the version -- he said, "I've seen what you said about this and I think you are wrong". So I said, "Yes, why?" 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 that made me think, ah ...

Q. In terms of when that conversation occurred, was that, if you can recollect, before or after you were contacted by the Inquiry?

A. The Inquiry not being Mr Les Brown's business but the business we are here --

Q. The team you have been dealing with here.
A. That would be ... When was I contacted by the Inquiry? Can you tell me? Because you made the contact.
(Pause).
LORD BRACADALE: I think we can't expect Ms Reilly to give evidence, Professor.
A. But she might have a date, that's all I'm looking for.
LORD BRACADALE: If you can concentrate on the question Ms McCall was asking you, whether it was before or after that contact came from the Inquiry.
A. It might well have been before actually.
MS MCCALL: When you were contacted by the Inquiry and giving your statement to the Inquiry, which was back in December of last year --
A. Yes, that is following the conversation in October with my learned friend here.
Q. Right. Did you at any point advise the Inquiry of the conversations you had had with Nat Cary?
A. I didn't -- I don't think I mentioned him by name but if I did it would have been in the transcripts, because that statement is not a written statement, it's a transcript of a phone conversation, so if it is not there, his name wasn't mentioned.
Q. I understand that.
A. Or it was decided not to include it when it was

transcribed. I didn't do the transcription.

Q. All right. Just for clarity I presume -- I may be wrong but I presume the Inquiry team sent you a copy of the proposed statement and asked you to -- gave you the opportunity to check it over?

A. Yes. Oh, yes. Which I did.

Q. So as far as the source of this information was concerned, additional information, we have identified Nat Cary as a source. Is there any other individual that you can identify as a source?

A. Not that comes to mind. I never -- I don't know, even socially, the pathologists involved in the original autopsy back in 2015. I didn't even recognise their names. And I have never met them.

MS MCCALL: Thank you Professor Lucas. Thank you, sir.

LORD BRACADALE: Professor Lucas, thank you very much for coming to give evidence to the Inquiry. I am grateful to you for the time you have spent on it. I am about to rise now, and you will be free to go then.

(2.39 pm)

(The Inquiry adjourned until 10.00 am on Wednesday, 24 May 2023)

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