Tuesday, 16 May 2023

(10.00 am)

LORD BRACADALE: Good morning Professor Eddleston.

A. Good morning.

LORD BRACADALE: Your evidence will be taken by Ms Thomson, Counsel to the Inquiry. We normally have a break at 11.30 but if you wish a break before that, please just let me know.

A. Thank you.

LORD BRACADALE: Would you now please say the words of the affirmation after me.

PROFESSOR MICHAEL EDDLESTON (affirmed)

Ms Thomson.

Questions from MS THOMSON

MS THOMSON: Good morning, Professor. What is your full name please.

A. My name is Michael Philip Eddleston.

Q. How old are you, Professor Eddleston?

A. 54.

Q. And you are a professor of clinical toxicology at Edinburgh University?

A. That's correct.

Q. Can I ask you to open up the blue folder that's in front of you. There are a number of documents there that you might find it helpful to refer to whilst you give your

evidence, so we will begin by just looking at what's in the folder. The first document in the folder should be a copy of your CV, that's witness 00006.

A. Yes.

Q. It's an extensive CV, I think it extends to 30 pages or thereby. We don't need to go through it in any great detail. The Chair to the Inquiry and the Assessors all have copies which they can read at their leisure.

The next document should be a letter of instruction sent to you by the Crown Office and dated 26 April 2017, that's COPFS 02360. Do you have that?

A. Yes, I have that.

Q. There should then be the report that you prepared in answer to the questions asked of you by the Crown Office.

A. Yes.

Q. Your report is dated 2 June 2017 and the reference is COPFS 00038.

A. Yes.

Q. Finally there should be a copy of a statement that you gave to the Inquiry -- we will perhaps bring this one up on the screen -- that's SBPI 00317 and if we scroll down a little, we will see that the statement was taken on 6 January, amended at the end of March, and if we scroll to the very end of the document, we should see that it

was signed on 4 May of this year. Sorry, just a little bit up from the references I think. There we are. Just a few lines down. There we go. 4 May 2023.

Professor Eddleston, your signature has been blanked out on the version on the screen but can you confirm please that you have signed or e-signed every page of the report --

A. Yes, I did.

Q. -- that's in the folder in front of you.

Returning to the screen at paragraph 142 you concluded your statement with the words:

"I believe the facts stated in this witness statement are true. I understand that this statement may form part of the evidence before the Inquiry and be published on the Inquiry's website."

A. I agree.

Q. As I have said, you can refer to any of these documents at any time if you would find it helpful to do so and if there's anything that you would like me to have put up on the screen, please just let me know.

I want to begin by asking you a little bit about your qualifications and experience and if we can perhaps scroll to the top of this document and go to the second paragraph, you explain that you are a professor of clinical toxicology and a consultant clinical

toxicologist and pharmacologist and you give your qualifications and there then follows a very long list of letters after your name so I want to ask you a little bit about your qualifications.

In paragraph 3 you explain that you began your university education with a bachelor's degree at Cambridge University. What subject did you read at Cambridge?

A. I read medical sciences.

Q. Medical sciences, and so you were awarded the bachelors degree and then subsequently the Masters of Arts; would that also have been in medical sciences?

A. The masters is non-specific for a subject. It simply means that four years before you finished your undergraduate degree.

Q. Right, I see.

A. It's simply a voting -- by becoming a masters you're allowed to vote in the Senate, so it's the old -- it's a 500-year old tradition.

Q. I see. So it wasn't a separate qualification as such?

A. I did no work at all. I probably just paid money.

Q. It's simply a token from the university four years after you graduate?

A. A gift, exactly.

Q. A parting gift.

In paragraph 4 you explain that in 1994 you received a PhD in neuropathology. That degree was from the University of Cambridge but your research was in California and you completed that in three years as opposed to the seven for an American PhD.

Again, just scrolling down a little to paragraph 5, you explain that you then began your medical degree in 1994 at Oxford on this occasion and during your time at Oxford you spent a year in Sri Lanka seeing poisoned patients. Now, elsewhere in your statement you explain that a poisoned patient is someone who has been exposed to a poison, so it might be convenient at this juncture to ask you to explain to us what is a poison?

A. So a poison -- there's a huge variety of different compounds which cause harm to the body and generally a poison is something that's ingested, so in contrast to venom. So a snake venom -- a snake is not poisonous, it's venomous, so things like a scorpion or a snake, they inject the poison, that therefore becomes a venom, into the body. Poisons are generally exposed to you via ingestion but they can be through the skin and it can be via breathing.

Q. So poisons would include then things like medicines?

A. For sure.

Q. And recreational drugs?

A. Yes, it's often about the dose.
Q. We will talk more about the dose perhaps later this morning. Returning to Sri Lanka, how did that year in Sri Lanka come about?
A. I was writing a textbook and I had no money and I had -- so I took a year off medical school to write the textbook and it was cheaper for me to go and run clinical trials in Sri Lanka and I paid $7 every two weeks for food and $50 a month for accommodation and became very slim and saw patients, wrote lots of papers and wrote the book. But it gave me the chance every day to see a poisoned -- to see all the patients coming to the hospital who would be described as poisoned patients.
Q. What types of poisoning did you see in Sri Lanka?
A. The most common ones there were plant poisoning and pesticide poisoning. As in the UK people would take medicine at moments of stress or crisis, in Sri Lanka they don't have medicine cabinets, there's no medicines in the houses, but they have pesticides. 85% of the communities around this hospital were using pesticides on a daily basis, so at moments of crisis when they grab something to communicate -- and mostly self-harm and self-poisoning is about communication, "I'm angry, I'm distressed, you're not listening to me". It's a very

dramatic way of getting a message across. In Sri Lanka it was pesticides and plants but some small amounts of medicine. So related to what I do here but not quite the same.

Q. So your interest in poisoning then was well-established even before you had completed your medical degree?

A. Yes.

Q. You graduated in 1998 and if we can perhaps scroll down to paragraph 15 you explain that following your graduation you started work as a junior doctor and then secured a fellowship to return to Sri Lanka where you spent four years seeing poisoned patients on a daily basis.

A. Yes.

Q. What was your role when you were in Sri Lanka? What post did you hold?

A. So I was a Research Registrar, which would mean I'm superfluous -- I'm an extranumerary within the hospital. I had medical licensing in Sri Lanka to practice and therefore to see patients independently but always under the supervision of a consultant, a Sri Lankan consultant. Because there were relatively few doctors on the ward and there were many poisoned patients and I had skills and I was recognised to be a competent doctor I really saw the patients by myself,

independently from the consultants and discussed cases. But it would mean -- effectively I was a supernumerary doctor but with a licence to practice in Sri Lanka, which would be unusual for a non-Sri Lankan.

Q. Again, were the poisons that you saw, the cases of poisoning that you saw, largely plant and pesticide-based?

A. So pesticide number 1, plant number 2 and medicines number 3. Recreational drugs were very rare.

Q. Moving on to paragraph 16, you have explained that you returned to Edinburgh in 2005 and completed your higher training in clinical pharmacology and toxicology with a speciality in clinical toxicology. I wanted to ask you a few questions around that. Can you help us to understand what is meant by higher training for a doctor?

A. So when we start out for the first two years we're trying to become vaguely competent with a more general knowledge looking at surgery or paediatrics or medicine or psychiatry. You're simply trying to get experience. There's a big step up from being a student to suddenly having responsibility, so those first two years roughly are spent just trying to get as much experience as you can and then the next two years are spent trying to work out do I want to become a paediatrician or do I want to

become a surgeon. You might start moving towards jobs that give you experience in those. Then after about four years you would apply for a position, for a training number, that's how it used to be called, of five years of training to become a surgeon, or to become a clinical pharmacologist in my case, or a general physician. It might take you eight years depending on how many different subjects you want to put together, it can take somewhere between five and eight years. At the end of that you become a consultant, at which point you are considered independent and competent.

Q. You mention clinical pharmacology and toxicology. Can you explain what those are and what the difference between them is?

A. So they are a speciality of medicine. If we think of paediatrics, medicine, obs gynae and surgery as the main areas, this is one of the specialties within medicine. So I would have done my first four years trying to do as much medicine as possible and then I have taken a choice to do clinical pharmacology. So clinical pharmacology is normally combined with general medicine, so the whole of general medicine -- leave it at that for now -- and pharmacology is about understanding how medicines work in our body, so how they get into the patient, how they spread around the patient and how they work in the

patient. So clinical pharmacology is generally about a therapeutic dose of a medicine and how a person might use those medicines if they take them every single day.

Clinical toxicology is a further subspeciality within clinical pharmacology when we are talking about overdoses. Those overdoses can be a moment of crisis when someone takes an overdose of a medicine or perhaps the medicine has a bad reaction in patients and we have adverse drug reactions to the medicines which -- some people have a problem with certain medicines and we study those as well. And we study things like recreational drugs and in my case plant and pesticide poisoning.

Q. When did you complete your higher training?

A. I got my first consultant job in 2008 in Newcastle where I went as a locum consultant, so in your last six years of higher training you can start looking to see if there are jobs available and there was an opening in Newcastle where I went and worked part-time as a locum consultant which is kind of that transition between the end of your higher training and becoming a full-time consultant.

Q. Am I right in thinking that in 2009 you then secured a full-time permanent consultancy in Edinburgh?

A. So that was with a senior clinical fellowship, so as an academic I'm employed by the university and my interest

is doing as much research as I possibly can to try and address questions I see in the patients in front of me. So I applied for a Scottish senior fellowship, I was awarded that which gave me a salary for four years. By getting that the university took me on and then NHS Lothian took me on as an honorary consultant. So I don't get paid anything by the NHS and I give one day a week of my university time to the NHS to do my work. So that's when I got my -- effectively my tenured job but it wasn't in the NHS it was in the university.

Q. Yes, you explain further into your statement, if we can perhaps look at paragraph 17, that you have a number of jobs. You say, "About five jobs", and you work a lot of hours, that your main role is as a Professor of clinical toxicology at the University of Edinburgh and that's been a post that you have held since 2013 and you say you are one of three Professors of clinical toxicology in the UK. Should we understand there are only three Professors of clinical toxicology in the UK?

A. Yes.

Q. So you explain -- and you told us a little about this already at paragraph 18, that one day a week you work as an honorary consultant clinical toxicologist and pharmacologist at Edinburgh Royal Infirmary. You donate your time, you're not paid for your time, to the role to

assist with your work at the university. Your research is tied in with the patients you see here in the UK and in Asia and you perform ward rounds and treat poisoned patients.

Can you help me to understand the difference then between your role as a professor at the university, which I presume is a more academic role, and your role as an honorary consultant which I think must be a more clinical role?

A. Exactly.

Q. Can you tell us a little bit more about these two roles that you hold?

A. So the simplest role is to describe the honorary consultant role where one day a week I go into the hospital and I see the patients there. So I have an honorary contract with NHS Lothian, I have the same rights, the same -- I'm perceived to be the same level as my colleagues who are NHS consultants and I fit into a rota of five or six consultants now and we share out the workload. I would see the patients in various parts of the hospital. I try and support their care and support their discharge really. That would be my routine work.

The rest of the week -- I do another day which I probably -- well, 19, if I put number 19 in now if

that's possible?

Q. Absolutely. Let's bring up number 19 for you.

A. It kind of works better -- well, so I see patients physically myself, actually examine patients and make decisions with them. At the same time I support the rest of the UK to care for these patients in hospitals where there is no clinical toxicologist. There are 16 of us in the UK, there are only six hospitals which have a clinical toxicology consultant in the hospital. So they will be seeing patients like I do one day a week and the rest of the time we -- well, one day a week each roughly we then support other hospitals across the UK to try and help their doctors care for patients.

So those are the two jobs, one of which is virtual effectively on the telephone and one is real, but they're very much the same job. Then the rest of the time I'm a university professor and I will be asked to teach, I will be asked to sit on committees to decide whether we should do various trials in the hospital. I -- but predominantly what I do is I write grants, I write papers and I run research. So every five years I get assessed and that's assessment on how many grants have I brought into the university, how many papers have I written and what impact has my work had globally, so that's what I'm judged on in through the university.

Q. Thank you. You have given me a lot of information so I'm going to ask you a few more questions if you don't mind.

Can we go back to paragraph 18 please. So at 17 and 18 you explain your position as a professor and your position as a consultant and you say at paragraph 18 that you donate your time to your role at the hospital to assist with your work at the university. How is it that your position at the hospital supports the work that you do at the university? How do those two interests come together?

A. That's a decision made by the university that the medical academics in the university should be contributing to the work of the local hospital. Clearly the -- we have skills and resource -- we have skills and time and we can contribute to the NHS care, so that is simply the university saying it's good for us to provide services to our local community.

