The Sheku Bayoh Public Inquiry

Witness Statement

Professor Michael Eddleston

Taken by [redacted]

Via MS Teams

on Friday 6th January 2023 (amended on 28th March 2023)

Witness details

1. My name is Michael [redacted] Eddleston. My contact details are known to the Inquiry.

2. I am a Professor of Clinical Toxicology and a Consultant Clinical Toxicologist and Pharmacologist. My qualifications are MA PhD BM BChir ScD FRCPEdin FBPhS FEAPCCT.

Education

3. I completed my Bachelor's degree at the University of Cambridge in 1990. Four years' later I was awarded my Masters of Arts, for which I have the professional letters 'MA'. The Master's degree is awarded by Cambridge University to all students who complete their undergraduate degree. This simply means that you can vote in the University's elections.

4. In 1994, I received my PhD degree in Neuropathology. Whilst I received my degree from the University of Cambridge, my research was performed at the Scripps

Research Institute in California. Whilst most American PhDs take 7 years to complete, mine took 3 years.

5. After returning to the UK in 1994, I started my medical degree (BM, BChir) at Oxford University. During my time at Oxford, I spent a year in Sri Lanka seeing poisoned patients. So even as a medical student, I was already seeing poisoned patients. I graduated from Oxford in 1998.

6. 'ScD' is a Doctor of Science. It recognises that I'm a specialist in my area and my research is of a high quality. I wrote 75 papers about poisoned patients which are published in major journals, were put together in a thesis, and submitted to Cambridge University. I received this qualification in 2014.

7. So those are my degrees where I've studied, completed exams or submitted a thesis.

Memberships and Fellowships

8. 'FRCPEdin' is the fellowship of the Royal College of Physicians of Edinburgh. One year after becoming a consultant in a permanent position, I applied for this fellowship to be taken in as a standard clinical consultant. I was a locum consultant from 2008-2009 in Newcastle and received a permanent consultant post in 2009 in Edinburgh. There are three Royal Colleges of Physicians in the UK – Edinburgh, Glasgow, and London. I happen to have a fellowship from Edinburgh.

9. 'FBPhS' is the fellowship of the British Pharmacological Society. This recognises that I'm a clinical pharmacologist, and that I do good quality work in clinical pharmacology. I received my fellowship in 2014. I have also received two major prizes from the Society – the GSK Prize awarded in 2008 for research in clinical pharmacology, and the Lilly Prize awarded in 2019 for longstanding leadership in clinical pharmacology.

10. 'FEAPCCT' is my inaugural fellowship of the European Association of Poison Centres and Clinical Toxicologists. This is the European specialist organisation I belong to. The Association brings all the poison centres and all the clinical

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toxicologists who work on poisons together on an annual basis to a congress. I was one of the first people to be awarded the inaugural fellowship in January 2014. I was probably one of the youngest of them.

11. So, these three fellowships simply say I'm recognised as being of reasonable quality by the organisations I work with.

Awards and Prizes

12. In 2017, I was awarded the Cullen Medal by the Royal College of Physicians of Edinburgh. It is awarded for the 'Greatest Benefit to the Practice of Medicine'. I received the 'Principal's Award for Impact' from the University of Edinburgh in 2020, building on the Cullen Medal. These simply reflect the work I've been doing for the last 20 years: preventing people dying from poisoning, mostly in Asia.

13. In 2007, I was awarded the Inaugural IUTOX International Congress of Toxicology Triennial Early Toxicologist Award. I've also been awarded the 'Best Emerging Medical Researcher in the UK' by the BUPA Foundation and the 35th Croom Lecturer Prize by the Royal College of Physicians of Edinburgh – both in 2008. These prizes also reflect the work I've been doing in Asia for the last 20 years.

14. More recently, I've been nominated on the Vox's Inaugural Future Perfect 50 List. The list features 50 people working to improve the quality of the earth.

Early Career

15. In 1998, I started working as a junior doctor. I spent three years as a junior doctor before I received a Wellcome Trust Fellowship to work in Sri Lanka. I spent four years seeing poisoned patients on a daily basis, mostly with pesticide, plant or medicines poisoning. There were relatively few patients presenting with recreational drug use in those hospitals I worked in.

16. I then came back to Edinburgh in 2005, completing my higher training in clinical pharmacology and toxicology, with a specialty in clinical toxicology.

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Current Role

17. I have about five jobs and I work a lot of hours. My main role is as a professor of clinical toxicology at the University of Edinburgh. I have been a professor since 2013, and I am also one of three professors of clinical toxicology in the UK.

18. One day per week, I work as an honorary consultant clinical toxicologist and pharmacologist at the Royal Infirmary of Edinburgh, NHS Lothian. I donate my time to this role to assist with my work at the University. My research is tied in with the patients I see, both here in the UK and in Asia. I perform ward rounds and treat poisoned patients.

19. My third role is to be on call for the National Poisons Information Service. I may receive a call at 3 o'clock in the morning saying, "I have a patient who's in cardiac arrest in Rotherham, please tell me how to help that patient.". There are currently 16 clinical toxicologist consultants in the UK, supporting the management of about 300,000 poisoned patients each year. Patients are mostly going to hospitals that do not have clinical toxicologists on their staff. The phone services allow us to take calls from doctors handling the more complicated patients, and to try and support them in the process. Alongside taking calls, I edit TOXBASE, which is an information database to assist in managing poisoned patients.

20. I am the Chair of the Clinical Standards Group which effectively means I direct the poison centres across the whole of the UK. I was appointed as the Chair on 1 April 2022. I'm now the senior clinical toxicologist in the UK and I do my research which, again, is almost completely based on poisoned patients.

Report - Preamble

21. I have been referred to the report that I prepared for the Crown Office and Procurator Fiscal Service on 2nd June 2017 (COPFS-00038), specifically to the second paragraph under the subheading 'Preamble'. I have written:

"I am a registered medical practitioner with a licence to practice and a Certificate of Completion of Training in Clinical Pharmacology and Therapeutics."

22. This simply means I'm a consultant. When we leave medical school, we become junior doctors and spend 3-5 years doing more basic work. This period allows junior doctors to figure out where they wish to specialise. Then we enter higher specialist training; mine was in clinical pharmacology and therapeutics, which includes clinical toxicology as a subspecialty. Once we've done those 3-5 years of higher training, we then get a 'certificate of completion of training'. Mine is in clinical pharmacology and therapeutics. I also see general medical patients as well.

23. *'registered medical practitioner'* means I have a GMC certification. Every year I have to reapply for a license to practice. I have to go through appraisal processes every year, and a validation process every five years. So that first sentence effectively says I'm doing all those things correctly.

24. In the same paragraph, I go on to say:
"I have over nineteen years' experience as a clinician and fifteen years' experience of treating poisoned patients."

25. I now have 24 years' experience as a clinician and 20 years' experience of treating poisoned patients.

26. A poisoned patient is someone who has been exposed to a poison, who may not actually be ill, but potentially could become ill, or is actually ill as a result of that exposure.

