Wednesday, 14 June 2017
(10.10 am)
THE CORONER: Yes.
MR WASTELL: Sir, before we hear further live evidence, there are some short statements to be read under Rule 23.
The first is a witness statement of Gary Nagioff, who gives evidence about the potential sale of a house called Pinebrook House, St George's Hill, in 2012.
Sir, you can admit this under Rule 23.1(d) on the basis it is unlikely to be disputed, subject to the now familiar gateway under Rule 23.2, that you announce the nature of written evidence, as I have just explained.
THE CORONER: Yes, I confirm that.
MR WASTELL: The full name of the statement maker, as I have just explained, that any interested person may object to the admission of such written evidence and that any interested person is entitled to see a copy of the written evidence.
THE CORONER: Yes, well I confirm all those things.
Thank you very much.
MR WASTELL: This then is the witness statement of Gary Nagioff, signed and dated 7 June 2017 and he says this.

Statement of MR GARY NAGIOFF (read)
MR WASTELL: "I Gary Nagioff of Pinebrook House, St George's Hill, Weybridge state as follows.
"I make this statement on the basis of matters within my own personal knowledge, information and belief. I am the owner of a property rental business called 'Aaron Properties'. My wife and I are also freehold owners of Pinebrook House, which is the property in which we now reside. Pinebrook House was a property which I was responsible for building. It had previously been known as Split Pines B and was one of three properties I built in St George's Hill.
"Pinebrook House was rented out through CHK Mountford between the following dates: (a) to [names redacted], tenancy from 1 August 2010 to 31 July 2011; (b) to [a different name redacted] tendency from 8 August 2011 to 7 August 2012.
"I can also confirm that in 2012 Pinebrook House had been on the market. The guide price was anywhere between £7 million and £8 million. I placed it with Curshods and Knight Frank but this sale was not advertised, it was more a word of mouth/whisper sale as I had previously been messed around by fraudulent buyers, including a young couple, 19/21-years old from Ukraine. I can recall that in 2012 I was told that

a Russian couple had been interested in the property and that they were then currently renting in St George's Hill. It was only after Mr Perepilichnyy's death, which I heard about through the media, that I was told that it was he and his wife who had been interested. I cannot now recall who told me this."
MS HILL: Before my learned friend moves to any further read evidence. We didn't object to that statement being read but I hope, sir, that you have been provided with a copy the representations we made on it, the 12 June email.
Do you have that, sir?
THE CORONER: I do and I've got it in mind, thank you very much.
MS HILL: Thank you.
MR WASTELL: Sir, the next short statement is in the hearing bundle, tab 28. It is from a Dr Paul Loxton, who was a general practitioner who saw Mr Perepilichnyy as a private patient in March 2011. It is page 343 of the bundle for those who don't have tabs.
Again, sir, you may admit this under Rule 23.1(d) on the basis it is unlikely to be disputed, the nature of the written evidence is as I have explained, the name is Dr Paul Loxton. Any IP may object and they are entitled to see it, which they have.
THE CORONER: Yes, and I confirm all those things, yes.

MR WASTELL: In a statement to the coroner officer, or a letter rather, dated 19 November 2012, this is from Dr Paul Loxton of the Virginia Water Medical Practice, about Alexander Perepilichnyy of The Coach House St George's Hill.
Statement of DR PAUL LOXTON (read)
MR WASTELL: "I saw Mr Perepilichnyy on 22 March 2011 as a private patient. He had registered with our practice on 22 March 2011. The address we were given is as above. He consulted me about a long standing pustular rash on his feet, I did not subsequently see him but I did refer him to a dermatologist.
"He told me at that time he was taking Xenical to control his weight and that he was an ex-smoker, having smoked 60 cigarettes per day. The consultation was somewhat hampered by his poor command of English and mine of Russian."
Sir, I don't propose to read the rest of that letter.
The next short written evidence is from pages 344 to 346 in the same bundle behind tab 29. This is from Dr Brian O'Connor, who is a consultant physician in respiratory medicine and allergy. He gives his address as both the Cromwell Hospital and the Lister Hospital, they are both in Chelsea. There are three letters. The

first relates to a clinic on 25 September 2012 and the second two to clinic on 4 October 2012. They relate to consultations with Mr Perepilichnyy regarding his health and some medical tests.

Sir, as before, you can admit this under 23.1(d) and again it is Dr Brian O'Connor, I have explained what the statements are about, all the IPs have it and no objections.

THE CORONER: I confirm all those things. Yes.

MR WASTELL: The first letter then is from Dr Brian O'Connor relating to the clinic dated 25 September 2012 and it is written to Dr Kenrie Li of The Group Practice in Earl's Court Road. It is about Mr Alexander Perepilichnyy, date of birth 15 July 68, The Coach House Granville Close, Weybridge.

Evidence of DR BRIAN O'CONNOR (read)

MR WASTELL: "Dear Kenrie, this charming young 44-year old gentleman apparently had a life insurance assessment declined because of elevated bilirubin, he tells me he may have had some gallstones in the past. As the remainder of his liver function tests are normal the benign condition of Gilbert's syndrome would seem to be the obvious diagnosis, however I have arranged an ultrasound of abdomen and I will see him for a follow up."

In relation to the clinic date 4 October 2012, again writing to Dr Kenrie Li about Mr Perepilichnyy, he writes as follows:

"As expected, this gentleman's abdominal ultrasound shows normal liver texture but also shows some small gallstones, I think the gallstones are an incidental finding and I am quite certain that his isolated raised bilirubin is a manifestation of Gilbert's syndrome.

"His spleen is slightly enlarged, which is not indicative of any pathology. I have reassured him that there is absolutely no reason why he should have any application for life insurance declined. On the basis of my assessment he ought to have a normal life expectancy, he is asymptomatic, he has had recent weight loss because he has been more active and changed his diet. He has no GI symptoms and no symptoms compatible with known gallstones, I have explained to him the gallstones very frequently lie dormant within the gallbladder and in the majority of cases do not cause any problems."

Then a letter of the same date, a letter of 4 October to Mr Perepilichnyy from Dr O'Connor:

"Dear Mr Perepilichnyy, I hope you are reassured by our findings. I am very satisfied that you do not have any illness that will in any way impact on your life

expectancy. The isolated trivial abnormality in liver function and raised bilirubin is very common and is associated with a totally benign condition known as Gilbert's syndrome. Your other liver function tests included hepatitis A, B and C profiles were all negative. Equally there is no need for you to have any concerns about gallstones. They often will be described as incidental findings and in your case are unlikely to cause any problems.

"Please forward this letter and indeed a copy of my letter to Dr Kenrie Li for any life insurance claims. I can assure you there is nothing to suggest any reduction in life expectancy."

THE CORONER: Can you just help me with one thing. We were looking yesterday at the question of when the house move was, do you remember?

MR WASTELL: Yes.

THE CORONER: In the context of another event and anxiety being expressed that, as it were, an address was now on the computer. I am just going back to Dr Loxton.

MR WASTELL: Yes.

THE CORONER: That Mr Perepilichnyy had registered with the practice on 22 March 2011 and The Coach House address was given then.

MR WASTELL: Yes.

THE CORONER: I just can't remember the other date.

Do you remember we looked at it with you yesterday, Ms Hill, you were drawing attention to --

MS HILL: Yes, sir I was. I think the D48 document.

THE CORONER: That was the one. Can you remember the date on that?

Thank you so much.

MS HILL: I think 31 May 2011 on D48 has the previous address, but then I think it is 26 June has The Coach House, so it was that interval that I was putting as a proposed window for moving.

THE CORONER: In fact according to this then The Coach House address has actually been given before. You were saying there is the anxiety and then there is the move, that was the effect of the point.

MS HILL: That is the effect of what I was saying.

THE CORONER: This suggests, does it, that the move was actually before the anxiety.

MS HILL: I will check the Loxton evidence but that is the proposition I put.

THE CORONER: I have that, but do you understand the point I wanted to see does it actually look as if this predates the anxiety?

MR WASTELL: Sir, certainly June 2011, was my recollection of the evidence.

THE CORONER: For the anxiety?
MR WASTELL: No, for the change between the addresses in the financial document that we were looking at.
THE CORONER: Yes. What I am interested in is the date of the incident that gives rise to the anxiety.
MR MOXON BROWNE: Sir, the date of the incident that gave rise to the anxiety is the end of May, not June, and I will give you the reference.
THE CORONER: If that were the case, then he is giving as it were the next address before the anxiety.
MS HILL: That doesn't though fit to be fair, sir, does it, without addressing you on the facts, there is plenty of other evidence of further June entries on the Western Union where the old address is being given, so I don't think even Mrs Perepilichnaya suggests that they moved by March, she suggests they moved in the summer. Without addressing you on it, I wonder if this is something that needs to be checked a little further -- my learned friend Mr Beggs agrees because I think she suggested they moved in the summer because her father died in July -- August, I am sorry.
This is March 2011 address does not fit with all those Western Union transactions either.
THE CORONER: It could be, couldn't it, as it were he registers in one address and when it is changed it just

looks as if that was the original one without it necessarily being it, all right.
MS HILL: I think that is right and I think the understanding is that -- well, in fact I am not sure about that but this would seem slightly anomalous, if I may say.
THE CORONER: Thank you very much.
MS HILL: Of course -- I'm sorry to rise again -- one does also have the later insurance documentation that gives The Coach House address much later and I can't recollect whether there are any 2011 documents on that. One would need to do a slightly wider search I think before drawing any conclusions from the Loxton letter.
THE CORONER: All right, thank you very much.
MR WASTELL: Sir, in which case we move to the live witnesses and starting with Dr Norman Ratcliffe.
DR NORMAN RATCLIFFE (sworn)
THE CORONER: Can you give me a divider number?
MR WASTELL: Sir, we are in now the core expert bundles.
THE CORONER: Yes, all right.
MR WASTELL: File 1, divider 22 onwards.
THE CORONER: Thank you.
Questions from MR WASTELL
MR WASTELL: Can you state your name for the court, please.
A. Norman Arthur Ratcliffe.