At the same time my individual research is very much based on the patients I see in front of me, so the questions I have and the grants I write are based on either patients I see in Asia or patients I see in Scotland.

Q. What are your current research interests?

A. So I have a study in Bangladesh, we're trying to recruit

3,300 patients -- we have recruited 940 at the moment -- to a study of how can I stop people dying after organophosphorous insecticide self-poisoning. So this is a type of pesticide and I'm trying to work on one little mechanism in that poisoning picture and I'm trying to stop people dying by giving them a medicine. That's a trial which has been going on with my Bangladeshi colleagues, so quite a lot of my life is spent working on that.

I have a study in Sri Lanka at the moment trying to find out -- people go to pesticide shops to buy a pesticide to then drink it as a way of communicating. Some people die and that's clearly not good news for the pesticide seller, so we're doing a trial of randomising 1,400 pesticide sellers across a region of Sri Lanka with 2.5 million people, trying to find out can we help the pesticide vendors not sell to people who are going to drink it because it's good for their business and it's good for the community. So that's a big public health study that's going on.

Do you really want to know all the stuff I do because I think this could go on for quite a long time?

Q. I think I did say this morning that we hope to complete your evidence in the course of today so I think those two examples give us a flavour of your current research

interest. Is there another --

A. I'll give you one more --

Q. One more.

A. -- which is a flavour because we could go on for quite a long time otherwise. So what we have just got money for is to do a study of flumazenil. So we have many drug deaths in Scotland of people who have taken opioids and benzodiazepines. This huge increase we have seen in Scotland in the last ten years is associated with that combination. We have a lovely antidote, if you can use the right word, of naloxone for opioids which works very well. But people are still dying, so the question is should we be using an antidote for benzodiazepines. One exists, it's called flumazenil, we don't use it because we worry about seizures. So we're trying to do a study going back to the beginning and saying: can we use flumazenil safely? And potentially in five or ten years' time we'll have a second antidote which may stop all these deaths. So my research is also based on Scottish work.

Q. So your research is wide-ranging and involves your patients both in the UK and far, far further afield in Sri Lanka and Bangladesh?

A. And Nigeria and places --

Q. And Nigeria too and other countries.

A. Yes.
Q. We have your CV, as I have mentioned, it's a very lengthy CV and it is there for us to read at our leisure and learn more about your research.
Returning then to paragraph 18 because I have another couple of questions for you about this paragraph. You say when you work at Edinburgh University on a voluntary basis, so on the basis that you are not remunerated for one day a week, you perform ward rounds and treat poisoned patients. What wards are you working on at the university?
A. In the hospital.
Q. Sorry, I beg your pardon.
A. So in the hospital there's an acute medicine unit which has six bays. There is something called bay 6, which is quite famous, we might argue, because this is the toxicology ward. So all the drug overdose patients, all the recreational drug patients get through there and we look after them in that ward. So it is called bay 6 of the acute medical unit in the Royal Infirmary of Edinburgh.
Q. You say you treat poisoned patients. You have told us about the type of poisoning that you typically see in Sri Lanka. What types of poisoning are you called upon to treat in your work at Edinburgh Royal Infirmary?

A. So the one most common would be overdoses of medicines. For example paracetamol, that's a particular problem we have here; antidepressants, things like fluoxetine, sertraline; sometimes some heart drugs like calcium channel blockers. There's a huge number of various medicines and whatever people have available is generally what they take. Sometimes people plan in advance who have a higher intent and more -- it's a more lethal attempt, they will go out and look for various medicines and get them from people or buy them. But most people will grab what is available in the house, so it depends what medicines they are taking.

That is the most common form of poison I would see. The second most common is recreational drugs or -- I'm not sure recreational is the right word, but non-medicinal drugs, for example cocaine, heroin, and then sometimes the medical treatments we use to support people who are addicted to drugs, like methadone would be the classical one.

Q. Let's look at paragraph 19. You took us to it already and you explained a little bit about the National Poisons Information Service. You explained that you and the other clinical consultant toxicologists across the UK, I think you said there were 16 of you?

A. Mm-hm.

Q. So 16 consultants and three professors in the UK. Those of you at the consultant grade between you provide an on call service for the National Poisons Information Service?

A. Yes.

Q. So let me ask you some questions about that. What is the National Poisons Information Service?

A. So in the 1950s there was a realisation that there was a growing problem in the UK and across the world of people taking overdoses of medicines and so there was a realisation there was a need for a speciality to grow up and a place to treat these patients. So Edinburgh had already had a consultant called Henry Matthews who had already realised this was a problem and started writing about it, so about four or five centres across the UK were set up as poison centres and they basically worked as independent centres for many years. And around 2003 the Public Health Protection Agency, which is the old Public Health England organisation, decided to bring everyone together into one organisation, so we became the National Poisons Information Service with the Edinburgh unit or the Newcastle unit or the London unit for example. These were all the units that were put together and we now have a single telephone call so when people telephone -- when a healthcare worker calls that

number they will get through to one of the four centres and we have people who take phone calls and then if it is a particularly problematic patient they will then call a consultant for further advice.

Q. When you are on call are you on call 24/7, 365 days a year or is it on a rota basis with the other consultants?

A. So the service is on call like that. I'm on -- almost uniquely I'm on call 365, 24/7 because of snake bites. So I have a special interest in snake bites, so if someone's pet cobra bites them in Birmingham they call through to the service. The UK has a set of antivenoms for lots of different exotic snakes which people like to keep as pets apparently and then I take a phone call at 3.00 in the morning saying, "What antivenom should we be using for this one?" And there is myself and a colleague in Liverpool, we take the calls, we're on call 24 hours a day, seven days a week et cetera for these particular cases, and there are probably ten a year. Otherwise we share out the rota in a more civilised fashion.

Q. You say at the bottom of paragraph 19 that alongside taking calls you edit TOXBASE. Tell us about TOXBASE?

A. So classically in the past before the mid-1990s everybody did phone calls, so it was all relayed on

phone calls which takes time and is not a very efficient way of getting information and actually limited the number of patients we could advise on. Around the '80s and into the '90s a database was set up which around '98 to 2000 went online. So now the situation is when a patient comes to hospital in the UK, say there's 300,000 poisoned patients a year, in practically every case the doctor who looks after the patient will go online and look up information about how to manage that particular poison by going onto a database. That database is edited by the National Poisons Information Service and let from Edinburgh.

This gives you the basics, so this allows you to care for 95% of patients and then there's a few patients who are more complicated, they get telephone calls. So now we have something like 1.6 million accesses to TOXBASE and only 50,000 phone calls, whereas in the past we would have had 300,000 or 400,000 phone calls. And then of those 50,000 phone calls we get maybe 5,000 go to consultants. So this allows us much more efficiently to provide information across pretty much any poison people will see coming into the emergency department.

Q. Thank you. If we could perhaps now move on to paragraph 20 in which you explain that you are the chair of the Clinical Standards Group which means you direct

the poison centres across the whole of the UK. What do you mean by you direct the poison centres across the whole of the UK?

A. So these were independent units and they have their own directors, so between 2012 and 2016 I was a director of the Edinburgh unit of the National Poisons Information Service. I then demitted from that and a colleague of mine took over. We have a chair who brings together the four units and is the face of the four units to the rest of the world and the rest of the UK so currently I'm in that position since April last year when my colleague Professor Simon Thomas retired.

Q. You also say:

"I'm now the senior clinical toxicologist in the UK."

Should we understand that to mean you are the most senior clinical toxicologist in the UK?

A. Yes, in -- as a result of that position and being one of the few professors of clinical toxicology.

Q. Thank you. If we could perhaps return briefly to the series of letters after your name, if I can ask for us to have paragraph 8 on the screen. Here you explain that you have a fellowship of the Royal College of Physicians in Edinburgh. You say that you secured that position a year after becoming a consultant, that would

have been in around 2010?

A. Yes.

Q. At paragraph 9 you explain that you also have a fellowship of the British Pharmacological Society and that the fellowship recognises that you are a clinical pharmacologist and that you do good quality work in clinical pharmacology and you received that in 2014 and have been awarded prizes from the society.

At paragraph 10 you explain that you have a fellowship of the European Association of Poison Centres and Clinical Toxicologists, which is the specialist European organisation that you belong to and it brings together all the poison centres and clinical toxicologists on an annual basis to a congress and you tell us that you were one of the first to be awarded the inaugural fellowship and you were probably one of the youngest.

If we could, sorry, jump back up to paragraph 6. You explain that you also have a Doctor of Science. You explain that you wrote 75 papers about poisoned patients published in major journals, put together a thesis and submitted to Cambridge University, and that's a qualification you received in 2014 and it recognises that you are a specialist in your area and that your research is of high quality.

75 papers about poisoned patients; were they based on the patients that you see in the UK or Asia? Asia?

A. So I have about 340 papers now. This was a 75 -- a block of 75 papers around a particular area which was about -- I think practically all were about Sri Lanka.

Q. Do you continue to travel to Sri Lanka and other overseas countries?

A. Not very much. I was in Bangladesh and Sri Lanka in November last year and I was in Nepal briefly in April, so I probably travel four or five times a year. Having lived there for a long time I have the connections which allow me to be more peripheral now.

Q. Thank you. I want to ask you a few questions about your previous experience of giving evidence. Have you been asked to give evidence before?

A. About three or four times.

Q. And have you advised on cases over the years in anticipation of giving evidence and perhaps that hasn't been required?

A. I have probably written maybe -- not very many. I'm a full-time academic so I don't do many reports but maybe 50 or 60 over the last -- this is one of my first ones in 2017. I have been doing it since about 2014/2015. Two or three a year originally, one or two a year, and now more like ten a year.

Q. So these are medical legal reports?
A. Yes.
Q. And are they in civil cases or criminal cases?
A. So the times I have been to court have been PFs, twice. Here and once in -- a coroner one in Manchester. I've never been to court yet for a civil one or a criminal.
Q. And who do you tend to be instructed by? Which side of the case?
A. Both sides.
Q. Both sides. And have you ever before been involved in a case involving a death in custody?
A. Yes. So what -- it depends what you mean by custody precisely. So one was a very similar situation, someone who died near Aberdeen who was restrained by the police and had been exposed to cocaine. And a second one -- again, a similar one, I can't remember where it was now, who had tried to be arrested by the police, took cocaine and then was transported to the police station and in the police station he tried from cocaine toxicity.
Q. Through your medical legal work do you have an understanding of the importance of being independent?
A. Yes.
Q. And of being objective and unbiased in your views?
A. As far as I can be, yes.
Q. And do you understand that your role here today is to

assist the Chair to the Inquiry in matters within your expertise as a clinical toxicologist?

A. I do understand that.

Q. Can I ask you now to look at the letter of instruction from Crown Office and we will bring that up on the screen. It is COPFS 02360. It is dated 26 April 2017 and if we could scroll down please. So there's a little background about the case and if we keep scrolling down please. If we stop there we see, some background information having been provided, the Crown say:

"Given your experience, the Crown wish to instruct you to provide a general opinion on the individual and any 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 two drugs may have had on the deceased's ..."

That of course is Sheku Bayoh:

"... mood, cognitive ability and behaviour."

So that was the -- that one paragraph there contains the question that was posed by the Crown. Were you asked at all in that letter or subsequently by the Crown Office to consider the effects of these drugs on the heart?

A. No, I don't believe I was. I believe the PF who wrote me the letter passed away some time after that. That

may be the wrong thing but I think there was a change of staff because it went quiet for a long time and -- maybe that's wrong but I think that's what I remember.

Q. All right. Let's scroll down to the bottom where I think there should be a list of the documents that were made available to you. So there's quite a long list but if we can read it short we see that there are some statements from witnesses, that's civilians and from police officers, medical notes, a briefing paper, post mortem report, toxicology report, neuropathology, medical notes again, a use of force standard operating procedure and a number of expert reports prepared by others in connection with the case.

So those various documents were sent to you under cover of the letter of instruction and in response you prepared your report and we will perhaps bring your report up now, it is COPFS 00038.

Now, I want to skip through this quite quickly at this point. We see your report is dated 2 June. I want to just familiarise ourselves with the structure of your reports so if we can just scroll through it fairly slowly please. There is a preamble in which at the first paragraph -- sorry -- you say you are aware of the role of an expert witness and of your need to deal with matters within your expertise and provide a fair and

accurate opinion with respect to the case.

There's a little bit of information there about your background and your qualifications. If we could scroll down please. The second chapter of your report is the instructions and again you repeat the list of documentation that was provided to you.

The third chapter is the query and you have copied over the question that we looked at a moment ago from the Crown's letter of instruction.