27. A poison can be so many different things. Typically, if we go to the wards in Edinburgh, they'll be medicines. People take overdoses of medicines, they have a moment of stress, of crisis, they take a medicine to harm themselves and it's often used as a method of communication. So, the majority of patients I see are patients who are self-harming or poisoning themselves. The other major group I see here in Edinburgh are patients who have taken excessive amounts of recreational drugs.

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So, they've taken heroin, they've taken cocaine, they've taken methamphetamines, and they're suffering the consequences of that. Many of the patients who are not so ill, as seen in the emergency department, are sent home from the emergency department. That management of the patient will be done with the TOXBASE database and the doctors in that department. The doctors treating those 'not so ill' patients may still call me for advice if they want to.

Crown Office and Procurator Fiscal Service (COPFS) Instructions

28. I remember receiving an email from Alastair MacLeod who was the Senior Procurator Fiscal Depute. The email was dated 29 March 2017 WIT-00042 and it asked if I had the relevant field of expertise to assist the COPFS. Then I emailed him back and said, "Yes I'm happy to do it." I try and support the government service as much as I can. Alastair then sent me the official instructions about a month later. The letter of instruction I received from the COPFS is dated 26 April 2017 (COPFS-02360).

29. I am asked if there was anything that I did not receive from the Crown that I would have expected to see. My answer is no. I would have asked for something if I'd felt there was something missing, and I don't believe I did.

30. In preparing my report I did not examine any physical items of evidence. I am asked if it is common practice for me to prepare a report without having examined any physical evidence. For me, it is. I'm a clinical toxicologist, not an analytical toxicologist. I don't measure things in bloods. I manage patients and try to understand from their history what happened to them. So, I take the information from the post-mortem report or from the toxicology reports and use that in my own report.

31. After producing my report, I was subsequently contacted two times, in January 2018 and April 2018 with follow up questions from the COPFS (COPFS-02018(a) and COPFS-02018). I was provided with a report from Dr Walker (COPFS-02408(a)) which confirmed a positive test result for anabolic steroids. I also

received a supplementary toxicology report, in relation to caffeine (COPFS-02382). I thought there was nothing there that affected my report.

32. I have been referred to the instruction from the COPFS on the top of page 2 of their letter of instruction:

"Given your experience, the Crown wish to instruct you to provide a general opinion on the individual and synergistic effects of MDMA and Alpha-PVP on the brain. In particular, the Crown are seeking to establish what effect the levels and combination of these drugs may have had on the deceased's mood, cognitive ability and behaviour."

33. I was provided with the Use of Force SOP by the Crown Office & Procurator Fiscal Service (COPFS), as part of my background reading for their toxicology instruction (COPFS-02360). I didn't go looking for it. I wasn't asked to comment on the SOP specifically in my instruction, however, I chose to refer to it because I thought that the way the incident was initially managed did not fit the SOP. I considered that the attempt at restraint was naïve and that, if Mr Bayoh had been treated as though he had a psychiatric illness, it might have resulted in a different outcome. This is not an expert opinion. I had the impression that all of the other expert reports I had been provided with concluded that death was inevitable from the point of contact with police. I felt that my view on this point was valuable, despite not being asked to comment on it. My view on this is based on my day-to day clinical experience on my ward.

Psychiatric Diagnosis

34. I am referred to paragraph 11 of my report, where I discuss Dr Lipsedge's retrospective diagnosis of Mr Bayoh. I state:

"Dr Lipsedge in this Expert Witness Report of 16th January 2016 provides a retrospective psychiatric diagnosis for SB. His review of the witness statements,

toxicology reports, and CCTV footage causes him to diagnose SB as having psychostimulant psychosis.

This is consistent with my interpretation of the witness statements. Clinical toxicologists would generally use the term 'drug-induced psychosis, in this case refined to 'stimulant or sympathomimetic drug-induced psychosis' (either ICD10 F14.5 or ICD10 F15.5, depending on the drug)".

35. 'Sympathomimetic' can be broken down into 2 parts. 'Mimetic' means mimicking, and 'sympatho' refers to the sympathetic nervous system. We have the sympathetic nervous system and the parasympathetic nervous system. The sympathetic nervous system is the fight or flight response. So, if someone gets stimulated, you get adrenaline surging around the body and you start either running away or fighting, whatever the situation you've come across. These drugs cause that natural fight or flight response, but at a greater degree. Stimulant and sympathomimetic drugs are very similar and can be very difficult to differentiate.

36. I am asked about the difference between the diagnostic terminology used by Dr Lipsedge compared to the terminology preferred by clinical toxicologists. I don't think there's a difference. So effectively, in two words, he's written what I've written in five words.

37. ICD-10 is the World Health Organisation (WHO) list of all the conditions that are recognised in the world. So, if I talk to a psychiatrist in my hospital, I would talk about drug-induced psychosis and that would be accepted by them because it's listed in ICD-10. The conditions are listed under specific codes.

38. Code F14 is mental and behavioural disorders due to use of cocaine. Code F14.5 says drug-induced psychosis, due to cocaine. They're very difficult to really distinguish their pathophysiological characteristics. If you could study in great detail, you might be able to see what's going on, but these patients are on the street or in the hospital having these psychoses.

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39. Code F15 is mental and behavioural disorders due to the use of other stimulants, including caffeine. So, caffeine is clearly a stimulant, it's what people take in the morning when they wake up. Code F15.5 says drug-induced psychosis, either with stimulants or sympathomimetic. If you go to Australia, the classic drug would be methamphetamine. Here, about 10 years ago, we had a problem with ethylphenidate, and all our drug-induced psychoses were ethylphenidate induced, which is a kind of amphetamine. There's lots of different drugs that can do this, and alpha-PVP is particularly good at doing it.

40. I'm asked if there are any equivalent codes in DSM-5. DSM-5 is the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders. I don't know. I'm not a psychiatrist. Psychiatrists are the only people who use DSM-5. It's a very valid system to use for psychiatric illnesses, particularly in the United States. It is also used globally. For all conditions - not just mental health ones - ICD-10 is a definitive system. In the UK, we do all our coding based on ICD-10. So, if you come to hospital with a drug-induced psychosis and you were discharged, you're coded using ICD-10, or maybe 11 now. It's just changing. While a DSM diagnosis may be discussed among psychiatrists, it wouldn't be used as coding in a British hospital at discharge and, as I'm not a psychiatrist, I wouldn't use that. A psychiatrist should be fluent in both systems.

Treatment of Psychotic Patients in UK Emergency Departments

41. At paragraph 20 of my report, I state:

"Patients with acute psychosis are frequently assessed and cared for in emergency departments across the UK. Doctors are familiar with how to manage these difficult and sometimes dangerous encounters."