Q. As of November 2012, you were a consultant histopathologist, is that right?
A. Yes.
Q. You are now required I retired, I think?
A. Yes.
Q. When did you retire?
A. Almost three years ago.
Q. You performed an autopsy on Mr Perepilichnyy's body on 14 November 2012 --
A. I did.
Q. -- correct?
A. Yes.
Q. There are a number of documents in front of you, there should be a bundle marked "Core bundle of documents for experts, file 1". I hope it is open?
A. Yes, I think that is file 1, yes.
Q. Can I take you first of all to tab 26. There should be page numbers in the top right-hand corner and it is page 131.
A. Yes.
Q. That is a post mortem report produced by you and signed on 16 November 2012 --
A. Yes.
Q. -- relating to the autopsy?
A. Yes.

Q. Turning back in the bundle to tab 23.
A. Yes.
Q. Page 119 to 121 in the top right, is that a further or expanded report produced by you and dated 4 May 2013?
A. Yes.
Q. As well as your reports, you have answered some questions put to you, haven't you, firstly at the direction of the coroner?
A. Yes.
Q. Just to identify those, behind tab 24, top right, page 122 to 124, do you see there questions from the coroner dated 15 December 2014 and your answers?
A. Yes.
Q. Are there any corrections to those answers?
A. Yes, the answer to question 2 is clearly an error and there was an earlier report.
Q. Just to be clear, question 2 is:
"Your autopsy report is dated 4 May 2013, ie six months after Mr Perepilichnyy's death, did you produce an earlier report?"
There you have said, "There is no earlier report".
A. Yes, that is a mistake.
Q. Yes, clearly, we have just seen an earlier report?
A. Yes.
Q. You also answered questions in May 2016 I think, if you

turn to tab 26, page 129, top right-hand corner are some questions posed on 4 March, do you see those?
A. Yes.
Q. Your answers at page 130?
A. Yes.
Q. Just before we put that bundle to one side, you also I think have provided some notes, back to page 25, top right-hand corner, pages 125 and 126 behind tab 25.
A. I have that as my -- yes, these are my handwritten notes, written immediately at the end of the post mortem.
Q. Written at the end of the post mortem on 14 November 2012?
A. Yes.
Q. Finally you took part in a meeting with Dr Fegan-Earl, the consultant forensic pathologist, didn't you?
A. Yes.
Q. If we turn to --
A. Sorry, which meeting do you have in mind? The meeting immediately before he performed his autopsy?
Q. Sorry, no we will come to that in due course but in terms of reports that you have produced for this court --
A. Yes.
Q. -- you took part in a meeting with Dr Fegan-Earl --

A. I did.
Q. -- in May of this year, correct?
A. Yes. Yes.
Q. Can we just identify the report. It is behind tab 97 in core bundle 3, so it is a different bundle, it should be there.
A. What tab?
Q. It is tab 97. Again top right-hand corner you should see some numbers.
A. 856.
Q. If you turn to page 864 --
A. Yes.
Q. -- and turning over to 865, is that the joint agreed statement you produced with Dr Fegan-Earl?
A. It is.
Q. Just at 876, it is 13 pages, and you have both endorsed it.
A. Yes.
Q. In each of those reports, answers to questions and in the joint statement, subject to the correction you have brought to the coroner's attention, are the facts in those documents, insofar as they are within your own knowledge true?
A. Yes.
Q. Are the opinions you have expressed your professional

opinions on the matters to which they refer?
A. Yes.
Q. Do you stand by those opinions?
A. Yes, I do.
Q. Before we come to the findings of your autopsy on 14 November 2012, can we just look at the information that was provided to you in advance?
A. Yes.
Q. If we go back to the bundle 1, behind tab 25, top right-hand corner, page 128.
A. Yes.
Q. What is that document?
A. That is a brief history provided before I undertake the autopsy to me by the coroner's officer responsible for the case, in this instance Mr Mansbridge.
Q. It is dated 12 November 2012 --
A. Yes.
Q. -- and just in summary, it tells you, does it not, that the police are satisfied this was the body of Mr Perepilichnyy?
A. Yes.
Q. That they had been told by his wife he had been out running on 10 November and had not returned?
A. Yes.
Q. That he had been found in the road unresponsive but in

jogging clothes?
A. Yes.
Q. CPR was given by a chef who was first aid trained and then paramedics arrived and took over?
A. Yes.
Q. He had been in asystole, CPR continued and he was pronounced dead, looking at the timings, 46 minutes after the first ambulance arrived?
A. Yes.
Q. The past medical history you were given, just in the middle of the page, was that he was a heavy smoker, but there was no reference there to whether it was a current or past history of smoking?
A. Well it was past, because it was past medical history but there was no precise dating of his cessation of smoking.
Q. Does that tell you he was not thought to be smoking at the time of his death?
A. Yes, otherwise it wouldn't be past medical history.
Q. Then the rash on his foot in March 2011 but nothing else?
A. Nothing else.
Q. You were asked to perform a coronial post mortem?
A. Yes.
Q. Can you just help the coroner, in broad terms, what the

difference is between a coronial and a forensic post mortem?
A. I suppose really it amounts to the fact that if there is any historical evidence or evidence found at the scene of death that there is a possibility of some kind of foul play, then the coroner or the police would direct that a forensic post mortem was performed rather than an ordinary coronial.
Q. Just in terms of what difference that makes, is it right that a forensic post mortem would be more -- I hesitate to say that your post mortem isn't thorough but more detailed?
A. Much more detailed, much more protracted and also associated with different standards of identification of evidence, be they blood samples, weapons, we don't do that with ordinary coronial post mortems.
Q. There would be an evidential chain to police standards?
A. Yes.
THE CORONER: Would it happen any quicker than yours did or not necessarily?
A. I think it depends really on the availability of the forensic pathologist. They are extremely pressured.
THE CORONER: Yes.
MR WASTELL: Presumably the fact that it is a coronial post mortem doesn't preclude you from identifying features

that you consider to be suspicious and asking the coroner to order a forensic post mortem?
A. No, indeed. And that has happened several times in my career.
Q. It is right, isn't it, in this case, that after completing the coronial autopsy and post mortem, at least the initial report --
A. Yes.
Q. -- you became aware that the police were now treating this death as suspicious?
A. Yes, approximately two weeks after I had completed my post mortem.
Q. The coroner indeed got in touch with you and told you that?
A. Yes, the next day I think.
Q. Clearly had you known that the police were treating it as suspicious when you were about to perform your autopsy on 14 November, you would have asked for a forensic post mortem instead?
A. Yes, I would not have undertaken the autopsy.
Q. Moving to your --
THE CORONER: Just help me with this, just so I have some idea of your own experience. How long have you been a pathologist for or how long had you been a pathologist for?

A. I was appointed consultant pathologist in 1977. I had been doing, as a trainee pathologist, post mortems since 1972, I think -- I have never actually counted them but it is in excess of 10,000 coronial post mortems, so I have seen quite a lot of material.
THE CORONER: Yes.
MR WASTELL: As we will hear, practices have clearly changed during that period --
A. Considerably.
Q. -- in respect of how you conduct the post mortem, what sampling you take and so forth. Is that right?
A. Yes, some things are changed.
Q. Yes, okay.
Let's move then to the autopsy and the post mortem report you produced. Looking at the first report, which is page 131 behind tab 26. Now, the autopsy is taking place four days after you are told that he has died?
A. Yes.
Q. You start with the external examination, height 186 centimetres, 1.86 metres, weight 93 kg, and you deduce from that a BMI of 26.9.
A. Which is marginally high, the upper limit is normally 25.
Q. The upper rate of being overweight or obese?
A. The normal rate is between 20 and 25.

Q. You found signs of paramedic treatment, did you?
A. Yes, certainly in the musculoskeletal system there was some rib fractures. These were unassociated really with any significant haemorrhage into the soft tissue and were typical of the injuries sustained during cardiopulmonary resuscitation. I also found that there was an endotracheal tube in --
Q. Sorry, just keep your voice up.
A. That was correctly located and there was vascular access by a cannula in the region of the left elbow.
Q. The antecubital fossa?
THE CORONER: Did you say there was no associated haemorrhage with the rib fractures?
A. There was very little. There is usually some, but fractures in life because there is an active circulation bleed extensively but these hadn't.
THE CORONER: So you draw a conclusion from that?
MR WASTELL: The absence of haemorrhage suggests to you it occurs post death?
A. Yes.
Q. Was there any scarring or abrasions to the body?
A. I didn't see any scars but there were several small abrasions on the left forehead, the left cheek, the bridge of the nose, the left wrist and both knees, but these abrasions were not associated with any bony

injury.
THE CORONER: From where are you reading that list?
A. This is really the first paragraph of my report, sir.
THE CORONER: Yes, sorry, so small abrasions, left forehead, left cheek.
A. Bridge of his nose, left wrist and both knees. I felt these were consistent with an agonal fall.
THE CORONER: A collapse, as it were?
A. Yes.
MR WASTELL: Indeed Dr Fegan-Earl, as the forensic pathologist, would have looked at those in greater detail.
A. He would, I am sure.
Q. Yes. Did you find any signs of third party assault?
A. No.
Q. Any evidence of offensive, defensive or restraint injuries?
A. None.
Q. In doing that, are you looking at the hands for example to see if someone has put their hands up?
A. Yes. Bruising by someone being restrained, needle puncture wounds, other than the one described already.
Q. The cannula in the elbow pit?
A. Yes.
Q. Moving to the organs. You weighed the heart, the brain,

the spleen, the liver and the kidneys, as well as the lungs.
A. Yes.
Q. Leaving the lungs aside, was anything abnormal about the weights of the other organs?
A. No, I think they were all within normal limits.
Q. How about the lungs?
A. They are considerably over their normal weight. There are various different tables of normal lung weights but they are roughly between two and three times the normal weight that you would expect.
Q. Am I right in thinking then that in a 40-year old male or so, a lung would be expected to weigh somewhere between 350 and 500 grams?
A. Yes, something like that.
Q. We will come to your internal findings about the lungs in a moment in detail but did the heavy lungs correlate to what you found on internal examination? Could you find an explanation for that?
A. In any situation where somebody has collapsed, and has undergone prolonged resuscitation attempts, I would expect to find the lungs significantly overweight.
Q. So it is not an unusual finding in somebody who has had prolonged CPR?
A. No. It is the expected finding.