There's then a section headed, "Background", which appears to be a summary of the information that seemed to you to be pertinent gleaned from the documents that were sent to you?

A. Yes.

Q. And if we move beyond "Background", there's a summary of the toxicology results that were contained within your papers. You then record a psychiatric diagnosis provided by Dr Lipsedge retrospectively. You offer a view on that.

There's then a section on the effects of two drugs that we know were in Mr Bayoh's system, alpha-PVP and MDMA, and if we can scroll through that for now, there's the role of these drugs in causing his drug-induced psychosis and then an addendum. At this point I would like to ask you some questions about the addendum. We

will look at the other parts of your report shortly.

So at paragraph 16 under the heading:

"Addendum - Police Scotland. Use of Force Standing Operating Procedure ..."

You say:

"I would like to make an additional comment on the [Police Scotland use of force] SOP which may be relevant to this case. I was provided with this document as background reading for my opinion."

Now, the addendum is fairly lengthy and I don't think there's any need for us to look at it in detail but do you go on to note that the standard operating procedure provides useful advice on how to deal with patients suffering from mental health disorders including psychosis and that that advice is consistent with standard clinical practice for the management of psychotic patients?

A. Yes.

Q. That's at paragraph 17. If we scroll down to paragraph 18, again reading it short, do you note that drug-induced psychosis is dealt with separately in the SOP and that the guidance offered to police officers dealing with patients with mental health issues is not repeated in the context of dealing with patients with drug-induced psychosis.

And you make the observation that:

"This is unfortunate since the principles are the same."

A. Yes.

Q. In the paragraphs that follow you set out how patients with acute psychosis are managed in emergency departments, you offer a view as to how Sheku Bayoh might have been handled differently by the police and you conclude, in paragraph 24, by recommending a discussion between police and emergency medicine physicians to increase understanding of how to manage these patients and to refine the current guidance?

A. Yes.

Q. Would that be a fair summary of the addendum to your report?

A. Yes.

Q. Now, the SOP, the standard -- standing operating procedure, you were sent that by the Crown?

A. Yes.

Q. Was there any explanation given to you as to why they were sending it to you?

A. No, it was provided as background reading for me to make a decision.

Q. Were you specifically asked to comment on it?

A. No.

Q. Were you given any instruction at all as to what you were supposed to do with it?
A. No.
Q. And what did you take from the fact that it had been sent to you at all?
A. I think it -- this was information about the situation and the procedures the police followed at the time of Mr Bayoh's death.
Q. And what was your reason for offering a comment or a commentary on what is said in the standard operating procedure?
A. My clinical practice in Edinburgh involves admitting many patients, in tens or hundreds over the years, of patients with psychosis where it is absolutely unclear whether this is due to primary mental illness or due to drugs or to a combination. So these patients are kept in our ward until it becomes clear whether it is drug induced, in which case it should fade away over a day or two, or whether it is mental health where it may not fade away and they may need admitting to a psychiatric hospital. They spend time on our wards, we try to understand what's going on. It's actually not possible, at least in the early stages, to differentiate at all, so the idea that there should be two ways of treating patients, one with a mental health primary cause and one

with a drug induced primary cause, doesn't fit with clinical practice.

Q. I see. And in your statement, if we could perhaps return to that, at paragraph 33, in paragraph 33 you explain that this was not an expert opinion and your view was based on your day-to-day clinical experience on your ward, which is what you have explained to us in your evidence today.

A. Yes.

Q. Professor, just for the avoidance of doubt, were you seeking to offer a view as an expert on use of force?

A. I'm not an expert on use of force. I'm a clinician who has to at times use force to safely care for a patient.

Q. And the comments that you make in your addendum you have told us were based on your clinical experience as a practising clinician and toxicologist who does ward rounds?

A. Yes.

Q. I would like to move on to ask you some questions about the drugs that we know were found in Sheku Bayoh's system, firstly alpha-PVP and if we could perhaps move to paragraph 63 of your statement. Much of your report has been copied into your statement, Professor, so to avoid us jumping between documents I will work largely from your statement going forwards. You referred to

a paragraph in your report where you said that:

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

And in the paragraphs that follow you explain the terminology, but perhaps in your evidence this morning I could ask you just to say a little bit more about alpha-PVP and what is meant by a synthetic cathinone stimulant drug?

A. So crudely the way I think about it there are drugs which are uppers and drugs which are downers, so stimulants are clearly drugs which are uppers, give you energy, give you various properties of being overstimulated. Again about the dose, if you get the dose right you can become charming and, you know, people get more confident. If the dose goes higher you get many of the more physiological complications of using these drugs.

So if we look at the words, stimulant, it's a type of drug which is going to give you that effect similar to cocaine or amphetamines. Cathinone is a particular structural class in itself, it's not particularly interesting but a group of drugs have this same structure and there may be some similarities across the drugs with that similarity. But actually what happens

when you tweak a little -- there are hundreds if not thousands of drugs like this now and as the chemists have put together new drugs they have put new little chemical structures in various places, that can completely change the way these drugs behave in human bodies. So actually it's -- the structure is not so interesting because you can't really tell how this drug is going to behave from the structure; until it goes into humans you don't really know. But the cathinone is about that kind of structure. It is synthetic, it is made in a laboratory, it has not come from a plant. Relatively new, it's not a classical drug like amphetamine and methamphetamine which have been around for a long time, alpha-PVP seems to have been something created in the last 20, 25 years.

Q. You have mentioned it being a stimulant and if the dose is right it may bring about effects that the user might find to be pleasant. Why would someone take alpha-PVP? Can you tell us more about that. What are the desired effects from taking this drug?

A. I don't really know about why someone would choose alpha-PVP. In the UK it doesn't seem people use it very much. There was a time it was used widely in the Russian town where that study comes from. I don't know why people might choose alpha-PVP in particular.

I don't think it has any property which is particularly different to the other ones.

Q. To your knowledge does it have a street name?

A. It might do but they change all the time.

Q. And to your knowledge is it ever used to cut other drugs?

A. I have not heard of that happening.

Q. So the Chair has before him a statement which is part of the evidence in the case from a man called James Hume who is a substance misuse harm reduction worker from the Fife area, who back in 2015 gave a statement saying that the local drug takers had little or no knowledge of alpha-PVP and that he was aware that it was getting mixed with other controlled drugs, including MDMA and coke, to bulk it up to make bigger profit margins, so the dealers were making a profit and the abusers didn't realise what they were taking and how serious the effects could be. Does that come as a surprise to you?

A. Well, it -- that would suggest to me that alpha-PVP was cheaper to make than the other drugs. You either -- either cheaper to make or cheaper to obtain or somehow -- you wouldn't bulk up cocaine with something that's more expensive than cocaine, so therefore it suggests that somehow the supply of alpha-PVP was allowing the people providing drugs to mix it with drugs

which -- more expensive drugs and therefore -- it has a similar effect to cocaine and perhaps cocaine was more expensive at that time. That would be the only reason I could think of for bulking things out, generally using cheaper things.

Q. I would like to read to you a very short extract from a report prepared by -- sorry, a statement given by another expert who said:

"There are 11 different forms of alpha-PVP. The laboratory did not test to distinguish which form it was. Why is that important? Well, because some types of alpha-PVP are more than 1,500 times more powerful than others."

You haven't heard that before.

A. No.

Q. That comes from a statement of a Steven Karch, but can I invite your comment on that?

A. So the chemical structures have various -- we call them isomers, so they have the same chemicals in there but each time there is a link, it might go off in a left direction or a right direction. So therefore when you have a different -- you have a compound, with multiple different things joining in, there may be different variations of it because of the direction, which way the compounds come off.

We have this in medicines as well. There are some medicines where there's a left and right form and the left form is the most potent so now we've got rid of the right form for medicines and we only have the medicines purely made for the left form. For example escitalopram would be an example where we've got -- we don't -- we still use citalopram but some people like escitalopram which is the one -- I can't remember if it's the right or the left. So this is quite well-known in medicine. To have 11 different forms suggests it is quite a complex structure to have these different angles going off. Yes, I could imagine they could be more potent in their interactions. I'm not aware of it, but the principle seems reasonable.

Q. Thank you. In your report -- in your statement, if we can move down to paragraph 78, you talk about what you call the side effects of alpha-PVP and you reference the Swedish STRIDA project which lists the common side effects of alpha-PVP and you say that tachycardia is the most common side effect and you explain that 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 you 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."

We have heard evidence recently in this Inquiry about arrhythmias and I wonder if you can help us to understand the difference between tachycardia and an arrhythmia?

A. So tachycardia has a rhythm, so arrhythmia or dysrhythmia just means a chaotic rhythm or an absence of a rhythm. With tachycardia you still have the rhythm. So your heart has two chambers -- two double chambers and what you want is the first one to push blood from the atrium to the ventricle and then the ventricle to go out and that takes time and the faster you get, the more difficult that process is and if it is chaotic they're not beating in order, in rhythm. By going in rhythm, the heart works well. So having a heart rate of 120, 140 I could probably sit there in front of you and function completely normally.

When you get to 180, that's a normal rhythm but it's very fast and so the heart is not really having time to fill properly in-between beats. Where it is chaotic or a dysrhythmia you don't get this rhythm so the

electricity of the heart has changed, the rhythm is not there and the heart doesn't work and the blood doesn't come out of it very well.

If we talk about something called ventricular fibrillation which is about -- it's a chaotic heart rhythm. There is no effective function going on at all. The electricity through your heart is all over the place. We need the electricity in the heart to be nice and rhythmic to cause the heart to work well and the blood to flow through it properly.

Q. Thank you. At paragraph 79 you explain that there is relatively little data on the cause of death in patients dying after exposure to alpha-PVP only, that means I think not in combination with other drugs, and that it is not clear whether alpha-PVP commonly causes very fast or chaotic heart rhythms causing cardiac arrests.

A. Yes, that's true, but that's partly because we don't have a lot of data on alpha-PVP. The best case series is from the Russian city, but also people are tending to -- if you die in a hospital on a monitor, we know what's going on. When you're dying in the community as a result of an interaction, or as a result of the drug itself, we have no way of looking at what's going on in the body, so it's very hard for us to tell what's truly happened.

The pathologists will try and make a guess, an estimate, or use their skills to try to say what happened before the death, but it is quite difficult when people are dying in the community.

Q. You say very little is known for the reasons you have explained, but you referred to a Russian study. Could you tell us just a little about that?

A. It would be lovely if you could show me the paragraph it was in, but this was a -- there was a city called Yekaterinburg which is a city in Russia where they had a drug problem and that drug problem on analysis turned out to be alpha-PVP. This is by far the biggest case series I have seen anywhere in the world where this particular drug became a problem, which simply meant a supplier started supplying that drug and there was a study done of these patients and they looked at patients who had only -- they managed to get blood samples because they came to hospital and they found that some patients only had alpha-PVP and others had alpha-PVP plus MDMA and other drugs and they were able to look and find out things like psychosis or -- and particularly they were looking at psychosis: what was the frequency of psychosis in patients just with alpha-PVP in their blood, versus patients who had alpha-PVP and other drugs and this was a -- it was

presented in -- I don't think it's been published. A colleague of mine was involved with the Russian toxicologists and they published -- they presented it at a meeting and I had the copy of those slides.

Q. I think you referenced this at paragraph 77.

A. Thank you.

Q. This is your colleague David Wood?

A. That's right.

Q. And it is the Yekaterinburg study?

A. You pronounce it better than I do, thank you.

Q. And you explain in this paragraph that the point you are trying to make in your report is that:

"... patients who have 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 the study and the presence and absence of other drugs did not affect the incidents of psychosis."

A. Yes.

Q. So what then would you take from that study, or invite us to take from that study?

A. That alpha-PVP is a -- has a particular risk of causing psychosis, more than most of the other drugs we recognise and a really remarkably high rate of psychosis.

Q. If we can scroll down a little bit as far as

paragraph 80 please. You mention a cluster of related symptoms. We have spoken about the tachycardia, which you say is the most common side effect. At paragraph 80 you explain that:

"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."

You go on at paragraph 81 to explain that:

"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's going on. You can't respond to stimuli and often have delusional thinking. You're thinking everyone is attacking you when they're not. With hallucinations you're getting strange visions and sensations because the brain synapses are being overstimulated."

Continuing on to paragraph 82, you talk about intense paranoia also being a side effect of alpha-PVP and again you say:

"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 will help you escape, but it becomes too much in these cases. You're overstimulated and because you're paranoid you're quite often violent and aggressive so you see the world attacking you. You are in flight or fight 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."

For completeness, can we look briefly at paragraph 119 of your statement where you say -- sorry, I think I've got the wrong paragraph number here, but you will correct me if I am wrong. I think you do say in your statement that you know that alpha-PVP causes a very high rate of agitation and psychosis.