42. In my experience, these are patients who are unable to be calm, unable to sit down, who are disruptive to themselves and to the ward. They are commonly under the influence of stimulant drugs. GBL withdrawal is a classic example we had for a while, and we have a particular medicine that works well for that, called baclofen,

in combination with diazepam. What I would do with those patients is to try to get them to understand the situation. Sometimes they have some understanding. I would use the words they're familiar with, and work in an empathic way, where I'm not aggressive towards them, and calm. I'm telling them they'll feel better with these medicines. They'll sometimes take the medicines, and you can see them just relax. Over the next five minutes, their body just changes, and they'll sit in a chair, and they become less confused.

43. Other times that doesn't happen. Sometimes I come onto the ward and there are police officers there. There are security guards holding people down because of aggression or because they have attacked another patient in the ward. We know we can get control of the situation by using physical restraint, but we would never leave it at physical restraint.

44. We would always then get intravenous access (via a cannula placed into their vein). If not possible we would give the medicine by the intramuscular route. We would use a medicine like diazepam or midazolam, which are sedative drugs (the opposite of a stimulant), which might settle them down. We might give a small dose of haloperidol, although we'd prefer not to if possible. We would dose them with diazepam until we've got control of the situation. Sometimes fairly small doses have an effect so that some of the police can leave or some of the security guards back off. Within 5-15 minutes, you'll see the patient fight less, start getting sleepy, and gradually you'll start backing off.

45. Ideally, you don't even lay your hands on the patient until the drugs are ready. So, if you can somehow keep talking to them and have them in one corner of the ward, you would keep talking to them while people draw up the medicines. When everything's ready, and there are people who know what they're going to do, we'll move in quickly, carefully hold the patient and try to give them drugs to calm the situation.

De-escalation Techniques

46. I have been asked to describe the verbal de-escalation techniques I would use when dealing with a psychotic patient. I would use a quiet voice, but one they can hear. Speak in a normal voice. You would ordinarily never yell at a patient. However, we do sometimes yell when things are going out of control and we're trying to shock them into not doing what they're doing, but generally we try and speak as calmly as we can. We try to always tell them what's going on. We tell them where they are to try to reorientate them. We try to encourage them, telling them that the medicines we're offering would be good for them. We're not getting informed consent for giving them medicines because they do not understand what's going on. I just want the medicines into them because I know it will calm them down. So just a calm voice, keep talking, use language they understand – don't use big words.

47. I've been asked to describe the body language I would use when trying to de-escalate a situation involving a psychotic patient. I would say 'warm and empathic'. You're aiming to not look aggressive. Sometimes I've seen in hospitals the security guards can be quite aggressive and get in a boxing position when these things are happening, which really doesn't help at all. Actually, I've sometimes asked security guards to leave because they are making the situation worse.

48. In general, I don't feel in danger with these patients. They could punch me, and I've been punched by a patient, but he was not psychotic; he was completely lucid when he punched me. Many junior doctors find these patients quite frightening, very difficult. But I think once you've been doing it quite a long time you generally know how to do things. You generally look after yourself; you've got people around you who can help you.

49. Trying to get that engagement with the patient, it's so important, that you sometimes put yourself in a little bit of risk to get yourself close to the patient. I remember one who came in with a boxing posture. He had big fists up, but he was so uncoordinated I was pretty confident that he wouldn't hit me hard, and I just talked him down to the bed and gave him medicine. He took his medicines, then went to sleep.

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50. I've been asked, in my experience, how successful using de-escalation methods have been in managing a psychotic patient. I'm not sure. I couldn't say it works in 80% of cases or in 20% of cases. I just know that de-escalation is where we start. There's so much variability between patients. It sometimes works very clearly. I can think of several occasions where it worked really well. Other times, it did not work and we've had to physically restrain people.

51. I've been asked, in my experience, if there are any kinds of approach that don't work as well with psychotic patients. Being aggressive, making the patient frightened, doing anything to stimulate their fight or flight response, is not going to help. So, you would always find in the hospital that people who are dealing with these patients are trying to be as calm as possible. It's wonderful to watch the nurses with these patients. They're really calm. They just try to settle things down. They're always talking in a nice voice, trying to get the situation under control, while at the same time someone will be phoning for security and for a doctor in case it doesn't work.

52. I've been asked whether I vary the approach I take, dependent on the cause of the patient's psychosis. Not really. The reason they come and spend time with us is because they've often got mental health illness, but we're not sure whether drugs have caused this particular psychotic episode. We work very closely with psychiatry colleagues. In terms of changing a patient's medicines or getting them to take their usual prescribed oral medicines, they will help us to do that. That's particularly true for a patient with mental health illness who's known to have psychosis regularly and comes into the ward. But for someone who we're not quite sure what's going on and where we think it's probably drugs, we would do pretty much the same thing, but we may not be successful at getting oral medicines into them.

53. I've been asked how long I would typically engage in de-escalation techniques before I consider the attempts to be unsuccessful. I'll try and give as long as I can, but it will also depend on the patient's actions. If they're physically assaulting staff or patients or kicking off violently then you would have a very short period of time to see if it worked. If the patient's quietly by their bedside and not harming

themselves or harming other people, you would probably spend longer. So, it would depend on the situation. But you would try to give that as long as you felt was safe. As soon as you realise that the patient's health, or another patient in the same ward's health, or healthcare workers safety is at risk, then you would move more rapidly to physical restraint.

54. I've been asked if the de-escalation technique can still be effective where the patient is initially displaying aggressive behaviour. It's going to be very variable with each individual patient. All I can say is that with some patients one is able to get through to them with these techniques and one is able to control the situation.

Chemical and Physical Restraint

55. Within paragraph 20 of my report, I state:

"Sometimes the agitation is too great for this approach to work and the person cannot understand the situation or calm down. In this case, the person must be physically restrained to allow rapid and safe administration of intravenous or intramuscular sedative drugs, such as diazepam or ketamine. Duration of physical restraint is kept to an absolute minimal to reduce the risk of complications. As soon as the patient is sufficiently sedated with medicines, physical restraint is withdrawn."

56. During the sedation process, those involved in the restraint would start to back off gradually as the situation starts to de-escalate. So, if 10 people were involved in the situation, 3 might back off, then 3 more until the patient is fully compliant. You wouldn't all take your hands off at the same time.

57. I'm asked if there is a recommended maximum amount of time before the restraint should be ended. I don't think there's a recommended time limit, but you're trying to keep it as short as possible, and you're hoping that within that timeframe, the drugs would start working.

58. In the last sentence of paragraph 20, I state:

"The process of physical restraint followed by sedation with medicines should ideally not be started until sufficient skilled staff are ready, roles known, and drugs drawn up."

59. This is a coordinated approach and one we're quite used to doing. Someone will take charge, usually the most senior doctor present. One doctor would have the task of getting access to the veins, putting a cannula in. The nurses are more experienced about handling medicines, so they will get and prepare the drugs for administration. Whilst the de-escalation process is going on, there's a discussion going on within the group of people present about how to take it to the next level. So even if you don't get to the point of giving intravenous drugs, they may well still be drawn up and ready. We just throw them away if we don't use them. The conversation may sound like, "We need the medicines now. Please prepare the medicines. Who's the person who can get the cannula in? Who's going to physically restrain?". This is a common situation in emergency departments. I'm sure in Edinburgh they do this at least weekly, and so people are just very used to it.