Q. Turning then to your internal examination, aside from the rib from a are that you have already referred us to, did you find anything unusual or of interest in the musculoskeletal system?
A. No.
Q. What about the central nervous system?
A. No, the scalp, the skull and the lining of the brain all appeared normal. On sectioning of the brain, that also appeared normal.
Q. And am I right that if you had seen, for example, a brain haemorrhage or some sort of brain pathology leading to death, you would expect on internal examination of the brain to see the pathology there?
A. Yes, you would.
Q. You then looked at the cardiovascular system. Just tell the coroner what you found there?
A. The sack within which the heart lies appeared normal.
The actual substance of the heart muscle and the size of the chambers and the four main valves again appeared normal.
There was mild disease of the right coronary artery, but not I don't think of any clinical significance whatsoever.
The other two coronary arteries appeared normal. What was extremely unusual were the actual origins

of the coronary arteries from the aorta. If you open up the aorta, you open up the aortic valve, it has three cusps. A left one, a right one and a non-coronary one. Immediately behind the cusp there is a small sinus and normally the left coronary artery arises from the middle of that sinus and the right coronary artery arises from the middle of the right coronary sinus. In Mr Perepilichnyy's case, the origin of the left coronary artery was situated above the junction of the left and right coronary cusp.
The potential significance of that is that in some people the two coronary arteries arise from the same sinus and this may be associated with an anomalous course of the artery around the heart. Normally the coronary arteries run around the aorta and the pulmonary artery, the two big vessels at the top of the heart. In people where you get origin from the same sinus one of the coronary arteries may run between the aorta and the pulmonary artery and these people are at risk from sudden death.
I felt at the end of the post mortem, since basically I had not found anything other than this unusual situation that this possibly could explain his sudden death, that he had an anomalous coronary artery anatomy and that this might have led to his death from

a cardiac arrhythmia.

In cases like this we have a set protocol, discussed and agreed with a coroner, about how to progress the case further.

Q. When you say "cases like this", is this cases of suspected sudden cardiac death?

A. Yes.

Q. The steps you took in respect of the heart, was to fix the heart and send it to a cardiac pathologist, Dr, now Professor, Sheppard?

A. Yes.

Q. Just explain to the coroner, it may be relevant later on in the Inquest, what does fixing the heart mean?

A. Basically what you try to do is the heart can get distorted during the course of what is a fairly minimal dissection on my part in cases like this. We try to reconstruct it, using cotton wool, to maintain the shape and size of the chambers and then we immerse the whole heart in a form of saline which stops the material that the heart is made of decaying or degenerating.

Q. Is the idea of that then to maintain the form of the heart and to stop any post mortem changes continuing?

A. Yes.

Q. So that when the cardiac pathologist looks at it, they are looking at it as close to it would have been to

A. It is the mouth down to the anus, really.

Q. There was nothing unusual in the mouth, tongue or oesophagus?

A. No.

Q. The stomach was normal?

A. Yes.

Q. You dissected the stomach?

A. Yes.

Q. And looked inside?

A. Yes.

Q. And you found stomach contents?

A. Yes.

Q. What did you see?

A. Well, I have described as partially digested food and bile, both of which are normal findings in a stomach.

Q. Was there any tablet residue?

A. No.

Q. Did you open beyond the stomach, so did you open a little bit into the small intestine?

A. Yes, you go through to the duodenum, because there are a couple of structures there that you need to look at. They were normal.

Q. Can you recall anything unusual about the stomach contents?

A. No.

death?

A. Yes.

The only other comment that is perhaps relevant to make is that if you think that you are going to send a heart on to Dr Sheppard, as she was then, your dissection is minimal, it is much easier for a pathologist to examine a heart that has not been very radically dissected. So it is always a compromise, you minimise your own dissection of the heart to enable the task of Dr Sheppard to be easier and more thorough.

Q. Aside from the apparently or potentially anomalous origin of the coronary arteries, am I right there was nothing else in the heart that would account for the cause of death?

A. Nothing.

Q. You mentioned that you did not find anything else of significance, so there was nothing, just going through the rest of your report of significance in the genitourinary system?

A. No.

Q. Nothing in the reticular endothelial system or endocrine system?

A. No.

Q. In respect of the alimentary system, that is the oesophagus down to the stomach and the intestines?

Q. Any particular odour?

A. No.

Q. Colour?

A. No.

Q. Texture?

A. No.

Q. If you had found anything unusual, would you have recorded it?

A. Yes. I would.

Q. We will come to what happened to the stomach contents a little bit later, I just want to complete your findings, if I may.

In respect of the respiratory system on internal examination, what did you find there?

A. The lungs were heavy, as we have discussed, and they were congested, they were rather darker in colour than usual, which usually implies that they are stuffed with blood, the vessels are abnormally large, and that on light pressure to the surface, the cut surface of the lung, a frothy fluid exuded, we call oedema. It is just accumulation of fluid within the tissues.

Q. Generalised congestion and oedema; is that right?

A. Yes.

Q. How severe was it?

A. I think the remarks we have made about the weight of the

lungs meant that this was quite is a significant finding, they were heavy.
Q. Was there anything unusual about the pulmonary arteries or pleural cavities?
A. No.
Q. Any sign of thrombosis?
A. No.
Q. You would exclude pulmonary embolism, would you?
A. I can.
Q. In terms of the finding of oedema and congestion of this severity, you have already told the coroner that the weight of the lungs was consistent with prolonged resuscitation. Is it right that the oedema and congestion are therefore consistent with prolonged resuscitation?
A. Yes.
Q. And also a range of conditions, they are non-specific?
A. They are non-specific findings.
Q. Including sudden cardiac death?
A. Yes.
Q. As well as being non-specific, can the oedema be caused by acute heart failure?
A. Yes.
Q. The congestion, is that caused, again, although non-specific, potentially by acute heart failure?

A. Yes.
Q. Just in broad terms, I think a point drawn out from the joint statement, in cases where you don't have brain death but the body continuing, for example a vegetative state, the terminal event is always the heart failing?
A. Well, the heart always stops, yes.
Q. This is an extremely common finding at post mortem?
A. It is, yes.
Q. Hence it not being specific to any particular pathology?
A. No.
Q. Again, it doesn't tell you, does it, what the cause of the heart failure was?
A. No.
Q. Was there any evidence of a major haemorrhage in the lungs?
A. A major haemorrhage?
Q. Haemorrhage in the lungs.
A. No, it was just a generalised congestion. The finding of blood in the lungs was a later finding on microscopic examination, but there was no major haemorrhage.
Q. Your conclusion as to cause of death at that time is simply that it is under investigation?
A. Yes.
Q. You send the heart to Professor Sheppard, or Dr Sheppard as she was?

A. Yes.
Q. You then took some histology --
A. Yes.
Q. -- some samples for histology?
A. Yes.
Q. Is this part of the protocol that you are following in cases of sudden cardiac death?
A. Yes, it is. It is.
Not every case of sudden cardiac death, cases where it is a suspected cardiac death with no really obvious cause.
Q. So unexplained, I am sorry, at post mortem?
A. Yes.
Q. If you open up and find a reason for the sudden cardiac death then there is no need to perform these extra --
A. Then histology would not be undertaken routinely.
Q. Here you have opened up, you have found no identifiable cause, you have found a potential in the origin of the coronary arteries.
A. Yes.
Q. As a result you are following through your protocols, that means taking samples for histology from the kidney, liver?
A. Yes.
Q. The cerebrum, cerebellum and meninges, that's parts of

the brain?
A. Yes.
Q. The myocardium, that's the heart muscle?
A. The heart muscle.
Q. And the lungs?
A. Yes.
Q. You are also taking samples for toxicology?
A. Yes.
Q. Why do you do that?
A. Because it is impossible reliably to exclude poisoning as a cause of death, most poisons produce entirely non-specific findings at death, so we routinely take samples that allow basic toxicological screening.
Q. Those samples are what?
A. The routine is to take blood, femoral venous blood, in an unpreserved and a preserved state and similar remarks for urine, unpreserved and preserved.
Q. Is that what you did in this case?
A. Yes.
Q. Two samples of blood, two samples of urine?
A. Yes.
Q. The preservation, how do you achieve that?
A. I am not sure what the preservative is that is used now, but it is one that the toxicologists have asked us to use. And we provide them with the samples that they

want.
Q. If we then move to your supplementary report, your expanded report, page 120, behind tab 3. 120 is the second page, it may be helpful simply to take it out and turn it round.
A. Sorry, do I have the right bundle here?
Q. It is core bundle 1, so it is the larger bundle.
A. I am looking at tab 3?
Q. No, tab 23.
A. 23, I beg your pardon.
Q. I am sorry. Behind tab 23 you should see your second report.
A. Yes.
Q. It is page 120 that I am interested in, second page.
A. Yes.
Q. Is it right that you simply have added the supplementary findings from your histology?
A. Yes.
Q. On 22 November?
A. Yes.
Q. So some eight days later?
A. Yes.
Q. Is that before or after you had found out the further information that the police were treating the death as suspicious?

A. Before.
Q. Nothing found on histology aside from the lungs. Are you looking through a microscope at the slides?
A. Yes.
Q. Nothing in the brain?
A. No.
Q. Nothing in the kidney or liver?
A. No.
Q. Nothing in the heart muscle?
A. No.
Q. In respect of the lungs, what did you find?
A. I found that both lungs showed similar changes, and there was congestion with microscopic evidence of intra-alveolar haemorrhage, that is bleeding into the air spaces.
Q. Was that significant, the bleeding into the air spaces?
A. Given the circumstances of his death and the resuscitation, it is what I would expect to find.
Q. It is consistent with resuscitation and particularly --
A. Acute cardiac failure and resuscitation.
Q. Can I just ask you, the 46 minutes between the paramedics attending and death being certified, is that a particularly prolonged resuscitation in your experience?
A. Yes, I think it is. I think the ambulance people are

usually directed to limit CPR to about 30 minutes, and if nothing has been achieved after 30 minutes, it is so unlikely that anything can be achieved that they should stop.
Q. Yes.
A. In fact, in this instance, they went on longer than 30 minutes.
Q. Yes, and we have heard from them last week about that. You found oedema and intra-alveolar haemorrhage?
A. Yes.
Q. You are not detecting a primary pathology for the haemorrhage are you, as you say it is consistent with resuscitation as you would expect?
A. Yes.
Q. You also found in the occasional bronchioles some material?
A. Yes.
Q. First of all, what are the occasional bronchioles?
A. Well, bronchioles are very small airways. There were one or two fragments of vegetable material and partially digested voluntary muscle -- meat in other words, they were gastric contents.
Q. Nothing in the major airways?
A. No, I didn't see anything. This was only something I could see down a microscope.