A. Yes.

Q. Which is effectively what you said earlier and what should be taken from the Russian study by your colleague David Wood.

A. Yes, and also the point that the presence of other drugs doesn't seem to increase or reduce it particularly.

Q. Thank you. We heard evidence last week from Dr Maurice Lipsedge and I think in fact you were provided with

a copy of his report by the Crown and you had it available to you when you prepared your report for the Crown Office. He is of course a psychiatrist, but he treats patients who are suffering from psychostimulant intoxication and he was asked about alpha-PVP. What I would like to do is just tell you what he had to say about alpha-PVP and then invite your comment.

A. Okay.

Q. So he said that alpha-PVP was a very powerful stimulant, "like a very, very potent form of amphetamine". Amphetamine "has the potential to cause extreme paranoia fairly quickly", within 20 minutes, 45 minutes:

"It acts quickly. It doesn't do that to everybody but in a person who happens to be susceptible then there can be a paranoid reaction which can be extreme ..."

Would you agree or disagree with that evidence?

A. I would agree. I think the interesting question is is there a dose effect, which I don't think we know, so if we take the average person and put the dose higher and higher in that person will they all become paranoid at the end, or is it true that what he is saying that only some people are more likely to get paranoid and we don't really know. It's quite hard to distinguish the two. I think they are both -- I think it's -- if you have

had -- if you have been paranoid before, then you're at higher risk of becoming paranoid with something else, so someone with a history of paranoia who takes a drug that causes paranoia is probably -- again the word is "probably" -- more likely to get paranoid.

But it is quite possible that there is a dose response as well and as you get so supremely stimulated you go through agitation, up to levels of paranoia and delusions.

Q. And what difference, if any, does it make if an individual is a naive user or a more chronic user?

A. I'm not aware -- I can't answer that question.

Q. And can you say whether over time a user of alpha-PVP would likely build up a tolerance or could the reverse be true, could they become sensitised to the drug?

A. I don't think we have the data to answer that.

Q. Okay.

A. I mean both are perfectly reasonable possibilities.

Q. Again I would like to tell you what Dr Lipsedge said in relation to sensitisation to alpha-PVP and simply invite your comment.

A. Okay.

Q. He explained that sensitisation is the opposite to habituation. He gave an analogy with alcohol: a person might become more tolerant to the effects of alcohol,

able to drink increasing amounts with less undesirable effects. He said that sensitisation is the opposite to that so with successive exposure to alpha-PVP, the brain reacts increasingly in a negative way, with increasing risk of the intoxication and an increasing risk of paranoia, suspicion and violence.

A. Okay.

Q. So I would simply invite your comment. I wondered whether you would agree or disagree with what Dr Lipsedge said in his evidence, albeit he is coming from the perspective of a psychiatrist and not a toxicologist.

A. Yes, so I come from the perspective of acute exposures generally. I'm not dealing with people who are taking a routine dosage or a daily dose, etc. I'm dealing with when they come to a crisis and they end up in hospital, or dealing with the ambulance services in the community, so I wouldn't have the knowledge to be able to comment on what he said. It sounds fine.

Q. Are you able to assist us, on the basis of your clinical experience, as to how long it would take a user to feel the effects of alpha-PVP?

A. I mean -- no, because I mean people are taking so many mixed drugs when they come in and also from my own personal clinical experience I've never known if I've

treated anyone with alpha-PVP because we are not analysing their blood to know which of the many drugs out there they are taking, so I don't know if this is an alpha-PVP patient.

Occasionally people come in and say "I've taken MDMA", and you're a bit sceptical because they could have taken so many things. No one has ever said to me "I have taken alpha-PVP" and I don't think the literature I have seen is clear enough about how long it would take, but clearly 15, 20, 30 minutes is a timeframe which is reasonable for many of these drugs.

Q. And the reason that you are unlikely to know what drug is in a person's system I think -- and we will discuss this in more detail later -- but it is because, as you explain in your statement, you treat patients symptomatically because it takes weeks to get the results of blood tests back.

A. Yes, exactly, and also they have taken many drugs. We do a study at the moment where we -- six weeks later we get the results back and many have taken five, six, seven or eight drugs, so they're dealing with a mixture of what they have taken.

Q. Will all users of alpha-PVP experience the negative side effects?

A. I don't know.

Q. Are you able to assist us with how long it takes for the effects of alpha-PVP to wear off?
A. No, I don't -- again, alpha-PVP I don't know specifically enough to -- it varies by people, it varies by dose, it probably varies by how much you're using over time. It's very variable by person.
Q. If you happened to know, perhaps because the patient told you, that you were treating a patient intoxicated with alpha-PVP, what would be your first line treatment?
A. Well, first of all we would have to work out what level of illness they are. People can come into hospital and they're quite well, but most people who come to hospital will be agitated and distressed and stimulated by the drug and the first line treatment would always be a benzodiazepine. I would typically use diazepam, others might use lorazepam or midazolam. These are drugs which overstimulate a receptacle, the GABAA receptor, and cause the brain to calm down and that would be our first line drug to try to get on top of the situation.

Hopefully we give tablets, depends if they are willing to take tablets and again it's about talking to the patient, trying to persuade them to take it, otherwise you might give it through the arm if they were really particularly agitated -- through the vein, sorry,

in the arm.

Q. And again later this morning I'm going to ask you in some detail about the clinical management of patients who have taken stimulant drugs, but for now if we can stay with the effects of alpha-PVP. You were asked in your statement if you could comment on the fatal range and if we can perhaps bring up paragraph 84. You say that:

"There is no precise range identified for fatal cases ..."

And you refer to two studies and reading those studies together, the range appears to be from 1.1 microgram per litre of blood to more than 20,000 micrograms per litre of blood?

A. Yes.

Q. That seems, to me as a lay person, to be a very wide range.

A. Absolutely.

Q. As you are aware -- and this is referred to, of course, in your Inquiry statement, that the level of alpha-PVP within Sheku Bayoh's blood was 70 --

A. Yes.

Q. -- which you observe, at paragraph 88, falls within the reported range of fatal concentrations, albeit at the lower end of the range.

A. Yes.
Q. And you say at paragraph 88 please:
" ... I would have said that Mr Bayoh was on the lower range of these case examples, but still very consistent."
By consistent do you mean still within the range --
A. Yes.
Q. -- in terms of the reported cases.
A. Can I say one thing here?
Q. Yes.
A. So if we look -- there are many drugs which have been around for a long time and many of them have been found in patients who have died and patients who have not died. For example, police pull people over by the side of the road, they take drug samples. So over time -- and there are a couple papers which list a thousand different drugs, medicines and drugs, looking at fatal -- they look at dosage you might find therapeutic, the dose you might find people being ill and the dose of death, so we have -- so when I say in alpha-PVP we don't have those ranges, they exist for many other drugs but still they are very broad. They are not very different to this. Even though it looks strange as we see, it's not different to anything else. And it comes back again to the fact that if I take a medicine, it has been

trialed in humans. You know, the first time this medicine comes out it goes into a human, we watch it really carefully, we measure how it gets handled in the body, whether it comes in your urine, or whether your liver breaks it down, we have that information.

Once we have done it once -- so the first doses are really carefully done -- we then increase the dose and you might go through six or seven different doses, but each time we're taking blood concentrations from the people - nearly always young men, nearly always men because of reproductive -- in women -- we wouldn't expose a woman to a new medicine routinely because of the risk of problems with an unborn child. So -- but then we may have eight people and we've got eight doses and by the end of that we've got lovely information about what is a dose which has an effect, what's toxic. But these drugs go into humans without any of that process. We have no idea what concentrations are working, so when people are at home taking these drugs and not coming to hospital, I don't know what the concentrations are in the blood. I don't know what's a good concentration, what's a bad concentration.

All we have in these people is if they have a problem, they come to hospital. If they come to hospital alive or they get -- the police find them and

take a blood sample because of driving, then we'll have some ballpark for that, and then we have the concentrations from the deaths.

Now, again they will often have multiple drugs, like Mr Bayoh had multiple drugs, but I think this is the most important one. The range is we have sometimes it is just one drug, sometimes it is two or three drugs. Sometimes the two or three drugs are important, sometimes they are not. Sometimes the blood samples are taken in a post mortem three days after they have died where things could have happened to those concentrations in the meantime. Sometimes they're taken in hospital two days before they die. It's kind of chaotic.

We're trying to get information which makes sense and kind of comes together to give us some information to say "Well, it looks like it's probably about right", but that's the only level of information we can offer because they have never been put into humans in any controlled fashion at all. Sorry to interrupt.

Q. No, not at all, that's very helpful. Thank you.

In paragraph 88 of your statement you say that on the basis of the studies and taking into consideration the limited research and writing around the subject, which you have just explained to us, Mr Bayoh's blood concentration of 70 micrograms per litre of blood is not

inconsistent with a fatal dose and then you conclude that paragraph by saying:

"That being said, the surrounding circumstances of the death should not be ignored."

What did you mean by that final sentence?

A. So Mr Bayoh was restrained at the time and was very agitated and was -- had no understanding of what was going on, I think it appeared and Dr Lipsedge's comment was that he was psychotic at the time. So the blood concentration is related -- so the fatal dose is related to how the person died effectively. Sometimes we have people who are left alone in a room and no one touches them and they're found dead in the room and those concentrations might tell us that that drug range by itself with that person just sitting there calmly, like I'm doing now, is enough to kill you, or it might say that person was very, very sensitive and for some reason we don't understand yet that drug interacted badly with that person, but in that situation that blood concentration is saying this is a lethal dose for that person in a calm situation.

Mr Bayoh was not in a calm situation. He was also restrained. It's possible because of the restraint he died at a lower dose than would have been expected if he hadn't been restrained and had survived or had died for

1 another reason.
2 Q. Thank you. I want to move on to ask you some questions
3 now about MDMA and again if we could perhaps pull up
4 paragraph 95 of your report. Again, we all have your
5 statement and can read it at our leisure, but from
6 paragraphs 95 to 100 you explain the type of drug that
7 MDMA is and the various effects that it can have. You
8 say at paragraph 95 -- and again it is a copy from your
9 report:
10 "MDMA is a widely used entactogenic phenethylamine
11 drug. It causes hyperstimulation of the central and
12 autonomic nervous systems via increased release and
13 reduced uptake of serotonin, as well as dopamine and
14 norepinephrine."
15 A. Norepinephrine.
16 Q. Thank you. There are lots of quite technical words in
17 there. Can you perhaps introduce us to MDMA and tell us
18 a little bit about the drug.
19 A. So entactogenic is the word below, it means "touching
20 within", and this idea that some drugs such as MDMA,
21 which work predominantly on the serotonin system in our
22 brain, causes emotional openness, affection, empathy at
23 the right dose. Again it is always about the dose and
24 the dose varies by people.
25 It's a phenethylamine, again it's a structure. It

happens to come from that kind of structure and it has been changed to create this particular type of drug.

So the way to think about them -- in our brains we have lots of synapses and lots of neurotransmitters and some key ones are serotonin, dopamine and norepinephrine. Norepinephrine is the fight or flight one in particular, so that's really stimulating your norepinephrine mostly more than your dopamine.

Dopamine is about reward systems and serotonin is very key for depression and for your mood. So when we have -- this is a nerve that's trying to send a message from one nerve to either the muscle or the nerve at the other side and so they're very, very close together and the area they meet we call the synapse and what happens is this nerve gets stimulated, an electric current comes along and it releases a neurotransmitter which goes across to the other side.

Now, you want in some way to stop that effect. You don't want that neurotransmitter to be released and then work for the next three weeks. It's going to have to work in a very short time, probably milliseconds, so as a result of that there are mechanisms in this synapse that is the junction between the -- we call this the pre-terminal nerve ending and then the post-synaptic nerve ending here. There are mechanisms in place to

bring that to an end quite quickly.

The typical ways -- so one thing to -- when the neurotransmitter comes out, it binds to receptors on the other side. So the signal comes electric, it causes the nerve to release the neurotransmitter, that goes across a very, very small passageway, binds to the post-synaptic receptor and then has that effect, an electric current will go on, so that's how they join to each other and how the brain talks to itself.

So what happens with drugs like MDMA, it affects this mechanism for ending it and causes it to keep going. So there's two key ones, one of which is -- the main one is about a transporter which takes it back up into the pre-terminal synapse so it has gone out, the neurotransmitter has gone out -- we will call it -- let's call it serotonin. Serotonin comes out, binds this receptor then comes off and you get rid of it by taking it back up. So fluxetine or many anti-depressants block that mechanism of getting rid of it, so therefore you have a longer effect of serotonin.