60. If physical restraint is going to be required, we will call security staff. However, if the situation looks particularly bad, we'll call the police because there's often police in the hospital. I might come onto the ward and find there's a mixture of police and security guards. Sometimes the physical restraint has already started, or sometimes I'm the person who will trigger it happening. Police and hospital security have a role in the physical restraint. My role would be to reduce the amount of time for physical restraint and lead or support the process of getting the medicines into the agitated patient.

61. When I say *"sufficient skilled staff"*, it's meaning having enough people that your physical restraint will work. What you don't want is having two people trying to physically restrain the patient when you need six, and you've got arms and legs thrashing about, and the patient's potentially hurting themselves because you're not holding them down effectively. They're more likely to hurt themselves if there's only two of you. It's also important for the safety of staff. If you're moving in to

restrain the person, and limbs are flailing, people do get hurt. But no one says, “You take the left leg.”. We just move in. We grab what we can. We know what we’re trying to do is physically keep the patient in the bed and safe until there’s medicines in the patient. We know that, within a few minutes of the drugs going in, there should be reasonably rapid de-escalation.

Excited Delirium

62. I am referred back to my report at paragraph 11, where I state:

“This is similar to the diagnosis of 'excited delirium' that is used in the USA. The term 'excited delirium' is not used in British clinical toxicological practice, being absent for example from TOXBASE, the NPIS database used by clinicians from primary care, ambulance services, and hospitals across the UK to guide management of poisoned patients.”

The use of the term excited delirium has been criticised and is controversial. The Royal College of Psychiatrists published a position statement in 2022 on the use of the terms ‘acute behaviour disturbance’ and ‘excited delirium’ in clinical settings.

Alpha-PVP

63. I am referred to paragraph 12 of my report, where I state:

“a-PVP is a relatively new synthetic cathinone stimulant drug, with similar effects to other stimulant drugs such as cocaine, amphetamine and methamphetamine.”

64. I am asked to explain what a ‘synthetic cathinone stimulant drug’ is. To provide some background, there are two large stimulant groups. There is cocaine, which comes from South America. Cocaine is purified from a a plant. The second group is amphetamines, which are mostly synthesised. Amphetamine is the core structure upon which so many different drug variations have been made. Methamphetamine is a variant of amphetamine. Hundreds or thousands of different compounds have been made based on the amphetamine chemical structure.

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65. ‘Synthetic’ means someone in a lab has made the compound by ‘tweaking’ the chemical structure of the drug compound. The danger is that these drugs are not tested on humans until they’re sold. The drugs are sold as a new cool chemical structure, and sometimes it kills a lot of people. When I say that the cathinone is ‘relatively new’, it means that it came out in the previous 10 years before the incident involving Mr Bayoh occurred.

66. ‘Cathinone’ is a sub-group of amphetamine.

67. ‘Stimulant’ means that it gives you this sympathetic effect where you run around. It makes you feel high. A stimulant drug is similar to caffeine, it gives you a buzz and makes you feel good, until you start crashing down.

68. In the next line of paragraph 12, I state:
“It blocks the dopamine transporter, increasing dopamine concentrations at synapses.”

69. Dopamine is one of the chemicals in your body, which is a neurotransmitter. Adrenaline and noradrenaline are the two drugs which you have surging around your body when you’re running because someone is chasing you. That’s a sympathetic effect. Amine is the chemical name. So actually, I should have written dopamine and noradrenaline transporter.

70. If you have your pre-terminal synapse on one side and your post-terminal synapse on the other, the chemicals are release from your pre-terminal synapse, pass through the gap, and hit the post-terminal synapse where there are receptors. When you release the chemicals from one side and it goes into the other side, you want to get rid of it afterwards. So, transporters take neurons back up into the pre-terminal site. Dopamine is released and it's a kind of really nice neurotransmitter. We all like having lots of dopamine around our body. When you block the transporters, there's too much there. You overstimulate things because you're not ending that stimulation.

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71. When we say that it blocks the dopamine and noradrenaline transporter, it means there's going to be more around in the synapse, so increasing the concentrations at the synapse. Therefore, you're getting more stimulation.

Experience in treating Alpha-PVP

72. In terms of my experience in treating patients who have taken alpha-PVP, I cannot accurately answer. One of the issues with alpha-PVP and many other recreational drugs, from a treatment perspective, is that we rarely have confirmation which drug the patient has taken. Confirmation can be achieved through taking a blood sample and testing for the presence of the drug. However that is not available in a useful timeframe to treat patients. I simply treat patients symptomatically. If the patient presents unwell and displays the symptoms of stimulant drug poison, I will treat them accordingly.

73. Testing for the presence of alpha-PVP in a patient's blood takes time. When I do my research studies, we always take a blood sample to say, "this patient's been exposed to X, Y or Z.". In clinical practice, it would take four weeks for that to come back and the patient is often either dead, alive, or home within 24 hours. We'd never get anything in a clinically relevant timeframe, so we don't do it as part of clinical practice. We simply treat patients according to how they look in front of us. So, if a patient comes in, and he looks like he's taken a stimulant or he looks like he's taken an opioid like heroin, we manage them according to the treatment pathways for those classes of drugs. They mostly get better once we look after them, but we don't know what precisely we're treating. If we were to take blood samples from all our patients and look at them over two or three years, we could see how things change over time; we can understand much better what's going on from a public health perspective. That tells us what is going on for future patients, but not for any current patients.

74. I don't think that alpha-PVP is particularly common in the UK. I've dealt with lots of patients with this syndrome of stimulation, where they are agitated and don't understand what's going on. On the ward, the patients can present physically

aggressive, and it's the doctor's role to talk to them and persuade them that taking some diazepam is a good idea.

Alpha-PVP Studies

75. Under paragraph 12, I have referred to a study reported from Yekaterinburg, Russia. Different parts of the world are affected by different drugs. So, if you go to New York, cocaine is really big. If you go to Sydney, methamphetamine is really big. In this town of Yekaterinburg, at the time of the study, there was a lot of alpha-PVP going around – much more than I've heard of anywhere else.

76. The researchers recruited a large cohort, over about two years, of 161 patients where they could only find alpha-PVP in their blood. Therefore, they were likely to be predominantly affected by alpha-PVP. Some people were taking ethanol (alcohol) with alpha-PVP. Ethanol is a downer, so sometimes ethanol with a drug can make the stimulant effect less severe. One might imagine that, if you're taking ethanol with your alpha-PVP, you'll be less sick, less psychotic – but maybe not, we do not have the data. In the study, there were people taking alpha-PVP with other drugs - not ethanol but other drugs including MDMA. What they found, when they compared these patients versus people who had taken other drugs, was that they were much more likely to have psychosis when they had taken alpha-PVP.