Q. What is that in your opinion consistent with?
A. In the process of cardiac arrest, vomiting is very common. And in patients who are in a collapsed situation, where consciousness is impaired, the normal defence mechanisms to aspiration of foreign materials of any kind is lost or impaired and it is quite common to see gastric contents in the airways.
I think that they are so irritant that they produce inflammatory changes in the airways very quickly, within a matter of minutes. No such reaction was present in this case, so my assumption is that this aspiration occurred at or around the time of death/collapse.
Q. We have the results of the histology, the other aspects of your protocol following was the samples of blood and urine. What happened to those?
A. Well, in the normal course of events, the samples would be labelled with a forename, a surname, a date of birth of the deceased, the date the specimens were taken and the nature of the specimen.
Q. Can I pause you there, do you permanently write those labels?
A. No, I asked our technicians, St Peter's are very capable and very experienced, "Can we get some blood and urine?"
They would take the samples, they wouldn't normally concern me, unless they had a problem getting them. In

which case I would go and assist and offer advice and try alternative methods if required, but that was not the case here. They would then --

Q. Just one moment. They are supposed to put those bits of data on the samples, they may not always follow that, presumably, the date of birth, surname et cetera?

A. I don't check each sample personally but knowing their quality and experience, I would be very surprised if they didn't fulfil those criteria.

Q. I interrupted you, I am so sorry, but we were talking about what you do with those samples or what you did in this case with those samples. What happened to them?

A. They would be stored in a 4-degree fridge and the coroner's officer responsible for the case would then arrange for their onward transport to the toxicology laboratory that we normally use, which at that time was an organisation called ROAR.

Q. Did that in fact happen in this case?

A. I am not sure, but I suspect not.

Q. You signed off your supplementary report recording that the heart had been examined in detail by Dr Sheppard?

A. Yes.

Q. The broad conclusion of her report macroscopically and microscopically normal?

A. Yes.

Q. You recorded the further historical information provided and that a second forensic post mortem, by which you mean the first forensic post mortem, the second post mortem?

A. The second post mortem, the first forensic one.

Q. Had been performed.

Then you signed off your report on 4 May 2013?

A. Yes.

Q. Why so far after the second post mortem?

A. My normal practice is in cases where there is ongoing investigation is to issue a temporary report, saying the matter is under investigation. I then normally put the temporary report in a pending file and as and when in this case the toxicology reports get back to me I would take my incomplete report out, complete it and draw whatever conclusions were possible from the completed investigation.

In fact in this case the toxicology reports never came back to me, so my second report stayed in my file until I was asked to produce it --

Q. Yes.

A. -- that is an oversight on my part.

Q. You weren't involved in the second forensic post mortem, in the sense of performing as a pathologist, were you?

A. No.

Q. You did attend the start; is that right?

A. Yes, and I was able to produce for Dr Fegan-Earl a copy of my report --

Q. Yes.

A. -- and the information from Dr Sheppard.

I had a lot of commitments myself, so I went back to them, knowing full well that Dr Fegan-Earl's investigation would take a protracted length of time.

Q. In terms of the samples, were you involved in transmitting the samples of urine, blood and any tissue samples to Dr Fegan-Earl?

A. No.

Q. Were you aware of whether or not they arrived with the body? Are you aware of that?

A. I am not aware of that.

Q. I dealt with the urine and the blood, but in terms of the tissue samples, is it right that you take tissue samples and create blocks in paraffin with them?

A. Paraffin wax, yes. Then they can be used to take very thin sections which can be stained and then we can look at down a microscope.

Q. At the end of looking at your histology, you would have both the slides, which are the slivers of tissue taken from the blocks and the blocks?

A. Yes, the blocks would be retained within the histology

department that processes them. They would give to me just the slides to examine down the microscope.

Q. Yes, you are not again involved in what happens to the histology blocks --

A. No.

Q. -- thereafter or how they get transmitted to the forensic pathologist?

A. They would routinely be stored within the pathology department at St Peter's Hospital.

Q. Let me then turn to the stomach contents?

A. Yes.

Q. You have described nothing unusual about it, what did you do with the stomach contents?

A. I don't remember specifically how much material was there. What we need to do is to look at the lining of the stomach in some detail.

Q. The lining?

A. The lining of the stomach.

Q. How do you do that?

A. You have to clean off the gastric content. If it is small quantities you can wipe it with a sponge. If there is anything adherent you would rinse it with a hose.

Q. What liquid are you using, either in the case of a sponge or hosing it?

A. Tap water.
Q. Just tap water?
A. Yes.
Q. Can you help us as to whether it was adherent in this case and whether you used the hose or not?
A. I can't remember but it presented no unusual features.
Q. How do you physically --
THE CORONER: The stomach lining, when you say it presented, do you mean the stomach lining?
A. No, the stomach contents.
THE CORONER: The contents. Hold on.
MR WASTELL: How do you physically remove it, is that using the hose or the sponge?
A. It can be either, it depends on how adherent they are.
Q. There is no instrument by which you are scraping it out?
A. No, no.
Q. What did you do with the stomach contents thereafter?
A. Again, I can't remember. If they are present in enormous quantities, I mean they would be disposed of down the sluice.
Q. Yes.
A. If they are only in small quantities that can be wiped off, they would be returned with the stomach back into the body.
Q. Why, if you are interested, pursuant to this protocol,

in potential toxicology tests don't you retain the stomach contents?
A. I mean the background of this is that probably about the first ten years of my involvement with coronial post mortems, a routine toxicology screen would involve retention of gastric content where it is present.
There was a discussion between the then coroner and the pathologists whereby the coroner felt that he had been looking at so many cases of toxicology where examination of the gastric content made no contribution to the final cause of death, over and above that that could be determined by examination of the toxicology of the blood and of urine.
It was deemed that we needn't take gastric contents as a routine, unless we felt there was something odd and unusual about them. If you saw many tablets or tablet residues or anything of a peculiar nature, we were then perfectly at liberty to take samples of gastric content and get them analysed.
Q. Just to check I am clear about that, a protocol was developed between pathologists and the coroner in that area?
A. Yes.
Q. Pursuant to which, as a matter of standard practice, stomach contents wasn't kept or sampled for toxicology?

A. No, that was normal practice.
Q. But with a caveat that if you saw something unusual, you would?
A. Yes.
Q. Can we therefore deduce, again you didn't see anything unusual in the stomach contents in this case?
A. Yes.
Q. Having handed over the body, literally, to the forensic pathologist, at that stage you can simply say as to cause of death it is under investigation, correct?
A. Yes.
Q. You would then defer to him as to opinion as to the cause of death?
A. Yes.
Q. From your perspective, all you can say, is this right, that you found no clear observable pathological process to account for his death?
A. Yes.
Q. But equally, no evidence of third-party involvement?
A. No.
MR WASTELL: Thank you.
I have no further questions, if you wait there there may be some questions.

Questions from MR MOXON BROWNE
MR MOXON BROWNE: Dr Ratcliffe, you record somewhat baldly perhaps in your report that Mr Perepilichnyy was overweight, I think you explained.
A. Marginally. The rules are quite simple, 20 to 25 is normal, above 25 is overweight or obese, he was mildly overweight.
Q. I understand that and you have explained, it is just over the limit?
A. Yes.
Q. I notice that Dr Fegan-Earl has described him as "well nourished" and the coroner may in due course need to make some record, so somewhere between well nourished and?
A. Technically he is overweight, if you go on the basis of BMI classification.
Q. Yes.
There were two findings that struck you at autopsy, two possibly significant findings.
One was very marked oedema in the lungs.
The other was what you thought was an anomalous position of the arteries.
They were really the two things?
A. Yes, they were.
Q. If we can just deal with the lungs first. You have

mentioned that that is a non-specific finding?
A. Yes.
Q. It could occur before death, it could occur after death, as a result of resuscitation?
A. Yes.
Q. If we look at the period before death, it could be due, for example, to the development of atrial fibrillation leading then to ventricular fibrillation, so the heart is working inefficiently for a short time?
A. That is possible.
Q. It could be due to what is conventionally called "heart failure", just the inability of the heart to function properly?
A. Yes.
Q. What it does indicate, if it is not a post mortem artefact, is that some trouble started a little while before death. It is not a case of a guillotine coming down, otherwise you wouldn't see that oedema, if it is not a post mortem artefact?
A. It is possible if the death was arrhythmic in nature that this would lead to heart failure and the accumulation of fluid in the lungs.
Q. I think we are agreed about this, but I just want to be quite clear. If it is not a post mortem artefact, and I appreciate it could be, but if it is not, it is

an indication that the heart wasn't functioning properly for a time?
A. I mean I would question the use of the word "artefact". I mean it is a condition that occurs at and around the time of death. I think what may be artefactual is the presence of the microscopic haemorrhage, which is possibly due in part to the trauma involved in resuscitation. I mean the process is sufficiently traumatic to fracture ribs, so I think that might be true.
Q. What we have here is a very marked oedema I think, really remarkable --
THE CORONER: Sorry, is it really remarkable?
A. It is what I would expect to see in someone who had died in the circumstances that Mr Perepilichnyy did.
MR MOXON BROWNE: As a result of something that happened after his death, are you talking about?
A. I am not sure if it is after or at the time of. I don't think I can separate the two.
Q. Do you agree or not that it postulates a period, not instantaneously but a period during which the heart was not operating efficiently?
A. I think that is quite likely.
Q. Thank you.
Before we leave the lungs, you found meat, chicken,

or something like that, and also vegetable material, in tiny quantities?
A. Yes.
Q. You said there is no vital reaction, that is what you said in your report, but I think you qualified that by saying something about a period of minutes? Can you just explain what is the evidence of no vital reaction?
A. If you look down the microscope, aspiration of gastric content occurring in life will provoke an inflammatory response characterised by, amongst other things, the accumulation of acute inflammatory cells in response to the material.
Q. That I understand --
A. That was not present.
Q. -- but I think you talked of a period of time, it is difficult to hear you always but --
A. Sorry.
Q. -- you mentioned three or four minutes before you get the inflammatory response?
A. I think it is often a little longer than that, perhaps 10 or 15 before you get congestion and the invasion by acute inflammatory cells.
Q. He could have had, to put it rather bluntly, a very violent coughing or vomiting incident as long as 10 minutes before death?