What happens is some of these drugs actually increases -- every time something happens there you have a certain amount comes out. You take some drugs, it causes it to come out without the nerve happening and also a larger amount will come out, so you get

overstimulation of the receptors at the other side so you overstimulate because you have got more of this neurotransmitter present because more is being released and less is being taken back up and this is how these drugs generally work.

Serotonin -- MDMA particularly works at the serotonin synapse, which is why we find the effects of it are more about mood. Other drugs work more at the norepinephrine, so cocaine for example I think is more dopamine and norepinephrine. Each of these drugs have slightly different patterns of where in the brain and which particular synapses they're going to work on, but the general idea is they're causing overstimulation by either preventing the uptake or increasing the release. That's how these work.

Q. And what are the consequences of that?

A. It would depend on the drug, depend on the pattern of the synapses, so in MDMA at a normal -- at a dose that people find beneficial you have this affection, emotional openness, enhanced sociability. It was the kind of clubbers' drug because suddenly everybody found you charming and exciting and a really nice person to hang out with. That was what MDMA was used for.

But there are problems with MDMA, there's something called sort of perseveration, so people -- they are told

to drink water because they get hot in a club and so they keep drinking, and they keep drinking, and they keep drinking because their brain tells them to keep doing that, they're not thinking what they're doing and they can actually die from drinking too much water and that's quite well recognised, that you have seizures from that.

These drugs are often not pure. Sometimes their dose is much higher. I think in the past 25 to 50 milligrams was a standard tablet. It suddenly went up to 250 so people were having consequences of these higher doses. Higher doses you find toxicity which predominantly means the temperature goes up, so that what we worry about when a patient comes to hospital we will measure their temperature and if it something like 39, 40, 41 we immediately start trying to bring the temperature down because the high temperature, which is caused by this overstimulation, particularly of the nerve to the muscle junction, not the nerve-nerve but the nerve-muscle, your muscles become really overstimulated and particularly if you have been dancing all night you're just -- your muscles are creating too much heat and if your body can't get rid of it you will die from being too hot. As a result of that your clotting stops working, your liver stops working.

So the predominant problem of this overstimulated effect is too much temperature, you're too hot, so we will put you in an ice bucket and cool you down. But that is what we will do in -- for example in cocaine -- in New York they have something called the Love Parade and everyone takes cocaine on it apparently and it's a hot -- you know, 35 degrees centigrade in New York in August, it's not a great time to take stimulant drugs. They will come into hospital. Immediately they will be sedated with the benzodiazepine drug we talked about before and there's a metal bath, a classic old metal tub, you get put in there, covered with ice, covered with water and your temperature comes down really quickly and you survive. If your temperature goes up to 41 or higher there's a 50% chance of dying, so yes.

Q. Thank you for that detailed explanation. If we can perhaps turn to paragraph 106 of your statement.

A. Is this the level of information you want? You should guide me more.

Q. Rest assured that either I or the Chair will soon interrupt if it is not the level of information that is found to be helpful.

A. Right.

Q. At paragraph 106 you explain that:

"MDMA commonly causes tachycardia and high blood pressure ... [and] cardiomyopathy can occur with chronic use ... Cardiac arrests do occur but rarely - probably due to restriction of arteries supplying the heart with blood causing myocardial ischaemia or infarction."

Then you say at paragraph 107:

"MDMA use is not associated with direct lung damage or with respiratory arrest."

And in fact I think -- and we don't need to go to it, but at paragraph 83 of your report you also say in relation to alpha-PVP that there are no reports that allow a conclusion to be drawn that alpha-PVP could cause damage to the respiratory system.

A. That's correct.

Q. So neither of these drugs appear to have a direct effect, as far as you are concerned as a toxicologist, on the lungs or the respiratory system.

Now, again I would like to read to you another -- really short, another small passage from Dr Lipsedge's evidence about MDMA just for your comment because you both treat patients suffering from MDMA intoxication but from slightly different perspectives.

Dr Lipsedge told us:

"Normally Ecstasy or MDMA is a stimulant that is not generally associated with severely adverse psychiatric

effects. There are some reports of it causing paranoia but that's unusual. The more general impact in terms of the way an individual who has taken Ecstasy would relate to others is actually they may feel better disposed than usual to other people. It is a drug that's usually taken in nightclubs and people take it partly because it is actually thought to generate feelings of non-aggression and friendliness rather than paranoia. However, there are some reports, but this is rare, of it having the opposite effect."

Would you agree or disagree with Dr Lipsedge's evidence?

A. Yes, I agree. It depends on your definition of rareness. So the large case series from Euronet(?), they reckoned that psychosis was about 4%. We have talked about alpha-PVP being 65%, in other drugs in that case series it was about 35%. The best data we have on MDMA is that it is uncommon. I wouldn't -- it seems not to be quite as rare as that, but it's very much the lower end.

Q. Are you able to comment on how long it takes or how long it would take a user to feel the effects of MDMA?

A. Not based on my clinical practice because I see them at the other end. If I go onto EREVID(?) and look at the websites there, you're talking half an hour would be

a time -- it's also about the level. You may start getting earlier effects and then it takes longer to become fully effective. Half an hour to an hour would be quite common for a drug to be absorbed and have its full effect.

Q. Will all users experience the more negative effects of MDMA?

A. I don't know. No, I don't know.

Q. Does it make a difference if a user has taken a small dose or a larger dose?

A. I think so because when the tablets started becoming 250 rather than 50 there seemed to be more patients coming to hospital. So I think there's clearly -- everything is always the dose, so it seems the higher the dose the more likely you are to get problems but that may be differences in sensitivity. Perhaps we have different receptors which bind -- or different transporters which the MDMA may interact with differently, we don't know.

Q. And would it make a difference if a user was a naive user or a chronic user?

A. That's the kind of question Dr Lipsedge is more likely to be able to answer because I don't see patients -- I'm dealing with the acute crisis rather than their long-term use of the drug.

Q. With MDMA would you expect, from your perspective as

a toxicologist, that over time a user would build up tolerance or conversely might they become sensitised to the drug?

A. I don't know.

Q. You don't know. What would be your first line of treatment if you were presented with someone who you knew was intoxicated as a result of MDMA?

A. Again, it would be about looking at what their clinical condition was. If they were agitated, which is the likely reason why they would come to hospital, because most wouldn't come to hospital, we would use benzodiazepines. If their temperature was going up and we were extremely worried, again we would sedate them with benzodiazepines and then do things to cool them down. We might use ice, we might use some medicines which keep the temperature down.

Q. Is there a known fatal range for MDMA?

A. There are records where those ranges are put into the literature. I don't have it on me at the moment. I have it in my laptop, but again they are very broad. You wouldn't say confidently this was definitely fatal and this was definitely not a fatal. They will overlap.

Q. And again, is it very variable and person-specific in the way that the response to alpha-PVP is variable and person-specific?

A. It seems to be. I mean we have people who take a single tablet and die and it's not quite clear why that happens, whether it's what they took, or whether it's their body's response to what they took. I think the body's response is definitely a key part of that because when we look in their blood afterwards and find what were the drugs they have taken sometimes it is only MDMA and sometimes they have died and it's not clear why.

Q. I would like to read to you a very short extract from the evidence of Dr Shearer from whom we heard last week. Dr Shearer of course was the pathologist who performed the autopsy on the body of Sheku Bayoh and in relation to alpha-PVP and MDMA she said:

"They are both stimulant drugs and by stimulant drugs I mean they can have an effect on the cardiovascular system, so they can cause an increase in heart rate and an increase in blood pressure and they can also cause arrhythmias. With any stimulant drugs that is one of the extreme complications."

So again, can I simply invite your comment. It would be helpful to know whether you would agree or disagree from your perspective as a clinical toxicologist?

A. So they're both stimulants, so they both can cause them but I think it would be much more common -- well, we

don't see patients presenting with fast heart rates commonly with MDMA but perhaps they're not coming to hospital. We can only see who comes to hospital. So, while MDMA is a stimulant, compared to cocaine, for example, it seems to be far less toxic, so it's much less common. I don't know how common alpha-PVP is because it's just not a commonly used drug and the records really are only from Russia. What was the key feature there was it was psychosis rather than severe tachycardias, etc, that was the problem.

Q. Thank you. Can I ask you a question or two about alpha-PVP and MDMA when taken in combination. Can you comment on the effects when those two drugs are taken in combination; are the effects compounded?

A. It seems not. Again the literature is very limited. It would be lovely to do studies where you give people both and look at how they respond. It's possibly, probably not ethical. Those kind of studies would give that answer. We don't have them. So the only literature we have is from Yekaterinburg, where blood samples were taken and they looked at the concentrations of these drugs and they looked at how the patients present. It was a very nice study and what was quite clear was it seemed to make no difference whether MDMA was in the body or not in terms of psychosis in particular.

I can't remember the data precisely for tachycardias, but it seems to be that alpha-PVP was a dominant drug and anything else was not really important in terms of the clinical presentation. So adding the MDMA to the alpha-PVP doesn't seem to make a difference.

Q. Where a person is known to have both alpha-PVP and MDMA in their system, is there any way that you as a toxicologist can determine whether those drugs were taken together or separately?

A. No. But clearly there are options. People do take drugs, sometimes they take two different ones, but often they are contaminated with each other and you have no idea.

Q. I would like to move on to ask you questions now about the anabolic steroid nandrolone, and I believe that you were sent, I think by the Crown, a copy of a report prepared by a Dr Walker, an analyst from King's College in London, which confirmed that he had analysed a urine sample taken from Sheku Bayoh and found nandrolone and its metabolites to be present and offered the view that the results were consistent with the recent administration of the anabolic steroid nandrolone. Can you tell us a little about nandrolone?

A. It's an anabolic steroid commonly used by bodybuilders and it is associated with cardiovascular complications.

For chronic use it's not a very safe drug to use. I don't think acutely -- we don't consider it acutely toxic, so if someone takes it recently we wouldn't expect that to affect their clinical presentation with any other drugs. If someone is taking it long-term there can be various complications of taking steroids.

Q. So what physiological effects then would taking nandrolone have on a person?

A. It might cause -- I mean it causes muscle build up. It would -- it may give you higher blood pressure, potentially a risk of psychosis. I mean, we know that steroids have -- are associated with psychosis, so it's possible that taking it long-term would make you more at risk.

Q. Can we look at paragraph 137 of your statement please. You say that you looked to TOXBASE for guidance between 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."

A. Yes.

Q. And in paragraph 138 you say that chronic use may have caused Sheku Bayoh's heart to be pathologically more

prone to problems.

A. Yes, but -- yes. I mean, if it had been severe you would have seen that at post mortem and nothing was really seen at post mortem, so it's possible that chronic nandrolone use may have caused his heart to behave differently under the effects of the other drugs but there was no clear evidence of that from the post mortem.

Q. So what effect would you expect if a person had nandrolone in their system and then they took alpha-PVP and MDMA in addition to that the nandrolone?

A. Well, the cause of death -- so the chronic use of nandrolone may make your heart more sensitive to have dysrhythmias. I don't think you would see anything in the heart -- you might see the nandrolone effect but you wouldn't see any effect of the alpha-PVP or the MDMA. There's nothing there present at post mortem.

Q. Again, I would like to share with you a little extract from Dr Lipsedge's evidence in relation to nandrolone simply for your comment. He said:

"These anabolic or bodybuilding steroids, there have been quite a lot of reports of them causing aggressive behaviour. However, more recent research suggests that the earlier papers tended to overlook the possibility that the bodybuilder was taking stimulant drugs at the

same time, that is to say drugs like amphetamine. Amphetamine in large doses can cause violence, aggression, hostility, paranoia and so on, so the earlier reports didn't disentangle the possibility that it wasn't just the steroids, the bodybuilding steroids, but it could have been other drugs taken at the same time and which were being commonly taken by bodybuilders."

Again, can I just invite your comment on that evidence?

A. I defer to him and his knowledge of people using nandrolone in particular. We know that people taking steroids, so medical steroids like prednisolone or dexamethasone, psychosis is a clear and common side effect of it. So while those studies didn't disentangle the two, I don't think that means nandrolone couldn't have been involved in it. Steroids can cause psychosis but many bodybuilders are using multiple drugs, so again to tease out which particular drug makes it more likely is difficult. It is often about consistency and it's about -- it's not unreasonable to think the nandrolone may have increased his risk of psychosis with the alpha-PVP.

MS THOMSON: Thank you. I'm conscious of the time.

LORD BRACADALE: Is that a convenient point to take a break?

We will take a 20-minute break.

(11.30 am)

(Short Break)

(11.52 am)

LORD BRACADALE: Ms Thomson.

MS THOMSON: Thank you.