77. I have provided the Inquiry with a presentation (WIT-00005) that was given by one of my colleagues, Dr David Wood. The data from the Yekaterinburg study is contained with the slides at the bottom of page 2. The point I am trying to make in my report is that patients who had taken alpha-PVP are more likely to show psychosis than the control subjects(who had not taken alpha-PVP), 63.9% versus 35.7% in this study. The presence and absence of other drugs did not affect the incidence of psychosis.

Effects of Alpha-PVP

78. At paragraph 12.2, I have referred to the Swedish STRIDA project which lists the common side effects of alpha-PVP. Tachycardia is the most common side effect. This is where the heart beats very fast. If a person wants to run off because they are being chased, they would want their heart to go faster since this means that you breathe faster and can therefore run faster. If a normal resting heart rate is, for example, 70 beats per minute (bpm), you would want your heart rate to increase to 120 bpm when running. As a stimulant drug, alpha-PVP replicates this effect on the body. However, if your heart rate rises too high - for example to 180 bpm - this is hazardous.

79. A patient reported in a Swedish case series died from a cardiac arrest while restrained in a police vehicle (1). Other deaths have occurred from bleeding into the brain, probably due to the drug causing elevated blood pressure (1, 2). There is relatively little data on cause of death in patients dying after exposure to alpha-PVP only; it is not clear whether alpha-PVP commonly causes very fast or chaotic heart rhythms causing cardiac arrests.

80. The second most common side effect is agitation. If a person is being chased, they want to be aware of their surroundings. The person becomes more alert, their pupils dilate, and they are taking in more information. Agitation might be considered a level above alertness. The person is looking around, may not be quite sure of what’s going on, and puzzled. The brain is overstimulated.

81. Delirium and hallucinations are also side effects, which are less common than tachycardia and agitation. Delirium is very similar to agitation, with the addition of confusion, of not understanding what is going on. You can't respond to stimuli and often have delusional thinking. You're thinking everyone's attacking you when they’re not. With hallucinations, you're getting strange visions and sensations because brain synapses are being overstimulated.

82. Intense paranoia is also a side effect of alpha-PVP. Again, if you're being chased by someone, being a bit paranoid is probably quite a good thing. Looking around, being really worried about what's going to happen: that'll help you escape, but it becomes too much in these cases. You're overstimulated and, because you're

paranoid, you're often quite violent and aggressive. So, you see the world attacking you, and you're in this fight or flight mode. Therefore, you start fighting against whatever you come across. What's quite clear, when you're like this, is that you can't process instructions.

83. There are no reports of alpha-PVP damaging the lungs or respiratory system.

Alpha-PVP: Fatal Dose

84. I've been asked if I am aware of a fatal dose for alpha-PVP. There is no precise range identified for fatal cases, with a great deal of overlap occurring with cases in which the person did not die (2, 3). In two small case series of 12 and three deaths, respectively, the blood alpha-PVP concentrations varied markedly, ranging from 1.1 mcg/L to 6,200 mcg/L (2) and from 30 to >20,000 mcg/L (3). The median concentration in the case series of 12 patients was 23.8 mcg/L (table 1 in the paper) (2) - this means that more than half of the cases (six) had concentrations less than 23.8 mcg/L. For comparison, the median alpha-PVP concentration in blood samples from 24 additional individuals stopped for driving under the influence of drugs was actually higher, at 32.0 mcg/L (2). Amongst the case series of three deaths, blood concentrations were 33, 54, and >20,000 mcg/L (3). However, the majority of individuals had taken more than one drug and it was unclear which individual drug, or mixture of drugs, was responsible for the death.

85. Looking to paragraph 10 of my report, I have listed Mr Bayoh's alpha-PVP concentrations in the blood (taken pre-mortem) as 0.07 mg/L or 70 mcg/L. (I used mcg/L in my report as the standard unit of concentration. These are the same value, with the figure given in milligram or micrograms per litre.) Mr Bayoh's concentration falls within the above reported ranges of fatal alpha-PVP concentrations, in the lower range where individuals have usually taken more than one drug.

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86. The other fatal cases I have presented in paragraph 13 of my report had reported blood concentrations of 62.6 mcg/L, 174 mcg/L, 304 mcg/L and 411 mcg/L, all well within the ranges reported above.

87. The time at which the blood sample is taken relative to death influences the value recorded. The sensitivity of the person is also important. Some people might be more sensitive than others and therefore may die at lower concentrations. The receptors or channels in their cells may be different to those in less sensitive people and more severely affected by the drugs. Other individuals may break the drugs down more slowly than normal, leaving their blood concentrations higher than usual and them at risk of problems. Unfortunately, since these drugs have never been formally studied in humans, the details of such variation and sensitivity is not well understood.

88. So, I would have said that Mr Bayoh was on the lower range of these case examples, but still very consistent. If these case examples were all 10,000 mcg/L and Mr Bayoh died at 70 mcg/L - that would be strange, but they're not. They're all in the right ballpark. On the basis of this study and taking into consideration the limited research and writing around the subject, Mr Bayoh's blood concentration of 70 mcg/l is not inconsistent with a fatal dose. That being said, the surrounding circumstances of the death should not be ignored.

89. I've been asked specifically about the patient described in paragraph 13 with the alpha-PVP blood concentration of 62.6 mg/L. Since the sample was taken 36 hours after hospital presentation, I've been asked if there is any effect of metabolism here that needs to be taken into consideration. Yes. So, the concentration would have been higher at the time of his cardiac arrest, I think that's fair to say. How much higher? We've got no idea. One of the problems is, with a medical drug, we've had volunteer patients who have been given the drug, we've measured the pharmacokinetics (how the drug is absorbed, broken down and eliminated in the body), we know how the body handles these drugs. We've got no idea with these illegal drugs that have never been formally tested. They're thrown into patients. Some die; some don't die. You don't know how pure the drug is. You don't know if

these individuals were more sensitive or had a particular reaction to them. We just don't really know. We can make crude comments that this is a reasonably low level, but when they had the cardiac arrest it was probably quite a lot higher. But I don't know what the drug half-life is. I don't know how quickly the drug breaks down in the blood. We also don't know how quickly it breaks down after death. So, if a sample was taken post-mortem there would be some form of breakdown probably. However, because the blood's not flowing, and the liver's not doing what it should be doing, the rate of breakdown is probably different to that occurring while the person is alive.

Poison Scoring

90. I have been asked to explain the references to 'moderate' and 'severe' poisoning as mentioned in paragraph 12 of my report. The European Association of Poison Centres and Clinical Toxicologists set up a scoring system called the 'Poison Severity Score, about 30 years ago. Clinical toxicologists use this to qualify whether things are moderate or severe. I have provided a copy of the EAPCCT's paper on poison scoring (WIT-00022), which states:
"In each case, the severity of poison was grading using the standardised poisoning severity score, and the severity index is based on the clinical symptoms observed and not on the amount of drug in the blood."

91. Therefore, the scoring is based on how people present. In terms of the score, zero means no effect. Some patients who will come with a history have taken a drug in either a small dose, or they haven't actually taken any. They would score zero. You still have to monitor those patients because they can be well for two hours and then suddenly become ill.