A. It is possible.
Q. Yes.
You have described why the stomach contents were removed and you have answered a question that I was wondering about, which is whether you purged the stomach with some chemical liquid, but the answer to that is no?
A. No.
Q. But you did wash it out?
A. I can't remember. I would have either wiped it with a sponge or rinsed it with a hose.
Q. Hmm, but either way there wasn't very much left, I am assuming?
A. I can't remember.
Q. If we could just look at a couple of points that you were able to agree with Dr Fegan-Earl in your joint statement, do you remember that you both signed?
A. Yes.
Q. I don't have a paginated reference for this but I believe that it is in bundle 3, page 97 that it starts.
MR WASTELL: Tab 97.
MR MOXON BROWNE: Tab 97. Yes, I never put tabs in my bundles so I was remiss.
A. Yes, starting on page 864.
Q. Yes, I am going to take to you paragraph numbers if that

is all right. Can we look at paragraph 11.
A. Okay.
Q. 868 I am told. Do you have that?
A. I have paragraph 11 here, yes.
Q. I just want to look at the statement that you agreed with Dr Fegan-Earl:
"It is impossible to state definitively whether or not traces of any substances present in the stomach contents could have been lost, given that the stomach had already been opened. That said, the food was found in a partially-digested state."
That is based on your observation rather than Dr Fegan-Earl's?
A. No, I think Dr Fegan-Earl must have found some kind of gastric content because the gastric content was submitted for analysis.
Q. I appreciate that. That was a sort of joint observation, was it?
A. Yes.
Q. "Which would imply that there had been a mixing of the contents of the stomach, which likely represented a relatively uniform medium for analysis."
A. Yes.
Q. If we can just sort of translate that, if I can put it like that. You are saying that because the stomach

content was all mixed around, a sample from it would have a reasonable chance of finding anything that had been ingested, that is the point you are making?
A. Yes.
Q. If, for example, three or four hours before his death, Mr Perepilichnyy had consumed a fairly substantial amount of sorrel, a vegetable, that is the sort of thing that you would have a good chance of finding?
A. Yes, I would think so.
Q. In your very long experience as a pathologist, are you sometimes asked to give opinions or do you investigate questions of how quickly food moves through the digestive tract?
A. It is quite unusual in my experience.
Q. What is, sorry?
A. To be questioned about that.
Q. To be questioned on that, yes. What I want to find out from you, whether it is sensible to ask you any questions about the stomach contents moving into the upper part of the digestive tract. Do you think that is likely after a fewer hours?
A. Yes, I think it is very likely.
Q. If there was nothing to be found in the stomach in relation to the last meal, you would probably find it --
A. I think there would be a reasonable chance of that, yes.

Q. Yes, and if we go all the way down to the ileum, which I think is the last section, that is I think a very long organ, 10 or more metres long?
A. Yes.
Q. If we think about the end of that, that is to say the very last section of the ileum, what does that represent in terms of the history of the ingestion of food?
A. Oh, it is impossible to say. Transit times really are so variable and influenced by so many different factors, nature of the food, whether it is take with -- there are so many different factors. Transit times are extremely difficult to --
Q. I do understand that. Can you say whether it is likely that that last meal would have found its way to the end of the ileum?
A. No.
Q. No.
THE CORONER: You cannot say or it is not likely.
A. I can't say.
MR MOXON BROWNE: Thank you.
I think you make that point in answer to a question at paragraph 12, where you say:
"It is difficult to comment as to whether there was a toxin in the stomach at the time of death which was not present in the sample taken by Dr Fegan-Earl, the

passage of food through the gastrointestinal tract is a variable phenomenon, dependent on the volume of food, nature of food ..."
You go on:
"It was for this reason that additional samples of the gastrointestinal tract further down from the stomach were taken to allow for optimum analysis."
What you are saying is if you don't find anything relevant in the stomach, you might find it further down?
A. Yes.
Q. Yes.
When you say, as you have done, that the finding of oedema is non-specific, you have identified that it could be something that happens after death, it could be something that happens before death, it can of course be something which is caused by a toxin?
A. Yes.
Q. I think you say, paragraph 22, of your agreed statement:
"In the absence of an obvious pathology to explain the development of heart failure and the absence of a toxin, the findings are non-specific."
You simply cannot say?
A. Yes.
Q. Then dealing with the sudden arrhythmic death at paragraph 30, you are explaining what is meant by

a diagnosis of complete exclusion --
A. Yes.
Q. -- as you understand it.
The point is that because by definition a sudden arrhythmic death leaves no trace, it is not a finding you can make unless and until all other possible explanations have been excluded?
A. Yes.
Q. You mention a full diagnostic triage excluding trauma, toxicological causes, causes outwith the heart and definite structural and observable changes in the heart then the cause is deemed to be SADS?
A. Yes.
Q. Of course one of the factors that may negative the SADS finding is evidence of an unnatural death, if you find a body in a bin bag in a skip then you are not going to say it is that, are you?
A. No.
Q. Similarly if the deceased's body is in perfect condition, there is absolutely no sign of injury, the toxicology is clear, but they leave a note for the coroner saying I have decided to end my life, you wouldn't be saying SADS, would you?
A. No, if they were toxicological corroboration of the poisoning --

Q. No, that is specifically not what I am putting. I am putting that if you found a body in a bin bag, or indeed a person in their bed in the morning, in the latter case with a note to the coroner saying they had decided to end their life, that you would hesitate to conclude it was a SADS death, even if there was no findings at autopsy and clear toxicology?
A. If the contents of the note were accurate, I would be very disappointed not to find a provable cause of death.
Q. Well, yes, I understand that, but you see the point I am putting, that if there is a pretty clear indication that the cause --
A. I think in any case, before reaching a conclusion about cause of death you have to consider the totality of evidence --
Q. Yes.
A. -- that is before you, not just the isolated findings of a post mortem. You are reliant on history and various other factors.
Q. That is a point that both you and Dr Fegan-Earl make?
A. Yes, and I think it is a very important one to make.
Q. We will no doubt have an opportunity to ask him questions about that.
I think you were both asked whether apart from an undetected poison or what we have described as

a channelopathy are there any other causes of death that leave no trace. I think the only solid one you come up with is epilepsy; is that right?
A. Epilepsy is one we know, for instance, that some diabetics just appear to die without any obvious cause.
Q. Yes.
A. Yes, it is not entirely excluded.
Q. No, I am sure not. I think epilepsy is the only condition that you have identified?
A. It is perhaps the commonest one that we see in day-to-day practice.
Q. Yes.
On the question of the anomalous artery, you came to a conclusion about that, you thought there might be something unusual there so you followed it exactly, if I may say so, what you should do which is to send the heart off to Dr Sheppard?
A. Yes.
Q. Who is -- she won't mind me saying, a preeminent expert in the field of cardiac pathology?
A. Absolutely.
Q. She came back and said there is nothing wrong with the heart?
A. Yes.
Q. I just want to be quite clear, although you did have

an opinion about it, you now entirely accept her view about that?
A. Entirely. I did in my report say it was probable.
Q. Yes. You did, if I may say so, you appear to have done exactly the right thing but I just want -- there is no reservation in your mind, you are not harbouring some private view, you are quite satisfied that whatever the cause of the death here was, it was nothing to do with that?
A. Yes.
MR MOXON BROWNE: Thank you.
Questions from MR STRAW
MR STRAW: Just on that point about the heart being sent to Dr Sheppard, presumably another reason for you to do that was she would be expected to have access to a range of equipment and testing which you yourself wouldn't be able to apply to that heart?
A. I think most of her examination involved simple techniques of dissection and examination down the microscope, but I have to respect her opinions of both the naked eye and the microscopic changes associated with -- she is far more of an expert than I am.
Q. You have described packaging and preserving the heart --
A. Yes.
Q. -- before it is sent off to Dr Sheppard.

A. Yes.
Q. Presumably that included the area that you had some concern about, the anomalous artery area?
A. Yes.
Q. She would have examined that area?
A. She would indeed.
Q. The stomach contents, you have described partially digested food in the stomach contents, can you give us any more detail about that?
A. No.
Q. Did you perform any microscopic analysis of it?
A. No.
Q. Or indeed any testing of the stomach contents?
A. No. No tests were done by me.
Q. Just to be clear, I was not quite clear about your answer, did you sample the stomach contents before they were disposed of?
A. No.
Q. Could you turn, please, to bundle 1, so expert bundle 1. Tab 24 and page 122, top right.
A. Yes.
Q. At the very bottom of that page you say the stomach contents were not sampled and no stomach contents were left in situ.
A. Not knowingly by me, no.