Professor Eddleston, can I move on now to ask you some questions about alcohol and can I begin by asking you to describe the physiological effects of alcohol.

A. Okay. So alcohol is a GABAA agonist, if we're going to talk pharmacologically. So this is a type of -- this is a receptor throughout the body but in the brain in particular. It's a downer, so when the GABA binds to this receptor more chloride goes through into the cell and the cell becomes calmer and less likely to do things effectively.

So what alcohol does is the same thing as GABA -- so benzodiazepines are also GABA agonists, meaning drugs which interact with the GABA receptor and calm things down, which is why we give benzodiazepines -- I'll come back in a moment -- we give benzodiazepines to people with seizures because we are trying to calm the brain down. So that's what GABAs do, the GABA receptor is involved in.

So alcohol stimulates these receptors and what it

seems to do in the frontal part in the brain, it interacts with different receptors in different parts of the brain at different doses so that when you start drinking alcohol it stimulates the GABA receptors in the frontal parts of your brain and the frontal part of your brain is about control, it's about executive function, making sure you do really sensible things in places like this. With alcohol you lose that. So you become a little bit more freer, people think they become more charming when they have had a couple of glasses of wine, and that's because the frontal lobe has gone. As you have more and more alcohol you start getting more and more suppression of your brain function until you end up comatose with certain amounts. The amount of alcohol you drink chronically will determine how well you can handle the acute drink you have, the drink you have today. So if you have lots every day, you can handle alcohol really easily. If you don't drink very much, you will have more effect from the alcohol you have today and there's clearly variability by people.

Q. Thank you. Does alcohol have an effect, chronic or acute, on the heart?

A. Yes, it causes a cardiomyopathy, it damages the heart in chronic use.

Q. What is a cardiomyopathy?

A. So cardio, heart; myo, muscle; pathy, not working. So if you put the three together, it's the heart muscle not working terribly well and there's various types of that. So taking steroids for a long time will cause a cardiomyopathy, taking alcohol excessively will case a cardiomyopathy. I'm not a pathologist and I can't particularly tell you the mechanism it does that but we recognise that having chronic heavy alcohol use will make the heart more prone to problems.

Q. What would you anticipate the effect would be if a person took alcohol in combination with stimulant drugs?

A. So alcohol is a downer, it stimulates the GABAs and therefore your brain gets quieter. Stimulant drugs cause an upper, so it's going to be a mixture of effects and will depend on which particular drug you have taken, the doses of them, the timing of them will all determine what would happen. One might imagine that alcohol would dull the stimulant effects of the other drugs and kind of make them less severe. One of the problems is that chronic alcohol use changes how your liver handles various drugs and so if by taking alcohol every day for a year, it changes your liver's ability to handle one of these stimulants, it may make the stimulant worse or may make it better. It depends on the drug, it depends on

the time. It's very variable. But it's reasonable to say that the alcohol may make the effect of the stimulant less severe.

Q. How quickly is alcohol metabolised?

A. I don't know the exact times of that. I mean we use -- so most drugs are metabolised or broken down according to the concentration in your blood. We talk about half-lives. So you get rid of a drug -- let's say amphetamines, you get rid of -- you go from this to half of this in a certain time point, it's called a half-life. With alcohol it's not like that so the concentration you have in your body doesn't determine how quickly you get rid of it because in most other drugs the more you have in your body, the more you get rid of. With alcohol you get rid of one unit per hour roughly, but individuals will change somewhat, it will vary between different individuals. But if you drink eight units, I'm not sure how many shots of vodka that is, but that's going to give you a certain number of hours, it's going to give you around eight hours to get rid of it, six hours to eight hours. But there's variability between people.

Q. There were reports that Sheku Bayoh consumed alcohol over the course of the evening and the night before his encounter with the police. However, blood and urine

screens were negative for alcohol. Can you help us to understand why that might be?

A. It would suggest that the time interval between the blood sample being taken and the alcohol being ingested was more than the number of units that had been taken, so if he had -- if you take five units over five hours, each hour you're getting rid of a unit so you are kind of gradually -- you haven't got a very high concentration. If you take a lot, six or seven units in one go, it goes high and takes six or seven hours to come down, so if you look after six hours you may see no evidence. Again, that's a rule for the whole population, that one unit per hour. There's clear variabilities between people and that's partly about how much you drink routinely.

Q. So is that another way of saying that the alcohol potentially was metabolised by the time the blood and urine samples were taken?

A. Yes, I should have said that. I agree.

Q. I think I might have been quoting Dr Shearer there, Professor Eddleston, in fairness. She gave evidence to the Inquiry when asked a similar question that alcohol is metabolised reasonably quickly and that could explain the negative blood and urine screens.

A. Very easily. I mean if you take 2 units, four or

five hours later there's nothing left in the body.

Q. Can I move on to ask you just a few questions about caffeine.

A. Okay, yes.

Q. Can you tell us a little about caffeine?

A. It's a stimulant. It's an adenosine receptor agonist, so it has a different mechanism to amphetamines but it's also a stimulant, it causes increased -- I could have read this up before I had come if I had thought -- it's a similar -- it works on a different mechanism to amphetamines, it's not working at the dopamine receptors, it's working via adenosine receptors I think, and it's simply a stimulant which we become addicted to and therefore in the morning when you wake up and need your cup of coffee, you're dealing with your dependence.

Q. Does it have an effect on the heart?

A. Yes, any stimulant will cause tachycardia, increased rate of the heart. If someone takes -- so now we have these caffeine tablets or we have Red Bull with large amounts and people become extremely sick and die now from caffeine overdoses by taking caffeine tablets in particular, and if you take too many Red Bulls you can become sick -- sorry, I will try and speak slower -- and people die from cardiac problems if they take too much caffeine, so their heart goes into a chaotic rhythm and

you will die and they're very, very difficult to treat if you take too many caffeine tablets.

Q. I think you may be aware that a supplementary toxicology report was prepared in this case and it confirmed analysis of Sheku Bayoh's hospital and post mortem blood for caffeine.

A. Yes.

Q. Between levels of 1.6 and 2.2 micrograms per litre. Can you help us to understand the significance of caffeine being present in his blood at that level?

A. Not at that level in particular. I'm not sure what the concentrations are after people drink coffee. But we do know that caffeine is used to cut drugs, so caffeine being present in someone who dies from a drug overdose is not uncommon. It could easily be explained by a coffee being drunk before the time or a Red Bull, or by having it present in the drugs they bought.

Q. I'm going to move on to ask you questions about CS and PAVA spray and again if we could pull up your Inquiry statement please and go to paragraph 130. While we're finding that, can you tell us a little about the physiological effect of these sprays on the human system?

A. So effectively they're irritants. They bind to receptors present in your mucosa and cause irritation,

so the -- if you leave it on long enough the tissues will get damaged and potentially die. The effect of this is to cause lachrymation, so whenever we get irritated around our face we start crying, we start getting rhinorrhea, fluid coming out of our nose, as the cells of our linings start expressing fluid. I guess you could argue teleologically that it is trying to remove this nasty thing that's on there. And what happens is if it's on your face you would just cry a bit and you would probably -- they're called tear gases, the idea is that you close your eyes as you try to get the fluid out so you can't see what you're doing. It limits your ability to do things, that is why tear gas is used in crowd control.

When you have too much or the concentration is high and it goes into your lungs then you have quite significant problems in the lungs. The mucosal linings of your lungs start responding to this irritant. The clinical problems with it tend to occur when in a closed room. There have been very few deaths from either CS or PAVA but where they have happened they have tended to be in people who maybe already have a lung problem and then they have been exposed to this gas either in a constrained space or put into a constrained space after exposure and are unable to clear their lungs with

fresh air. Yes.

Q. Thank you. We now have paragraph 130 up so if we could perhaps look at that. You say there are over 17,000 poisons listed on TOXBASE:

"I am aware that CS is not known to be a cardiotoxin."

What is a cardiotoxin?

A. So cardio, heart; toxin is a toxic substance, so a cardiotoxin is something that damages the heart, which you would expect to cause primary heart problems. Sorry.

Q. No, thank you:

"... 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."

And you mentioned a moment ago that the reported deaths generally involve individuals who have been in restricted spaces --

A. Yes.

Q. -- when exposed to these sprays. And you go on at

paragraph 134 to offer an opinion that it is unlikely on balance that the sprays had an effect on Mr Bayoh's heart and at 135 you say that you don't think you have treated patients directly, but you have provided advice to clinicians over the phone where those clinicians have been dealing with patients who have been exposed to sprays?

A. Yes.

Q. And further down at paragraph 136 you say that that advice is generally that it will settle with fresh air and observation and that recovery is usually spontaneous and quick.

A. Yes.

Q. Can we perhaps move away from your statement for a moment and bring up on the screen your report, the one that you prepared for the Crown Office. Can we go to paragraph 11 please. So you have a heading above this paragraph, "Psychiatric diagnosis", and as we discussed earlier, amongst the papers that were provided to you by the Crown Office there was a report from Dr Lipsedge. You say:

"Dr Lipsedge in his expert witness report ... provides a retrospective psychiatric diagnosis for [Sheku Bayoh]. His review of the witness statements, toxicology reports, and CCTV footage causes him to

diagnose [Sheku Bayoh] 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' ...."

And you give the ICD-10 codes for both of these diagnoses:

"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."

I would like to ask you some questions about that paragraph. We don't need to go back to your statement at this stage, but in paragraph 36 of your statement you say that although you and Dr Lipsedge use different terminology, you don't think there's a difference in terms of what you're actually saying you consider the diagnosis to be?

A. No.

Q. So as a toxicologist you prefer the term drug-induced

psychosis and I just wanted to be clear whether drug-induced psychosis is a diagnosis that you make as a clinical toxicologist?

A. So it's a working diagnosis we make, so -- but I work very closely with liaison psychiatrists who are psychiatrists who work in the general medical hospital and work with patients expressing mental health illness in the general hospital, rather than psychiatrists -- adult psychiatrists generally work in a separate psychiatry hospital. So we would have a patient come in, I would make the diagnosis, "I believe this is drug-induced psychosis", and then every patient I present to a psychiatrist and we would then have a discussion. At the time it is very difficult, as I said before, to make a clear diagnosis because this may be the mental health illness in someone who takes drugs. So this would be the diagnosis I would proffer to a psychiatrist who would then refine it and have the ultimate say. The psychiatrist would decide ultimately what it was.

Q. And what led you to the conclusion, having reviewed the papers that you were provided by the Crown, that Sheku Bayoh was suffering from drug-induced psychosis?

A. He displayed many of the features which were typical of the patients I would see in my ward in Edinburgh, in the

Royal Infirmary of Edinburgh, of a psychosis, of a period of not being able to understand what's going on, of being not interacting with your environment, and the likelihood of being drug induced as a result of the toxicology reports I had read as well.

Q. I would like to take you again to a passage from Dr Lipsedge's evidence last week. He was asked to explain to the Chair what psychostimulant psychosis is and he said:

"Psychostimulants are drugs like amphetamine, like cocaine, like the group of drugs called cathinones in which they have a sort of exciting, excitatory effect on the brain and the nervous system and there is a release of dopamine, which is the pleasure seeking component. There's also a release of adrenaline and noradrenaline which can raise -- very commonly raise the heart rate, raise blood pressure, and cause strain on the heart to make the heart vulnerable. And if a heart is deprived of oxygen, which can happen during restraint, then there is a risk of cardiac arrest due to disturbance of the rhythm of the heart."

From your perspective as a toxicologist would you agree with his description of psychostimulant intoxication or drug-induced psychosis?

A. Yes, absolutely. I mean there's a difference there

between psychostimulant excitation -- he says psychostimulant psychosis and I agree drug-induced psychosis. He then said psychostimulant intoxication, which can involve the heart. That's the whole panoply not just the psychosis element. So there's a slight difference in terms because he is talking about the whole effect of being on drugs while we're talking here -- the drug-induced psychosis is only one element of that panoply of effects you might see as a result of taking the drugs.

Q. I see, thank you. Can you offer a view as a toxicologist as to the likely primary cause of Mr Bayoh's drug-induced psychosis?

A. The literature suggests that the blood concentration -- of the two drugs he is taking, that alpha-PVP is by far the more likely drug to cause psychosis. It's uncommon in MDMA. The most data I have seen is 4%. The cases we have seen of patients in Russia was 65%, so on balance of probabilities it was the alpha-PVP that caused the psychosis.

Q. Again, we don't need to go to your statement at this moment in time, but you offered the view in your statement at paragraph 101 that the psychosis, on the balance of probabilities, would not have been due to the MDMA?