92. Minor poisoning can be described as "mild transit and spontaneously resolving symptoms". This could be a headache, slightly fast heart rate, feeling anxious. Symptoms that settle down without the need for treatment.

93. Moderate poisoning can be described as "pronounced and prolonged" symptoms. This means that it is quite obvious that the patient is not appearing normal. Where

this is the case, the patient may require medicines to help them, for example benzodiazepines like Valium (or diazepam). This will help the patient to feel much calmer in the situation.

94. Severe poisoning can be described as “severe or life threatening”. The last category is death. So, somewhere between moderate and death, there’s severe where people are at risk of dying. If you have a heart rate of 180 bpm, you're so psychotic that you can't understand what's going on and you have to be pinned to the bed by six security guards while doctors give you medicines - that's severe. A severe score means that the person has to be treated rapidly or there’s risk of dying.

MDMA

95. At paragraph 14 of my report, I state:
“MDMA is a widely used entactogenic phenethylamine drug. It causes hyperstimulation of the central and autonomic nervous systems via increased release and reduced uptake of serotonin as well as dopamine and norepinephrine.”

96. ‘Entactogenic’ means ‘touching within’. Drugs such as MDMA instil feelings of empathy, affection, emotional openness, and enhanced sociability. So, MDMA is quite different to cocaine or alpha-PVP, that's why it's so popular. It's called ecstasy. It's a relatively safe recreational drug. They don't hallucinate very much. They don't go around having fights. They don't get into their car and crash their car.

97. ‘Phenethylamine’ is a class of amphetamine drugs.

98. ‘Hyperstimulation of the central and autonomic nervous systems’ is where your brain gets overstimulated with this increase of serotonin. Serotonin is another neurotransmitter. So, again, it's about increased release. You get more of these neurotransmitters in the synapse, so therefore you get overstimulation and also reduced removal.

Witness Signature …… [redacted] …….

99. The central nervous system is your brain and your spine. The autonomic nervous system is all of the kinds of unconscious bits of a person's peripheral body function. So, when your heart goes up, it's driven by your sympathetic or parasympathetic nervous systems, which are these unconscious day to day systems. They need instructions, they're unconscious instructions. You don't say, "I want my pancreas gland to work well now." It just works, and that's because of the autonomic nervous system. So, your fight or flight response is controlled by your autonomic nervous system. You don't think, "Oh my gosh, I've got to run really quickly now because there's a man chasing me.". You just do it and that's your autonomic nervous system kicking in.

100. At the next sentence in paragraph 14 of my report, I state:
"Severe toxicity is relatively rare, with a poor dose-response relationship perhaps due to individual variation in metabolism of the drug."

101. When I say that severe toxicity is relatively rare, this comment is backed up by the case series referred to in paragraph 14.2 of my report. This is the biggest case series that I'm aware of. I was asked to talk about brain effects, so I haven't gone to all the other effects of MDMA, but in terms of psychosis in this case series, psychosis was only 6.3%. I have already referred to psychosis being present in 60% of patients with alpha-PVP intoxication. I have also discussed the fact that the presence of other drugs does not appear to increase ore reduce the problems associated with alpha-PVP. Three drugs in the study: tryptamines, MDPV and methylphenidate, had high rates of psychosis. The researchers were looking for psychosis but found very little with MDMA. So, was Mr Bayoh's psychosis due to the MDMA? The evidence suggests that it was probably not.

102. I've been asked if the drugs listed in paragraph 14.2 of my report – tryptamines, MDPV and methylphenidate – are similar to MDMA. Tryptamines are a quite different class. MDPV is a phenylethylamine, so it's more similar to MDMA, and methylphenidate is a stimulant. Methylphenidate is used to treat attention deficit hyperactivity disorder (ADHD) and sometimes narcolepsy.

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Effects of MDMA

103. At the last sentence of paragraph 14.1 of my report, I state:
"Hyperthermia is well recognised, resulting sometimes in disseminated intravascular coagulation, multi-organ failure and death."

104. Since MDMA is a stimulant, you are getting some aspects of stimulation, and one of those is increased temperature. So, when a person has taken too much MDMA and the body is responding badly, the person's temperature increases. That's a key cause of death in these kinds of drugs.

105. 'Disseminated intravascular coagulation' is where you have problems with your clotting system. Your clotting system goes haywire. We need the clotting system because if someone cuts us, we need to stop bleeding. We need to clot and cause a scab, but sometimes that system in our bodies goes wrong. The problems are driven by the high temperature, and then you stop clotting. Your organs start failing. So multi-organ failure – your liver, your kidneys, your lungs stop working and then you die.

106. MDMA commonly causes tachycardia and high blood pressure (4). Cardio-myopathy can occur with chronic use (5). Cardiac arrests do occur but rarely - probably due to constriction of arteries supplying the heart with blood causing myocardial ischaemia or infarction (5, 6).

107. MDMA use is not associated with direct lung damage or with respiratory arrest. It has been associated with pneumothoraces ('popped' and collapsed lung) on a few occasions (4); this was not reported in My Bayoh's case

Experience of treating patients with MDMA

108. I believe I have been involved in the management of 5-10 patients who've died from MDMA. We think it's MDMA based on the patients' histories which described them as taking MDMA.

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109. The patients who present with a history of MDMA typically present not too differently from alpha-PVP intoxicated patients. They'll often come in very stimulated, very agitated. You bring the patient's foot back and it starts flapping (termed 'clonus'). That tells you there is over-stimulation from the brain down to the junction between the nerves and the muscles. We treat these patients with diazepam as our first line of treatment. We try and give them as much diazepam as we can to calm them down, to get their temperature down. If that doesn't work, there are particular drugs called serotonin antagonists that we will give.

110. The problem is there's too much serotonin in the synapse. There's too much stimulation of that nerve or muscle. If you can administer a medicine which blocks the receptors, then it doesn't matter how much drug or how much transmitter is there because the effect can be blocked by the drug. So, we'll give patients a medicine such as chlorpromazine to block the receptors. Alpha-PVP doesn't seem to affect the serotonin receptors so much, so we wouldn't be giving that drug to them if we knew it was alpha-PVP. If we didn't know it was alpha-PVP and they looked the same, we would probably give that drug, but it wouldn't be so effective.

Post-Mortem Consultation Note

111. I have been referred to a consultation note (COPFS-04194(a)) from Dr Kerryanne Shearer, who was the lead pathologist and performed the original post-mortem. The note is dated 4 June 2018.

112. At the bottom of page 1 of the note, it states:

"A toxicologist is the best person to speak to the effects of MDMA and Alpha-pvp. MDMA is ecstasy and makes people 'happy' and Alpha-pvp can make an individual agitated and hallucinate. Both drugs can cause sudden death due to cardiac arrhythmias. Either drug could have killed the deceased. Taking both drugs would have caused compounded effects."

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113. This comment is talking about the cardiac effects. We know that drugs cause cardiac arrhythmias; cocaine has a particular problem with this. Any stimulant can cause the heart to go very fast and sometimes into chaotic rhythms. MDMA is a less severe stimulant and from a clinical toxicology perspective, I don't consider MDMA to be a drug which commonly causes cardiac dysrhythmias and death, which I would consider cocaine to be.