THE CORONER: Not what, sorry?
A. Not knowingly by me.
MR STRAW: Given that, is it possible that a substance that was in his stomach at the time of death was disposed of before it came to the time of Dr Fegan-Earl's post mortem?
A. Yes.
Q. You mentioned that during the post mortem, you didn't notice any obvious odour?
A. No.
Q. Have you any experience of conducting post mortems on those who have died from exposure to cyanide?
A. No. Only -- well the exception, I have done a number of post mortems on people who have died as a result of inhalation of smoke fumes, domestic fires. And they inhale, amongst other things, carbon monoxide and some cyanides generated by the combustion of modern synthetic furniture materials and so on.
Q. But in amongst all of the other things?
A. In amongst a lot of other things, yes. As pure sort of cyanide ingestion, the answer is no.
Q. The duodenum, as I understand it the first part of the intestine, did you open that partially?
A. Yes.
Q. Did you also dispose of any contents of that at the same

time as disposing of the stomach contents?
A. I can't remember. I would certainly have cleared it far enough to examine the structure that we are always interested in, that is the ampulla of Vater, particularly in someone who had gallstones. So I would have probably wiped some of it clear anyway.
Q. Finally, coming to your conclusion. I am looking at the conclusion of the joint report that you produced with Dr Fegan-Earl, and if you need to see it, it is in bundle 2 and tab 97.
A. Yes.
Q. Page 875. Is it bundle 3? Sorry, I am told it is bundle 3.
A. Yes, 875, I have it, "Overall conclusions".
Q. Yes. Moving on to paragraphs 48 and 49, were you by this stage aware that concerns had been raised that Mr Perepilichnyy may have been poisoned by --
A. Yes.
Q. -- the Russian state or others with access to rare or sophisticated poisons?
A. Yes.
Q. Was that relevant to your conclusion?
A. Yes, it has to be.
Q. The easiest way may be simply to read out the conclusion. Is it right:

"The only conclusion that can be made if there is an undetected poison is that the cause of death is unascertained, and that could only be refined if the poison was detected and specified or if poison could be completely excluded, whereupon sudden adult death syndrome could be posited, with the circumstances as they are it is our opinion it should remain unascertained."
A. Yes, that is true.
MR STRAW: Thank you very much.
Questions from MS BARTON
MS BARTON: Good morning.
May I just ask you one or two questions arising from what you have indicated already. You have conducted several thousand autopsies?
A. Yes.
Q. There was nothing unusual at all about this one in terms of your findings?
A. No.
Q. Apart from the coronary arteries?
A. That was the only thing that I thought possibly might be relevant to death at the time of completing.
Q. Specifically I think you have referred to two matters, first of all had you believed --
A. To two what, sorry?

Q. Two matters you have referred to in the course of your evidence.

One is them is that had you identified any features which you believed to be suspicious, you could have referred the death to a forensic post mortem through the coroner?

A. Yes.

Q. That is of your own volition?

A. Yes.

Q. Yes, and you didn't see the need to do that in this case?

A. No.

Q. Secondly, had you seen anything unusual about the stomach contents, you could and would have taken a sample of those contents?

A. Yes.

Q. The only reason you didn't is because there was a protocol in place between you and the coroner, or pathologist and the coroner, which dictated that you would not take such a sample if there was nothing unusual about the contents. Is that correct?

A. Yes, that's correct.

Q. What it comes down to is this, that at the conclusion of your first autopsy, you saw nothing suspicious at all about the death of this individual?

A. That is true.

Q. Can I just ask you this. In terms of the involvement of the police in the post mortem, it is a matter for the pathologist, isn't it, to decide what samples to take and what tests to carry out?

A. The police weren't involved in the autopsy that I conducted. I think that is a question perhaps would be better addressed to Dr Fegan-Earl.

Q. You have answered that question. In the autopsy you conducted, you were not directed in any way by the police?

A. No.

MS BARTON: Thank you.

Questions from MR BEGGS

MR BEGGS: You were taken to one of your conclusions, indeed the final conclusion, inevitably counsel tend to pick and choose the conclusions they want to emphasise but just so that we have a balance of your conclusions, could you look at paragraph 45 on page 875.

A. Yes.

Q. You make the point that pathological findings must be considered in context.

A. Yes.

Q. Yes, and then you say this:

"The conclusions that can be drawn from the post

mortem examination are that there was no pathologically observable evidence of third party assault or restraint."

Just pausing there, earlier in your report, as the learned coroner sees, you have explained how you came to that conclusion?

A. Yes.

Q. No pathological evidence of injection marks, correct?

A. True.

Q. Then no definitive evidence of natural disease of a severity that could explain the death?

A. Yes.

Q. The other conclusion in this section I wanted to confirm with you was that at paragraph 45, please. This conclusion is based on an assumption that the deceased was not poisoned, so I make clear that --

THE CORONER: Where are you reading from?

MR BEGGS: 48, sorry, my fault, I misspoke. Paragraph 48 I am sorry.

A. 48?

Q. Yes, I will repeat that so there is no unfairness.

This based on the assumption that the deceased was not poisoned, so if that assumption is in place, you are asked: what is your conclusion as to the likely cause? Then you say the death would be attributed to sudden

adult death syndrome?

A. Yes.

MR BEGGS: Thank you very much.

Questions from THE CORONER

THE CORONER: Can you explain, while we are on the conclusions, if you go to the very last one that you were asked about, I want to try and understand what that means, do you have that one? Page 874 for me at the top right.

A. 874?

THE CORONER: Yes.

A. Specifically where?

THE CORONER: You have that, the first sentence:

"The only conclusion that can be made if there is an undetected poison is that the cause of death is unascertained."

On one view, if there is an undetected poison -- no, you are not with me?

A. 876.

THE CORONER: I have 874 at the top of mine, but not to worry, the last page?

A. Yes, the last page.

THE CORONER: Do you have that? Do you see the first sentence:

"The only conclusion that can be made if there

an undetected poison is that the cause of death is unascertained."

A. Yes.

THE CORONER: If there is an undetected poison, do you follow, I am just struggling a bit with this, you answer it whole, then it goes on:

"... that could only be refined if a poison was detected and specified or if poisoning could be completely excluded, whereupon sudden adult death syndrome could be posited."

Then you say:

"With the circumstances as they are, it is our opinion that it should remain ascertained ..."

What circumstances are you taking into account? Are you just looking at the post mortem findings?

A. No, no, we are looking at the whole picture, sir.

THE CORONER: Right, so what else -- do you know --

A. Well, the comments, the sort of circumstantial evidence raised by I think Hermitage that --

THE CORONER: Have you gone into all of that?

A. It has been presented to me, yes, sir.

THE CORONER: How much of that have you seen then?

A. Oh, what I have read in the papers --

THE CORONER: Oh dear.

A. Not a lot, sir.

THE CORONER: Not the lot or not a lot.

A. Not a lot, but you cannot ignore it.

THE CORONER: That is just really what I want to explore with you.

The material that Hermitage or some of the material that Hermitage have presented, you have taken account of?

A. I mean I have heard in the background that one possible scenario is that Mr Perepilichnyy was poisoned.

THE CORONER: No, that is right. That is what you were dealing with, with here, but that is rather different to saying one possible scenario is he is poisoned, so when you say with the circumstances as they are, so we understand what you mean, do you mean you have come to a view of your own about such things as you have heard of the background material?

A. I have been presented with quite a lot of evidence in the expert reports that I have had to read, I have read the evidence of the toxicologists that have been involved in the case, all presented to me in a bundle. I have read material in the paper, I read the paper on a daily basis.

THE CORONER: Right.

A. There is clearly considerable doubt that has been raised, raising the possibility that Mr Perepilichnyy's death was not a natural one. I think as pathologists we are obliged to look at the whole picture, so not just the pathological findings in isolation.

THE CORONER: This opinion is because of your view of such of the other material as you have been presented with?

A. It is not my view. It has been presented to me quite clearly that there is a possibility that Mr Perepilichnyy did not meet a natural death. That has been presented to me in several ways, mainly through the bundles which I have been presented with.

I mean the extent of the toxicological investigation in this case has been enormous and that has all in summary form been presented to me.

THE CORONER: I mean if matters are disputed, or there is an issue as to what inference if any should be drawn from an agreed matter, do you come to a view -- I mean have you reached your own view about that? Because without knowing, as it were, exactly what you have seen and what your views on -- I might have a different, I might come to a different view in the end and it might be that if I came to a different view, as it were, my conclusion would be different from yours but it is a bit difficult to know without having gone through everything you have been shown and knowing ... do you see the problem?

A. I see your problem, sir.

THE CORONER: I don't know everything you have seen and every conclusion you have come to.

A. It is all in the bundles that I have been --

THE CORONER: There is lots of materials in bundles, that is a slightly different question. (Pause)

There we are, that is your opinion anyway in the last paragraph?

A. Yes.

MR WASTELL: Sir, it may be I can assist on this.

THE CORONER: Yes.

Further questions from MR WASTELL

MR WASTELL: Dr Ratcliffe, is it right that if there is a live possibility of another cause of death, other than sudden adult death syndrome or sudden arrhythmic death syndrome, as I think it is called, is that right?

A. Yes, they are synonymous.

Q. If there is another live possibility, then you cannot reach the conclusion as a pathologist of sudden adult death syndrome?

A. By definition SADS is a diagnosis of exclusion.

THE CORONER: Yes.

MR WASTELL: Yes.

As to whether there is a possibility of poisoning or the magnitude or the chance that there was poisoning,

you would defer to the coroner who listens to all the live evidence and hears evidence in court and indeed written evidence?
A. Absolutely.
MR WASTELL: Thank you, I have no further questions.
MR STRAW: Sorry to rise, there is one question if I may which arises out of the issue which you raised.
THE CORONER: One?
MR STRAW: One question, yes.
Further questions from MR STRAW
MR STRAW: On that point, Dr Ratcliffe, in expressing your opinion about whether the cause of death is unascertained, and in particular whether the possibility of poisoning can be completely excluded, is the evidence of the toxicologists that you have seen, such as Professor Ferner, relevant?
A. It is absolutely relevant. I mean ... yes.
MR STRAW: Thank you.
THE CORONER: You can ask -- one is very unusual.
MR STRAW: There is just one more then.
Is that toxicological evidence, Professor Ferner and so on, part of what you were referring to earlier when you said that the other circumstances, as they are, are relevant to your conclusion of unascertained?
A. Yes, I think we have to look at the whole picture. It

is not just the pathological findings at autopsy.
MR STRAW: Thank you.
MR WASTELL: Sir, I hope that assists.
THE CORONER: Yes, thank you.
MR WASTELL: It may be once Dr Ratcliffe is released a break for the transcribers would be in order.
THE CORONER: Thank you very much indeed.
Yes, we will do that.
(11.50 am)
(A short adjournment)
(12.13 pm)
MS HILL: Sir, might I just come back to you on the issue of the Loxton letter and the address, do you remember you asked me some questions about that. I have just made some checks since you raised it.
A. Thank you so much.
MS HILL: For your reference, sir, the document that I took the witness to yesterday.
THE CORONER: D48, was it?
MS HILL: D48, there are different versions of it but the document that is most helpful to look at I think is in volume 5, page 121.
If it helps you, that suggests that when Mrs Perepilichnaya had contact with the police on 30 May 2011, she was by then at The Coach House.