A. Yes.
Q. Can you offer a view as to what part, if any, was played by the nandrolone?
A. If I look at what evidence I would want to look at to try to understand the role I would want to know more about how the -- the history of his taking. Was it a chronic use of nandrolone or was it a short-term use? If it was a chronic use there's more likely to have been a problem resulting from it, but long term use of nandrolone is more likely to have caused problems with the heart which would have been visible at post mortems and nothing was seen at post mortem.

So since the heart wasn't obviously affected, the role of nandrolone is likely to be low but not -- it's not absent. If he has been taking it and it has caused problems with the heart which has not yet caused changes which you can see by microscope, it may have had a role. I don't think there's a need -- there's a necessary need to say that nandrolone was involved in the psychosis.
Q. Certainly Dr Lipsedge, coming from his perspective as a psychiatrist, gave evidence that it was his view that the steroids were unlikely to have contributed significantly to Mr Bayoh's paranoid and violent behaviour which was better accounted for by the alpha-PVP.

A. Okay.
Q. Would you agree with that statement?
A. I accept that, yes.
Q. Returning to the screen and at the bottom of the page -- we have looked at this already -- you say that the diagnosis is:

"... similar to the diagnosis of 'excited delirium' that is used in the USA."

And you go on to say:

"The term 'excited delirium' is not used in British clinical toxicology practice~..."

Can you explain what you mean by that? It may be stating the obvious, but when you say it is not used in British toxicological practice what should we understand?

A. So the diagnosis is not accepted as a relevant diagnosis for a patient who is presenting to hospital. So clinical toxicological practice is about the care of patients in a hospital and I've never heard the word used in clinical practice. We don't think in terms -- it seems to have developed out of the American post mortem police restraint literature and really just hasn't penetrated, you might argue, into the UK literature. We use ICD-10 which has the terminology which is required to explain how Mr Bayoh was

presenting.

Q. And we have heard that excited delirium is not listed in ICD-10?

A. Absolutely.

Q. Or for that matter in DSM-5?

A. Okay.

Q. I would like to ask you some questions about your experience of treating patients suffering from drug-induced psychosis. Where do you encounter such patients; would it be on your ward rounds at Edinburgh Royal Infirmary?

A. So generally in the ward and the ward round is a certain time of day when we go and see each individual patient and sometimes that psychosis may be present in the patients we see on the ward round but they may also be present in the day and I would go back to the ward to support the management of those patients.

I also see it in the emergency department. So if I'm asked to go through and help, if a patient that comes in who they believe is a drug-induced psychosis we might be asked to go and support the emergency department in conditions -- managing the patient, then I would stand back and be talking to the senior doctor there and just giving advice as the emergency doctors would be caring for the patient.

Q. Typically do you know what drugs the patient has taken?
A. It depends what you mean by know. So I will be told, "This patient has taken cocaine", and there may be some history, but that person may have thought they were buying cocaine and what they truly bought is anyone's guess. So do I really know what each and every patient is affected by? No.
Q. And if you did have that information would that make a difference to your management of the patient?
A. Not particularly but if we had really good information that this drug was more dangerous than this one and this one causes this, then we could bear in mind what we would expect to see. But that's really the only use we would have for it, to say actually this patient may get sick in 6 or 7 hours because we have data that's what happens with these patients, but fundamentally the care we offer to the patient is based on how they are in front of us so actually knowing what they have taken is actually not very important.
Q. We touched on this in passing before the break, but do I understand that you treat patients symptomatically because it would take several weeks if you were to take a sample of blood for analysis?
A. I mean, irrespective of what they have taken they are presenting at a hospital with a problem and that problem

is something we can try to help them with. That problem might be agitation, it might be psychosis, it might be a high temperature, so we try to assess those problems, whether they are present or not, and then we treat those problems irrespective of what they have taken.

Q. Typically do you know a patient's medical history?

A. Mostly yes, but I'm fortunate because by the time I see them in the ward the medical records have been integrated together. When they first present to the emergency department where my colleagues in the emergency department will generally see them first, 50/50. It would depend if they know the name, how much information they have. Because everything is electronic now we can go back on and look at the medical records. There will be some people come in as "unknown unknown" and we have no idea who they are until they recover and then we find out who they are.

Q. When you treat a patient who is presenting with psychosis can you tell whether their psychosis has been caused by drugs or mental ill health, or potentially a combination of the two?

A. So again you used the words "Can I tell". So I would have hypotheses. A lot of medicine is about coming up with an idea which you test and try to understand whether you are right or not. So if someone comes in,

they often have a label: this is drug-induced psychosis, and we might try to get a urine sample to go and test what drugs are in the urine which gives us a very crude idea. It is not very specific for particular compounds, it might say they have taken cocaine or an opioid or an amphetamine. But we need a urine sample for that which is quite difficult to get from an agitated patient clearly, so there's a general presumption when people come in there's a good chance it's a drug causing the psychosis.

Many people with mental illness do use drugs and so therefore it's -- it's actually impossible to tell initially what's caused the psychosis but there's often drugs involved. But the thing about drugs involved is it will go over several hours or days, so our general presumption is: this is probably drug-induced psychosis, and we will watch and see and support them and keep them safe during that time it takes for the drug to wear off.

If after three days there's no effect at all, and perhaps by then we have managed to get a urine sample and there's nothing in the urine, then we say quite confidently at that point: this is probably not drug-induced psychosis, this is most likely to be mental health, a direct mental health illness. But they're very overlapped. People with -- people with mental

illness treat themselves as well with drugs, so therefore there's a big overlap.

Q. Thank you. Can we go back to your statement please and to paragraph 52. In paragraph 52 you say:

"I have 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 have 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 is going on and where we think it is probably drugs, we would do pretty much the same thing, but we may not be successful at getting oral medicines into them."

So should we understand that the cause of a patient's psychosis doesn't really change your management of the patient, it's about treating them symptomatically?

A. Yes, that's true, especially in the first -- in the

acute situation.

Q. Yes. Can we look at paragraph 72 please. You were asked about your experience of treating patients with alpha-PVP and say you can't 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."

So should we understand that although you have treated many patients suffering from the effects of stimulant drugs, it's simply impossible for you to say whether you have ever had a patient who has taken alpha-PVP?

A. It seems unlikely, but I can't say for sure either way.

Q. And at paragraph 73 you explain that testing for the presence of alpha-PVP takes time. When you do your research you take a blood sample to say this patient has 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 has taken a stimulant or he looks like he has 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 precisely what we're treating. If we were to take blood samples from all of 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 current patients."

You mention there treatment pathways for different classes of drugs. What would be a treatment pathway for a stimulant like alpha-PVP or MDMA?

A. It's really very limited. We don't have a pure antidote, we don't have a drug -- we don't have a treatment or an antidote which reverses the effect of the drug which we have for heroin with naloxone. So for stimulants we can only try and treat them

symptomatically. And those -- again, it's trying to get agitation under control, so we use benzodiazepines, so drugs a little bit like alcohol which calm the brain down; we would look for their temperature and independently try and bring their temperature down; and if they were psychotic we would initially use benzodiazepines but we would also potentially use antipsychotic medicines, with discussion with our psychiatric colleagues.

Q. Thank you. Can I ask you how a person suffering from stimulant drug-induced psychosis would present? How would you expect to see them on the ward?

A. They would often be brought in by the police or by their family, so they wouldn't themselves bring themselves to hospital. They might come in a private car, they might come by ambulance. They may have therefore received some medicines at the site from the paramedics but if they have come in with their family or friends they're not going to have received anything.

They will arrive at the hospital and generally be very agitated, be unsure what's going on, sometimes aggressive if they don't truly understand people are trying to help them. Their heart rate will typically be fast. They may come into the hospital and require restraint, so someone -- I mean we have a busy

department, there's lots of people there, if someone comes in who is very paranoid or very aggressive there's a potential for violence to occur which will influence staff, the patient themselves and also users of the emergency department.

So there's a lot of variability but agitation is commonly there and we will see that because you don't need to -- you can see that as they come in, they're agitated, or they may be psychotic and just completely confused. Then we would try and -- they would be seen very rapidly in the emergency department and then we might try to take observations, look at their blood pressure, look at their pulse, try and get an understanding of their temperature. But often that's not possible until they've got restrained and the patient has been calmed down.

Q. That brings us nicely to a discussion about how you manage these patients on the ward, so when you are presented with a patient who is as you have described, perhaps confused, almost certainly agitated, maybe psychotic, how would you approach that person? What would be your first line, as it were, in terms of treatment and management of that patient?

A. So the first line is to assess the situation: what level of danger is available, how violent is this person?

I mean, an 82-year old woman with psychosis offers a very different situation to a 30-year old man with psychosis, just in terms of physicality, and both of those occur and they both need very careful care but there's a certain difference.

If the situation is not dangerous, if it appears that we can talk to the person the first thing we do is start talking. You talk and try and persuade them -- to tell them where they are, try to reorientate them to what's happening, that they're in a safe place, this is something, that we would try to look after them, but that's -- the key thing is try to create communication with them because the level of agitation will determine how much they can understand. Clearly if they're truly psychotic they will probably understand nothing, but if they're less severely affected something will come through and if they can gradually realise they're in a safe place, there's no aggression going on, that people are calmly talking to them there's a chance they will calm down.

At that point we might talk to them and simply trying to find out what's going on, will they tell us they have taken a stimulant or are we relying on what other people have told us? I would typically use benzodiazepines as a first choice, so diazepam, I take

20 milligrams in my hand and go and sit down and talk with them and try and persuade them to take them and that is about just saying, "This will make you feel better, this is what it is", try to get through to the point where they're understanding. If they can understand they will often take the medicine and then they will feel a lot better. There's other medicines we can use but that's the first one we would typically use.

If it's unsafe, if there's aggression going on that we feel people are in danger, we would call the security and the police within the hospital and use physical restraint. The problems with physical restraint is it is basically a dangerous thing to do and something you try and keep as short as possible, so if that was required because of the situation we would then immediately start drawing up drugs to give either intramuscularly or via a cannula to try to get on top of the situation because the general feel is the shorter the amount of time that someone is restrained, the better the situation. That doesn't mean short restraint is always safe but it's safer than one hour of restraint and if someone has a lot of drugs on board and if we don't give them medicines you will have to physically restrain them for a long time.

So in the hospital you assess the situation and if

we can't use talk to try and de-escalate the situation, we will very rapidly call in physical restraint but someone is drawing up medicines to get on top of them.

The medicine can take a little bit of time to work so the point maybe is -- you know, if I give 20 milligrams of diazepam, how long is it going to take to work. It is partly about how long it will take to work fully and how long it will take to have any effect. A drug going in might have any effect within a couple of minutes. It may not be fully having its effect until 5, 10 or 15, but it might begin to de-escalate the situation and make the person fight less against the restraint that's going on which they can't understand at all.

Q. Thank you. So you say you begin by assessing the situation, effectively carrying out a risk assessment, looking at the level of danger that the person is presenting?

A. Yes.

Q. Would that be to themselves as well as to other people on the ward?

A. Yes.

Q. And if it is safe to do so, then you will begin by talking to the person?

A. Yes.

Q. Can we look at paragraph 42 of your statement please. You have spoken about the importance of communication and of telling the person that they're safe and keeping the conversation going. If we can scroll a little bit further down that paragraph please. You describe the presentation of patients on your ward and say:

"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."

So you talk about using familiar language, empathy and remaining calm and talking to the patient.

Can we look at paragraph 46. You have been describing a process which I think may be known as de-escalation. Would that be an appropriate label?

A. Yes.

Q. And you were asked here to describe the verbal de-escalation techniques that you would use when dealing with a psychotic patient and you say:

"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, language they understand - don't use big words."

So the same themes arise in this paragraph as in the one we looked a the a moment ago: calm voice, keep the conversation going and use simple language.

At paragraph 47 you were asked to describe your body language when you're dealing with a patient who is psychotic and you describe your body language as "Warm and emphatic". You're aiming not to look aggressive:

"Sometimes I have 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 have sometimes asked security guards to leave because they are making the situation worse."

So in your statement you speak about the importance of your tone of voice, the language that you use, the information that you convey and also your body language in terms of trying to de-escalate a situation.

If we can perhaps look at paragraph 51, where you are asked about things that don't work so well with psychotic patients and you say:

"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."

So you have given examples in your statement about how you practice de-escalation on a ward in terms of the language you use, the approach you take, your body language, tone of voice and you have also given examples

of things that will only serve to aggravate the situation and you say anything that will stimulate the fight or flight response is not going to help.

Again, I would like to take you to the evidence that we heard last week from Dr Lipsedge and invite your comment. Dr Lipsedge told us that what he would teach medical students is in a situation like that you have to pretend you've got unlimited time:

"Of course you haven't got unlimited time but you have to give the patient the impression that you're able to listen to them and that you're not in a hurry. Now, of course for professional reasons you might well be in a hurry but you have to convey the feeling that you have unlimited time to spend in discussion with the patient."