114. The sentence *"either drug could have killed the deceased"*, yes that's true but it's much more likely to be alpha-PVP because sudden death is not a common problem with MDMA.

115. The sentence, *"taking both drugs would have caused compounded effects"*, well we know with psychosis that's probably not the case as a result of the Russian research study but that's looking at one particular problem. I don't know the data for cardiac dysrhythmias. Without having this particular data, I can't give you a precise answer, but I still think it's more likely to be alpha-PVP than MDMA.

116. I have been referred to the first paragraph on page 2 of the consultation note, where it states:

"The level of the drugs present is not necessarily significant and toxicity is not necessarily dose dependent. If you take them there is always a chance they will cause death. It is their presence that is significant."

117. A better word for 'level' would be concentration. The concentration of the drug in the body, it's very difficult to be precise about what that means. Where the note says, *"it is their presence that is significant"*, I agree with that. You get some information from the concentration. If it's a very, very low concentration of the blood, which is not consistent with other cases in literature, you would have to say on the balance of probabilities perhaps that probably wasn't the important drug. The note says that the concentration is *"not necessarily significant"*. It can be significant, and I've tried to show that the alpha-PVP concentration measured in Mr Bayoh was similar to what we found in the literature, so I think that's consistent.

118. The *"toxicity is not necessarily dose dependent"* is absolutely true because some people we think are probably sensitive and some people are not sensitive, but we don't know because we haven't done the studies to evidence this. We can't do the studies really; we can just think that's probably the case.

119. *"If you take them, there's always a chance they will cause death."*. Yes, but I think with MDMA, it's much less likely to cause death than alpha-PVP. There are very large numbers of people taking MDMA in the UK and there are relatively few deaths from MDMA. There are very few people taking alpha-PVP that I'm aware of, and there are cases in the literature of death. Anyone who's severely agitated is a higher risk of death than someone who's not severely agitated, and we know that alpha-PVP causes a very high rate of severe agitation and psychosis.

120. I have been asked to comment on the fact that Mr Bayoh's friend, Zahid Saeed, has also provided evidence to the Inquiry that he consumed the same drugs before the incident on the morning of 3rd May 2015. I understand that Mr Saeed did not display the same symptoms as Mr Bayoh. The problem is the purity of these tablets vary by batches. If Mr Saeed truly took exactly the same number of the same tablets from the same batch with likely the same purity of the alpha-PVP (because I don't know what the purity was of these tablets), then having such major differences would be unusual but not impossible. Clearly, we do think there's a difference in how people respond to drugs and perhaps their own mental status at the time would alter that, because clearly how your body is behaving when you take the drugs may well affect it.

Role of Alpha-PVP and MDMA in causing drug-induced psychosis

121. I am referred to paragraph 15 of my report, where I state:

"It seems likely that a-PVP was primarily responsible for SB's drug-induced psychosis since this feature is a common consequence of a-PVP exposure resulting in presentation to hospital. It is possible that exposure to MDMA increased the risk of drug-induced psychosis; however, the same study showed no clear

increase in the incidence of psychosis in patients taking other recreational drugs as well as a-PVP, compared to those taking a-PVP alone. This evidence makes MDMA unlikely to be the primary or secondary cause of the psychosis."

122. I still agree with this comment. I think based on the data from the presentation[1] I have shared with the Inquiry, where evidence said that 65% of patients were psychotic with alpha-PVP intoxication compared to other studies showing 4% of patients were psychotic with MDMA intoxication. I still agree with this comment even considering the concentrations found in Mr Bayoh's blood samples.

123. I continue at paragraph 15 to state:
"It is possible that exposure to MDMA increased the risk of drug-induced psychosis; however, the same study showed no clear increase in the incidence of psychosis in patients taking other recreational drugs as well as a-PVP, compared to those taking a-PVP alone. This evidence makes MDMA unlikely to be the primary or secondary cause of the psychosis."

124. I still agree with this comment. From the data we have, that seems perfectly reasonable. I think that's true on the balance of possibilities.

Report of Professor Mary Sheppard

125. I have been shown the report (COPFS-00027) prepared by Professor Mary Sheppard, dated 1 December 2015. I have been referred to page 5 of the report where Professor Sheppard has been asked about the physiological effects of the drugs detected in the toxicology sample, both individual or in combination on Mr Bayoh in the circumstances of his arrest. Professor Sheppard has commented:
"While the drugs may have an effect on the heart, there is no evidence pathologically of any damage to the heart due to drugs."

126. You would not expect pathological evidence of any damage to the heart with a toxicological death. So, if a drug caused death by cardiac dysrhythmia, you would

[1] WIT-00005

not expect to see pathological death damage to the heart due to drugs. For example, cocaine blocks a channel called a sodium channel and, when that channel is blocked, the heart rhythms don't work well. Patients go into a chaotic rhythm and resulting in rapid death. There may be some post-mortem changes, but the actual changes are going on at the cell level or a blockage of channels. So therefore, you would not expect any pathological changes in the heart when a person has been killed directly due to the effects of the drug. It's not like a heart attack. If you have a clot in your blood vessel causing a heart attack, that takes time, and one can see the pathological effect on the heart. You don't see that with drugs.

127. I was not asked to consider the effects of MDMA on the heart when I was originally instructed. In my clinical experience, it mostly causes your heart to go fast. The common feature for MDMA, but particularly alpha-PVP, is tachycardia. We can see from the STRIDA project that 80% of cases had tachycardia when they came to the hospital. I don't know what it would be for MDMA, I suspect it's quite a lot less. I don't consider either MDMA or alpha-PVP to block the sodium channel or the potassium channel, which would be the commonest causes of a drug-induced cardiac death. Restraint also cause hypoxia (a lack of oxygen in the body). You're getting more stress on the heart, you've got the sympathomimetic drive which is causing a very rapid heart rate. And so just the effect of having hypoxia and a very fast heart rate may well be enough to cause you to have a cardiac arrest and you to drop dead from an effect on the heart, but it's not a specific effect of the drug on the heart. It's an effect of the drug on the cardiovascular system as a whole where it drives it to go fast because of the effect on the brain, but if you're hypoxic because of the restraint or because of exertion, you then are at risk of having a ventricular fibrillation or chaotic heart rhythm and dying.

128. The absence of damage does not prevent those two drugs having caused the cardiac dysrhythmia in the context of the fast heart rate, restraints, and hypoxia. I think it's much more likely to be alpha-PVP than the MDMA in that circumstance. I've been asked whether any further testing or examination of the blood and urine samples held for Mr Bayoh would assist in being able to comment on the effects of both drugs on the heart. No, I don't think so. There are clearly cases of people who

drop dead having taken these drugs, I think even for MDMA. So, there is sudden cardiac cause of death, but we don't know what the underlying issues are with those people. Have they taken much higher doses than most people use? It's just not very common with MDMA. It sounds like it's probably more common in alpha-PVP. One of the problems is the cases where people get to hospital, they are more likely to survive. A great majority of deaths occur in the community, and therefore you don't get the blood samples one needs for these kinds of studies.