I then took the witness as well to a group of Western Union payments --
THE CORONER: Yes.
MS HILL: -- on volume 5, page 250.
They showed payments on these dates in 2011 from The Coach House address, 20 May, 23 May and 8 June.
All of which of course are after the March address, but perhaps most significantly I have identified within the insurance bundle a document at volume 7, page 354/73.
THE CORONER: Yes.
MS HILL: Which is a callout for Thamesdoc, which is an emergency medical service, on 30 April 2011, and that -- sorry, I am talking about The Coach House, I am meaning to say at this point the Virginia Water address, I'm sorry, I got them the wrong way round.
30 April 2011 is at the Virginia Water address, so all of those dates that I have just given you, 30 May for the contact with the police, May and June Western Union payments, and then that emergency call out on 30 April are all at the Virginia Water address.
So when Dr Loxton has The Coach House in March, there is quite a bit of documentary evidence to suggest that that is anomalous.
THE CORONER: Yes.

MS HILL: Just for completeness, if I may, at the other side of the move, if you like, the document that I took you to that suggests that they were in The Coach House by 26 June is also D48 that, is 5/121. There is also then on 5/250 Western Union payments from 15 September 2011 at The Coach House.
Just finally on this, I don't want to labour the point but I have gone through the insurance bundle and I am happy to save some time in case someone else wants to do it. On these dates there are letters sent to Mr Perepilichnyy at The Coach House address in 2012, earlier than 25 July which is the date that Inspector Pollard thought he had moved, all right?
These dates are: 8 May at The Coach House, 7/95; 22 June 2012 at The Coach House, 7/182; 20 July 2012 at The Coach House, 7/174.
When at volume 1, page 190 Officer Pollard posited the suggestion that they had moved on 25 July 2012, there is not only Mrs Perepilichnaya's evidence that he is wrong about that, and that is perhaps a renewal agreement, but there is quite a lot of documentary evidence that they had moved to The Coach House long before then.
Indeed those documents that I have taken you to I think support the proposition that they moved fairly

proximate to the contact Mrs Perepilichnaya had with the police.
THE CORONER: Thank you very much.
MR BEGGS: My Lord, might I just raise in case you thought my silence connoted lack of cooperation, that naturally we have asked our client to see if she can delve her memory and documents to help you on this point.
MS HILL: In fact, sir, my junior reminds me that the page numbers have changed in volume 7 because I have my sticker on it. The 30 April 2011 entry where the Thamesdoc callout is a callout to the Virginia Water address is page 78 of volume 7, it is not 354.
THE CORONER: Say that again, page 78?
MS HILL: 78, I have given you the original numbering but to be fair, without addressing you on the facts you can see that document, that is not only correspondence but that is a callout to the address of an emergency doctor, it seems that Mr Perepilichnyy had had a gastric problem that had lead to an emergency callout, so the doctor goes to that house so Dr Loxton's letter seems to be anomalous.
THE CORONER: Thank you very much.
MR SKELTON: Sir, going back to the tenancies, there is clearly a period of time when Mr Perepilichnyy and his family do move, it may be worth us trying to find out if

we can get copies of the tenancy agreements for these properties, insofar as they are still available.
THE CORONER: It is on my list of things we might look at, yes.
MR SKELTON: Sir, may I call Professor Sheppard.
MS HILL: Sir, just before the witness is sworn I am sure at some point today we are going to have some submissions on the Buzzfeed issue, I anticipate it is going to be raised at some point.
MR SKELTON: Yes.
MS HILL: Good, thank you.

PROFESSOR MARY SHEPPARD (sworn)

Questions from MR SKELTON

MR SKELTON: Professor Sheppard, would you state your full name to the court, please.
A. Pardon?
Q. Would you state your full name to the court, please.
A. Yes, my name is Mary Noelle Sheppard.
Q. Your present occupation?
A. I am a cardiac pathologist working at St George's Hospital Medical School in Tooting, London.
Q. How long have you held that position?
A. I have held that position for four years as Professor, but I have been a cardiac pathologist for 25 years. Specialising in heart disease.

Q. It is right to say you are not dual qualified as a cardiologist?
A. No, I am not qualified as a cardiologist.
Q. Insofar as I am starting to trespass during my questions onto cardiological territories, for example signs and symptoms of heart disease or failure, I should direct my questions elsewhere, to Dr Wilmshurst?
A. Yes.
Q. Thank you.

You produced an initial letter or report in the form of a letter in response to Dr Ratcliffe. You will find that in the bundle I hope you have in front of you at tab 53?
A. 53.
Q. 53, it should be in file 2?
A. Yes, here we are, yes.
Q. You can probably get rid of the other file because I doubt I will refer you to it.
A. Right, you can take that away, thank you.
Q. Page 413.
A. Number? Sorry? Sorry it is number which?
Q. Tab 53, page 413?
A. 91 is what I have. I am not on 53.
Q. There is some pagination at the top right-hand corner, is that what you are looking at, Professor, in bundle 2?

A. It is file 1, is it?
Q. File 2.
A. No, file 2 is not here.

Yes, that is it, thank you very much.
Q. There we go.
A. 53, yes?
Q. Tab 53, page 413, according to the pagination at the top right-hand side.
A. That's correct, yes.
Q. This is your letter in response to Dr Ratcliffe and he was requesting a detailed pathological examination of the heart?
A. That's correct.
Q. Primarily on the basis that he thought he found an anomalous vascular structure?
A. Yes.
Q. Could you clarify how you go about examining the heart?
A. When I look at the heart, I normally will look at the exterior, will weigh the heart to make sure it is a normal weight -- this is very important, if the weight is particularly heavier than normal, so weigh the heart, remove blood clots if there is blood within it, examine carefully the coronary arteries on the surface of the heart, because as we are well aware people dying suddenly of any cause, the most common is coronary

artery disease.

You have to look carefully at the coronary arteries to eliminate it as a possibility and then you look at the rest of the heart, the muscle and the valves and the whole structure of the heart itself to make sure it is normal or abnormal.

Q. And are you dissecting into the heart to look at the ventricles and the atria are you?

A. Yes, correct.

Q. Likewise the valves?

A. Yes.

Q. And the arteries on the outside of the heart?

A. Yes.

Q. You are looking for any form of abnormality, but presumably there is some variability in the vascular structure sometimes?

A. Yes, there is variability in the location, obviously of the openings, when you look into the vessel coming out of the heart, the main artery that supplies the blood, that is where the two coronary arteries are and you have got to look carefully into that, as already described by Dr Ratcliffe, the sinuses these are the right and the left.

But, as I published, there can be variation in the location. What you have got to look for carefully is:

is the opening patent? That it is normal, and that the vessel is not going an abnormal course once it leaves the aorta or the vessel, that it is not an abnormal course. Whereas in this situation, there was no abnormal course and there was nothing in the heart that gave damage to the heart that could be due to an abnormal coronary artery.

Q. Was he in a minority of patients that may have that form of vascular structure?

A. Correct, correct. In that location.

Q. Presumably you have seen that structure before on many occasions?

A. I have and it is a normal -- it is a variation on normal, like height, where the location of the ostium, we call it, but the opening where the blood goes into the artery itself.

Q. Thank you.

A. In 1990s we published on that showing it could be quite close to the what they call the commiseur(?), where one sinus meets another. As long as it is not obstructed and not an abnormal course, this is a normal variation.

Q. In this case you found in abnormalities on macroscopic examination?

A. Correct.

Q. Just to be clear, does macroscopic examination include

any form of examination that does not involve a microscope?

A. Yes.

Q. Thank you.

Then, in terms of microscopic examination, do you take slivers or little samples by --

A. Small tiny samples we will take. Once we look, as I say, I examine all the chambers, his heart weight was normal at 393 grams, that is very important. I look at the vessels, I look at the right side of the heart, the left side, I make measurements, and these measurements, over many years I find to be normal. All his measurements were absolutely normal for the muscle, the valves of the heart and the structures of the heart.

Q. That includes, for clarification, the mitral valve -- have I pronounced it correctly, the mitral valve?

A. The mitral valve, absolutely, I looked at the mitral valve circumference, which was 80 mms, and this is normal for the average adult person.

Q. Would you expect to spot a prolapsed valve on post mortem?

A. The annulus may be dilated, it would be increased above 100 mm, is what I accept, but also the valve itself looks abnormal in prolapses. What prolapse is, is where the valve leaks, not normally when we close the

chambers, the heart is a one-way chamber, it has to be to allow blood to -- if you get up a back up behind, it will not pump efficiently. Those valves work like traffic lights letting in and out. If they don't work properly, they are incompetent, they prolapse. That means they leak, they pull apart, they stretch and they become thickened.

I did not see any evidence of thickening or pulling apart of the leaflets, there are two leaflets, there are two leaflets of the mitral valve.

Q. Had he suffered an abnormality of the mitral valve in some form or other, would you have expected to have found it post mortem?

A. I would have expected to see it. You cannot have an invisible prolapse, you always get an effect on the valve of prolapse, it will thicken, it will balloon, it will stretch apart. It will not be normal.