Is that a statement that you would agree with or disagree with?

A. Yes, I think people need time, they need calmness and rushing is not a good thing.

Q. Earlier in your evidence before we return to your statement, you told us about the approach that you might require to take either when de-escalation has not been successful, or you might perhaps reasonably assume if your initial risk assessment tells you that it is just not a safe option. Before we look at that in more detail, is it often the case that you have to move

directly to restraint and administration of medicines, or more frequently than not are you at least able to attempt de-escalation first?

A. So in my clinical practice I'm generally seeing them after they have left the emergency department and they're already in a ward and in such a situation I would always use de-escalation. I can't remember any times when I have gone straight to restraint. But in the emergency department when they're coming in from the street and they're first coming into this constrained environment, potentially with the police, sometimes not, there there will be times when it goes straight to restraint.

Q. And what would the reason for that be?

A. The level of danger. You've got an emergency department full of people, you have got a patient potentially at risk of harming themselves and harming the healthcare workers looking after them, so if there's a feeling of urgent potential danger to anybody, the patient or the people around them, then they would move more forward that. By the time they have come through to us they have generally had some medicines so it's not quite such an acute situation of what I generally deal with.

Q. Do you have experience of dealing with the more acute situations?

A. Yes, that's when I get called to the emergency department, I get called down and they say, "Please come and help us we have a patient", but I'm not generally the one leading those arrests -- not arrests, dealing with the situation is dealt with by the emergency department but I stand with the consultant and we talk.

Q. Thank you. If we could perhaps go to paragraph 55 of your statement which again is a reference back to your report and you say that:

"Sometimes the agitation is too great for this approach [that's the de-escalation 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."

Could you perhaps describe the process of restraint as carried out in an emergency department, one that you have perhaps witnessed when you have been talking to the consultant?

A. So there are security people available in the hospital. There are police. There's a small police station at the

back of the emergency department in the hospital, so if a situation is agreed that it is urgent, restraint is required, a phone call will go out to security to ask for people to come and generally -- six people would be a normal number to actually try to get on top of things because you don't want too few people. You want to make sure you can physically hold limbs and stop them kicking out and have enough people there to hold the person down in a safe position while the medicine is being administered.

Q. You said earlier in your evidence -- when we were looking at the addendum to your report -- "I'm not an expert in use of force, I'm a clinician who has to at times use force to safely care to a patient"?

A. Yes.

Q. So the purpose of using force, should we understand that purpose to be as a means to an end and that end is to provide care to the patient?

A. Absolutely, and also to hold them -- they may be harming themselves at the time. Getting hold of arms and limbs can keep them safe from whatever they are doing. So not only is it a means to an end to get a chemical restraint in -- we talk about physical restraint and chemical restraint. Physical is the physical holding of hands, chemical is using medicines. So we use physical to get

to the chemical, but actually the pure act of physical as well may keep them safe.

Q. I would like to talk to you about two hypothetical situations. We have heard evidence at an earlier stage in the Inquiry from experts in the use of force and police tactics and restraint and two scenarios were discussed that both involved engagement by the police.

The first scenario was described as de-escalation and that was described to us as a scenario where the officers would engage and negotiate and de-escalate, try to understand what was going on, to allow them to inform their decision-making about the process. It was an opportunity to communicate, it was key to building rapport and they would attempt to de-escalate, engage and negotiate. So we will call that the de-escalation scenario.

Another scenario that we heard about in evidence was what was referred to colloquially as a hard stop, or otherwise a verbal dominance approach and it was described as an authoritarian approach, wanting to try and control the individual, a verbal dominance approach of communication, a methodology of trying to dominate the individual by getting them to comply with the instructions to minimise the risk or minimise the requirement to possibly use other force.

So we were calling that the verbal dominance approach or the hard stop and I wonder from your perspective as a clinical toxicologist, and given your experience of managing and treating patients suffering from drug induced psychosis on wards, whether you might help the Chair to understand the difference that each approach might lead to in terms of dealing with someone who is intoxicated and suffering from agitation and psychosis?

A. In a way you could say they are the two options in a way. Our risk assessment says can we go in with de-escalation, and that works in some, many cases, I can't give you a number but that is an approach which clearly works and allows you to get medicines into these very agitated people and calm the situation quite quickly.

The second thing you're describing is not that dissimilar to a situation where someone has come in and the risk assessment says this is too dangerous, we cannot allow this to continue, what he or she is up to, and we need to get on top of the situation. That might be the situation where you would yell but I think the big difference between the situation -- Mr Bayoh's situation and what we have is we are, you know, at home in a way. We know our set up, we know our individuals,

we're working as a team and we're not arriving somewhere, the person is coming to us.

So we don't -- our hard stop is different in a way. We might not yell at all to the patient. We might simply go, "We need drugs now, we need security now", we've got 3 minutes, 4 minutes to get on top of the situation so the security guards will be there and they will go straight in and get on top of the situation, we will go straight in and give medicines.

So if you were describing our routine -- if our routine approach was to do the hard stop approach, that would inflame the situation as it wouldn't be required in many of the cases. But again we're very trained in the hospital, we know what we're doing in these situations, they're common, we build up skills, so we're working from the situation where we really -- the hard stops would be done differently because the restraint is purely to get on top and then get drugs in, which is not available in the community at the moment.

MS THOMSON: Thank you. Can you bear with me just a moment please.

(Pause).

Thank you, Professor Eddleston. I don't have any further questions for you, thank you.

LORD BRACADALE: Are there any Rule 9 applications? Just

the one. Professor Eddleston, I wonder if you would withdraw to the witness room while I hear a submission.

(In the absence of the witness)

Yes, Dean of Faculty.

Rule 9 Application by the DEAN OF FACULTY

DEAN OF FACULTY: My Lord, I'm obliged. It is very brief. In the course of the examination of the witness he was taken to his original report and at pages 27 to 28 of the transcript learned counsel looked at paragraphs 17 and 18 in the original report and then at line 5 on [draft] page 28 said:

"Question: In the paragraphs that follow you set out how patients with acute psychosis are managed in emergency departments. You offer a view as to how Sheku Bayoh might have been handled differently by the police and you conclude in paragraph 24 by recommending a discussion between police and emergency medicine physicians to increase understanding of how to manage these patients and to refine the current guidance."

"Answer: Yes.

"Question: Would that be a fair summary of the addendum to your report?

"Answer: Yes."

My Lord, the short point I would like to explore is

that in that summary, and indeed in the witness statement, there's been no consideration of paragraph 23 of the original report which might contain material of assistance to the Chair in the ultimate determination and I would welcome five minutes, and I don't think it will take more than that, to explore paragraph 23 and the first sentence of paragraph 24 of the original report with him.

LORD BRACADALE: Could we get the report up on the screen please and these paragraphs.

DEAN OF FACULTY: I think it is there so if we just keep scrolling. Yes. It's 23 and the first sentence of paragraph 24, my Lord.

LORD BRACADALE: Just let me read that.

DEAN OF FACULTY: Indeed.

(Pause).

LORD BRACADALE: Yes, very well. I shall allow you to explore that with the witness.

(Pause).

(In the presence of the witness)

Professor Eddleston, Mr Dunlop, who is Dean of the Faculty of Advocates, represents certain police officers who attended and he has some questions for you.

Questions from the DEAN OF FACULTY

DEAN OF FACULTY: Professor, just very briefly please. You

will see on the screen we have I think the report from 2017 to which you were taken earlier.

A. Yes.

Q. I would just like to explore briefly the content of some of what's on the screen. At paragraph 23 you say:

"Overall, the situation on that morning was difficult and fast moving. A powerfully built psychotic man had been violent to people and cars. He did not respond to clear police instructions."

Is that consistent with what you had been told regarding this matter?

A. Yes.

Q. You go on to say:

"Unfortunately, his psychosis prevented him from understanding the situation or the instructions. His paranoid thoughts caused him to attack the police, resulting in physical restraint until submission, without the aid of medicines to induce sedation administered by paramedical or medical staff."

If we just pause there, I wonder if you can help us with a couple of aspects of this. Firstly the psychosis preventing him from understanding the situation, can you help his Lordship -- what's going on in the mind and what is the process that prevents the person undergoing the psychosis from really comprehending the actuality of

what's in front of him?

A. I'm not an expert on this, so Professor -- Dr Lipsedge would have been a person better able to describe what's really going on inside their head. What I see from my clinical interaction with these patients is a puzzlement, overstimulation of the brain and a lack of understanding of anything that's going on with them once they get to a certain level. Clearly by my de-escalation I'm trying to either bring them down to a level where they understand -- it wasn't attempted with him so I'm not quite sure whether it would have worked, but he didn't seem to understand what was going on.

Q. Okay, thank you. The second aspect of that, you say:

"His paranoid thoughts caused him to attack the police~..."

Now -- I mean again, would your answer be the same there, that that's really for Mr Lipsedge to look at, or are you able to help us with your reasoning in that regard?

A. We know that paranoia is part of psychosis and paranoia is a certain interpretation of the world around them which involves danger to the person and the person is responding to that. I think what I was trying to do here was explain why I had come off subject of what

I wasn't asked to answer on to try to explain the situation as I saw it, but it wasn't as an expert, which I did say at the beginning.

Q. Yes. I'm interested then, just before we close, in the last sentence at paragraph 23, you say:

"At the point physical restraint was started, his prognosis was poor."

A. Yes.

Q. And again, if you could just assist the Chair in that regard please?

A. We know that the longer the physical restraint goes on, if someone is violently fighting back against physical restraint that puts them at risk of hypoxia, of low levels of oxygen in the blood, which would increase the risk of cardiac dysrhythmia or something called ventricular fibrillation occurring. In that case, unless that happens in a hospital that's a very dangerous thing and will likely result in death. CPR can be started by bystanders, which can sometimes be effective, but I think the fact that the physical restraint was started without the drugs being drawn up, without a plan in place to actually administer those drugs, then his prognosis was poor.

Q. And you say that at the point the restraint was started the prognosis is poor so looked at at the moment the

restraint starts, what is it that makes the prognosis poor at that point in time?

A. Because he is not understanding what's going on, he is fighting against the restraint and that restraint -- that is going to go on until he is exhausted and that puts him at risk of not being able to breathe properly and his heart not working properly and he could die from it.

If that restraint had been started and there had been a plan, an availability of a paramedic or a doctor, able to provide drugs within a few minutes, then the prognosis would have been different. It would have been better, I am not sure how good it would have been. But I think almost as soon as you get medicines into a patient there is a beginning of the process of chemical de-escalation, but by going ahead and doing this without that plan in place the prognosis was very poor.

Q. And of course whether or not that plan is able to be in place will depend upon the particular circumstances?

A. And also the systems in place within the healthcare systems, can we do that, for sure.

Q. Finally, Professor, at 24 you say:

"Psychosis is a well-recognised complication of stimulant drug use with a poor prognosis when public

safety requires physical restraint without medical support."

Is that really just a follow on from what you have just told us?

A. Yes. Medical support is about having a clinician around, it could be a paramedic or a doctor or a nurse, who is able to give medicines that will calm the situation down.

Q. So that's the ideal but --

A. That's the ideal, and it does happen sometimes. For example, in Edinburgh there's a vehicle sitting outside the hospital which gets called out to various situations. In general that's for trauma, so a car crash they will go out and they will be there to supply them. Occasionally in the past it has been used to go and deal with patients like this and provide drugs, I have talked to emergency medicine consultants who have been in the community to provide these drugs. But there's no process in place, there's no systems in place, I'm not sure it's funded to do that. So it's not the standard approach but it would -- if it was possible in our health system to provide that care at the site of these interactions, it would make things safer. I don't know how safe but it would make it safer.

Q. Do I apprehend from what you have just said that while

that might be an ideal, in truth it rarely happens for various reasons that we can doubtless imagine?

A. I believe so. I don't know. I don't -- from the perspective of an emergency medicine consultant or registrar going out, I don't think it happens very often. But ambulance paramedics, some of them have the skills to be able to do some way along that pathway. They can give diazepam intramuscularly. I don't know the protocols precisely within the ambulance services. So there is some partial way towards that. It's not an ideal situation at the moment.

DEAN OF FACULTY: Yes. Thank you, Professor. I'm obliged, my Lord.

LORD BRACADALE: Professor Eddleston, thank you very much for coming to give evidence to the Inquiry. I'm grateful to you for the time you have given. I'm about to adjourn and then you will be free to go.

A. Thank you.

(12.51 pm)

(The Inquiry adjourned until 10.00 am on Wednesday, 17 May 2023)

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