129. I am referred to the second question on page 5 of Professor Sheppard's report where she is asked about the physiological effects of the CS/PAVA spray, both individual or in combination on Mr Bayoh in the circumstances of his arrest. Professor Sheppard has commented:
"While the CS/PAVA may have an effect on the heart, there is no evidence pathologically of any damage to the heart."

130. Whilst I am an expert in clinical toxicology, there are over 17,000 poisons listed on the TOXBASE database. I am aware that CS is not known to be a cardiotoxin. I would not expect the level of exposure that Mr Bayoh had to the CS or the PAVA to be a problem for his heart, but these are irritants. These are substances which work on your airways. If a person is sprayed with CS, their eyes are going to be streaming, their nose is going to be streaming. However, as long as they're not in a restricted space, they're probably going to be fine.

131. Most CS or PAVA deaths have occurred following exposure in restricted spaces. Individuals sprayed with these irritants are put into a small enclosed area and are not able to ventilate it off. The substance goes into the lungs, producing lung damage. People can die from this lung damage.

132. I am aware that at the time that Mr Bayoh was potentially exposed to CS/PAVA, he was outdoors walking along the street. He displayed no obvious effects to exposure. Mr Bayoh appears to have been psychotic and therefore unable to respond to what happened to him, but what you're expecting is airway problems. These irritants work because people start weeping. They're in pain, you're worrying about sneezing, coughing, sore throat, wheeze, shortness of breath, streaming

nose, and chest tightness. Where the person is breathing in contaminated air, which is causing an irritation, their lungs constrict – similar to asthma. A person can get breathless with that, and so respiration may be irregular with period of apnoea. Apnoea is where a person stops breathing. Therefore, you can do what you want with them, effectively. The agents are not particularly dangerous, and the only deaths tend to occur when they're used in an enclosed space.

133. Where a person has been sprayed with an irritant, they are going to have a big sympathomimetic surge. People don't like being sprayed with the CS gas or PAVA, so therefore they've got the fight or flight response, so therefore the heart rate will go up. If a person has got drugs on board, perhaps that'll make things worse. From reading through the witness statements disclosed to me in preparation of my report, My Bayoh did not seem particularly affected by the exposure to CS/PAVA spray. He didn't try and run away. So, exacerbating his fight or flight response may have been a bad thing but, on the balance of probabilities, I don't think it had an effect.

134. I have been asked whether I would expect to see any pathological damage to the heart as a result of exposure to CS and/or PAVA spray. I would not. Whilst the CS/PAVA may have had an effect, I think it's unlikely, on the balance of probabilities, to have had an effect on the heart.

135. I've been asked if I have any direct experience in treating patients that have been exposure to CS or PAVA. I don't think I have treated patients directly; however I have experience in providing advice to clinicians over the phone who are dealing with exposed patients.

136. I've been asked about the advice I would be giving to clinicians who are dealing with CS/PAVA exposed patients. I would advise that it mostly will settle down with time, but good ventilation is important, and that's pretty much it. We'd observe the patients for a period of time. If exposure to CS is confirmed, hospital treatment is rarely needed because spontaneous recovery usually occurs rapidly within 15 to 30 minutes of cessation of exposure. So, again, care is about keeping the patient in fresh air and not letting them re-inhale the stuff from enclosed spaces. Then,

really, you're thinking about respiratory complications. If someone's been quite heavily exposed and they've unfortunately got respiratory complications – perhaps they have severe asthma, perhaps they have a higher dose of exposure – then you might expect them to have respiratory problems. You'd see that by someone having difficulty breathing, and we'd bring them to hospital. However, most of these patients wouldn't come to hospital.

Effects of Steroid Use

137. Nandrone is a form of anabolic steroid. I have looked to TOXBASE for guidance on the link between Nandrolone use and cardiac arrhythmias:
"Toxicity following acute overdose of these steroids is expected to be low. Chronic exposure may lead to serious symptoms including cardiovascular effects. Deaths associated with chronic use usually have a cardiovascular cause with findings at autopsy."

138. So, you can get cardiomyopathy. If you have chronic use of nandrolone, your heart just stops working very well, and you get problems with the muscle cells. Your heart has to get bigger to ensure it does enough work. You can have blockages of blood vessels causing infarctions. So clearly chronic nandrolone use may well have caused Mr Bayoh's heart to be pathologically prone to problems.

139. I note that Dr Karch's report (PIRC-02527(a) refers to myocardial remodelling of both ventricles. I have referred back to the post-mortem report (PIRC-01445) which states at page 9 that, "*the heart (430g) was of normal size and configuration. The coronary arteries, myocardium and cardiac valves were normal.*". When I read this, there is no indication of chronic drug use causing heart damage (cardiomyopathy). The heart gets damaged, stops working well, and then you start seeing changes in the heart to compensate for that. If it were true that Dr Karch did identify myocardial remodelling of both ventricles - and that doesn't seem to be consistent with the former post-mortem - then you could say there's damage to the heart occurring from chronic use of possibly nandrolone, or some other drug, which

has caused this person to be at an increased risk of dysrhythmias. However, the post-mortem report suggested that there was no damage to the heart.

140. I've been asked if any dose level of nandrolone can be attributed to causing myocardial arrhythmias. I don't know of it. I doubt there is a dose level, since it is daily use for many months, if not years, that's causing these problems with the heart. Therefore the concentration you see on any day is going to be irrelevant, it's not going to be the key reason. It's not as if this, by itself, causes the cardiac problems or makes you more at risk. It's the chronic use causing the cardiotoxic effects. So, the cardiac remodelling is about expansion, ventricular hypertrophy, to try to compensate for these problems. Your heart changes, and that's fairly easy to see. Any pathologist, from my understanding, would be able to see that to know that there was a problem or not.

141. I understand that the Chair to the Inquiry will reach a conclusion, on the balance of probabilities, as to the factors contributing to cause of death. Having considered the evidence, I have found it difficult to reach my own conclusion as to the actual mechanism of death, although it is understandable that an individual would die under similar conditions. It is my opinion on the balance of probabilities that but for Mr Bayoh's encounter with the police that morning, and the subsequent restraint, he would not have died. I consider that a-PVP may have made a material contribution to Mr Bayoh's death, as it caused psychosis, which led to him being unable to understand instructions, and would also have increased his heart rate. I understand that the term 'material' in this context means more than minimal. As such, it is my view that the mechanism of death was multifactorial. I do not consider, on the balance of probabilities, that MDMA or steroids made a material contribution in Mr Bayoh's death. MDMA and steroids could have increased Mr Bayoh's risk of developing psychosis, but we can never know at an individual patient level, we can only work at a population level and, I think, on the balance of probabilities, it's unlikely.

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142. 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.

May 4, 2023 | 6:02 PM BST

Date .....

New references

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