Q. Are you certain that has been excluded as --

A. I am certain that has been excluded.

Q. Going back to the details of the microscopic examination, you are looking at the muscle itself in the heart?

A. Yes.

Q. You are also looking at the vessels within the muscle, the intramural vessels?

A. That's correct.
Q. Are there any other tissues beyond that that you are checking, do you take samples of the valves for example?
A. Yes, I take samples of the valve and I take samples of the muscle and of all the layers from the outer to the inner layers of the heart, including the coronary arteries as well. I look at the arteries, just to make sure they are normal, it is simply confirming if I find the heart to be normal, it is confirming it because you can have a normal looking heart, naked eye, macroscopically, but microscopically you may find abnormalities that you don't see by naked eye. That is why every cardiac examination should include microscopic examination.
Q. Do you have a standard way that you go through this process, that you invariably follow?
A. Yes, an established standard way I examine.
I look at all the chambers, take samples from each of the chambers, the upper chambers, the lower chambers and the valves and the arteries.
Q. It is a comprehensive assessment, macroscopically and microscopically?
A. Very extensive.
Q. Roughly how many samples are you taking in all this?
A. I am taking 10 to 12 blocks of tissue from various

areas.
Q. Thank you.
Did you find anything in the macroscopic or microscopic examination that was cause for concern?
A. No, nothing. It was within what I consider normal findings that I look to see microscopically in the heart.
Q. If one assumes it is still a cardiac cause of death, what are you then considering as possibilities of?
A. When the heart is normal, and when no other cause is found at autopsy you consider sudden adult or arrhythmic death, but you also obviously have to include toxicology and all your other inquiries before you come to this diagnosis.
Q. Yes. Toxicology is obviously outside your expertise?
A. Absolutely.
Q. Can you eliminate channelopathies by the kind of examinations you conducted?
A. No, what my examination tells the cardiologist, because many of these conditions are genetic, it tells the cardiologist, "Well, when I examine the family I will look for channelopathies", because that is the most common cause of sudden arrhythmic death when you have a normal heart, it's an electrical abnormality, so it is invisible to my eye.

Q. Are there any structural problems that you would need to conduct more histopathological testing for?
A. No, the conduction tissue is normal. The electrical pathways look normal to the naked eye, and microscopically they are normal. It is an abnormality in the channels conducting electricity between the cells. It is at a cellular level and I cannot look at the individual cells and I cannot see what they call the channel abnormalities, because they are genetic mutations and it is only by doing electrical activity and analysing living patients that you will find, detect the abnormality in a living relative of the person who died or in an ECG they had before they died.
It has to have electrical activity so with that, all electrical activity ceases so the pathologist sees nothing.
Q. In answer to some questions that arose after your report, in the context of this Inquest, you were asked about Wolff-Parkinson-White syndrome and the possibility of histopathological testing for that. I think you gave the answer, for reference it is on page 416 in the bundle in front of you, at paragraph 2.
A. 416, yes.
Q. 416, you can see paragraph 2, you accept it can cause sudden death and although I am skipping ahead I will

come on to some definitions later, but just on the examination point:
"While I looked at conduction tissue, I was not able to sample all of the atrioventricular junction to look for an accessory pathway as this would have taken thousands of histological sections and is totally impractical."
Are you effectively saying you can do it but it is very, very difficult or you simply never do this?
A. Pathologists never do this. We can't serially sample what is -- between the upper chambers and the lower chambers, they are totally isolated from each other apart from the conduction tissue, to allow one impulse to get through from the upper chambers to the lower chamber. The rest of the heart is isolated by a band of what we call collagen or fibrous tissue. We serially section that, but in the normal heart you do get bundles of muscle coming down from the upper to the lower chamber. In the normal heart.
Indicating that this is a Wolff-Parkinson-White on a little bundle of muscle is impossible for me as a pathologist, it is a normal variation again.
Number 2, Wolff-Parkinson is generally a clinical diagnosis based upon electrical activity and ECG findings, it is not based on pathology.

Q. Thank you.
A. It never is or rarely is and I wouldn't take a small single muscle bundle and say that person had Wolff-Parkinson-White based on one tiny little strand of muscle. I simply wouldn't be able to, and neither would any other pathologist in the world.
Q. In order to diagnosis it effectively you need diagnosis in life?
A. Absolutely, in my experience.
Q. Sudden arrhythmic death syndrome has it now coalesced to the point where it is accepted as a definition, because there had previously been sudden adult death syndrome, although that was exclusive to adults?
A. Correct, it is in children as well. That term is not liked by a lot of people because children suffer this as well, outside of the sudden infant death, which is under the age of a year.
We prefer to use sudden arrhythmic, although there is no definitive evidence you died of a cardiac arrhythmia, particularly in the circumstances where you are found dead, but it is about the best -- sudden unexpected death is used, SUDS, SADS, they are interchangeable.
Q. I was going to ask you that, because arrhythmic death implies cardiac, that is the rhythm that has gone wrong

or stopped.
A. Yes, it does.
Q. Is there a danger that the use of that term in fact could be a misnomer, in circumstances where for example it is an epileptic death?
A. No, it is a spectrum. Sudden death in epilepsy is a spectrum of SADS, we call it SUDEP, sudden unexpected death in epilepsy. Where epilepsy has a higher than average incidence of sudden unexpected, or we say sudden death.
Whether the epilepsy causes the cardiac arrhythmia or whether the cardiac arrhythmia is involved and there is a connection between heart and brain, it is well known and we are only now doing research into it where people are found dead in bed, but they are obviously you know poorly controlled epileptics, some polypharmacy as well, so drugs come into it. So it is a multifactorial entity sudden death in epilepsy.
It is a spectrum of sudden expected death, 10 per cent of my cases in my database are epileptics, or have a history of epilepsy.
Q. The role of the heart in death, a stopped heart equates with death, unless I suppose you are undergoing surgery, in which case you may have some situations where the heart is controlled with circulation provided by

mechanical means for example.
Are there any ways of effectively dying or being declared dead without your heart having stopped?
A. My goodness -- yes, I presume when you have been resuscitated and your circulation is restored, but you have got irreversible hypoxic brain damage. That is quite common and the ventilator is turned off and the heart and circulation ceases, but that is after a cardiac arrest, generally.
Q. When you talk about epilepsy, or any other sort of brain problem, a problem with the brain or the spinal cord, is the end result cardiac arrest, arrhythmia leading to arrest that causes death?
A. We all die of cardiac arrest eventually, it is the terminal event, that your circulation ceases.
Q. That is where I was getting to, I think. Are there any other forms of non-cardiac event in the brain for example, that lead to arrhythmia and death?
A. Oh yes, sub-arachnoid haemorrhage, if you have a haemorrhage in the brain, particularly from what we call the sub-arachnoid layer of the brain, haemorrhage from an aneurysm, that can lead to cardiac arrhythmias.
Q. I should have clarified, any occult events that you don't find pathologically?
A. Obviously in epilepsy they are claiming there may be

abnormalities, but it is only on a research basis, there is no localised -- yes, there can be brain tumours or brain lesions but generally in epilepsy there may be neurological subtle abnormalities which a neuropathologist can comment more than I can, but there is no specific abnormality in the brain in sudden death in epilepsy that would say it affected the heart.
Q. Are there any other conditions, either in the brain or other major organs of life, which will not be found pathologically but which will lead to arrhythmia and death?
A. No, nothing in the rest of the body that can lead to a suggest arrhythmic death, maybe sometimes rare tumours which raise the blood, noradrenaline, what we call paragangliomas, but they are found if you know what I mean, at autopsy they would be found, they could cause arrhythmias in the heart but then it wouldn't be a sudden arrhythmic death, because you have found the cause.
Q. That leads me on to my next question, which is that it is a diagnosis of exclusion?
A. Absolutely.
Q. To clarify, that means no structural abnormality or disease of the heart?
A. Correct.

Q. No other disease process or poisoning process or other manifest cause of death on pathological examination?
A. Yes.
Q. When the SADS is clearly a cardiac cause, or likely to be a cardiac cause because you have ruled out epilepsy for example, are the most likely causes ion channelopathies?
A. Yes, we would follow up on the families, we would clinically follow up on the families.
Q. You would normally check with the family after the person has been diagnosed or died?
A. The person has died so you cannot find electrical activity in the dead, unless they have previous ECGs which you can review. In addition, you can take genetic material at what we call now the molecular autopsy, we take genetic material in any sudden death in order that genetic testing can be carried out for the possibility of these channelopathies, but that has only come in the last three years in practice among pathologist.
Q. Could you just explain for the layman, if that is possible, what an ion channelopathy is?
A. An ion channelopathy is where ions like calcium, potassium, sodium, these are ions, they are chemicals that go through from one cell to another, through channels, that is why they are channelopathies, that

communicate rapidly, that allow electrical activity to go on. Our hearts beat 70 times a minute for an average 70 years of our life. That electrical activity has to be very coordinate and very fast, so these are super channels and each cell aligned them to communicate with each other and beat together in harmony. When there is an abnormality in these channels they will not beat in harmony and it leads to cardiac arrest with ventricular fibrillation.
Q. Thank you.
You are not an epidemiologist --
A. No.
Q. -- but you do publish on the issue of SADS --
A. Yes.
Q. -- which involves to some extent some epidemiological data?
A. Yes, and I work very closely with my cardiology colleagues, because follow up on the families is so important for me to know what they are finding, so I am informed.
Q. Is it possible to say how many people die from SADS in the UK at present?
A. Tragically we still haven't got the exact statistics, because some cases are still reported as unascertained, which I find a very vague unfortunately term, so they

are not reported as SADS, just they are reported as cardiac arrest and what caused the cardiac arrest? So the Office of National Statistics are not quite precise enough to give us, but we think there are 800 per year, at least 800 per year, according to my database, that is how many I get.
Q. 800 from a cohort of?
A. From the population of England. At least, and I do not get every case. But that is 12 per week.
Q. Sorry, out of those 800, are you saying those are your 800?
A. They are also from other studies, from the Office of National Statistics' studies that have been done by cardiologists, I would see about 500 of those 800.
Q. Is that comparable to data in other jurisdictions that you are aware of?
A. Yes, it is. There is very few statistics from any other country in Europe, or the USA, very little specific statistics. We are very lucky in this country we carry out autopsies on all sudden deaths, in other countries they don't. When they deem it natural they do not have an autopsy, so we are very lucky that we have a good system here.
Q. Do you happen to know, I do appreciate I am pushing you perhaps into an area you may not be familiar, but out of

the 800 in how many cases do you get to the point where you do find the combination of molecular pathology, clinical history, having been looked through and genetic testing of the families lead you to a diagnosis of a channelopathy for example?
A. 40 per cent, we are now finding. In our latest study 40 per cent.
It is increasing, it is getting better because of clinical experience with the conditions, they have new tests, combined with the genetics. It has to be very much interpreted very carefully by both the geneticists and the cardiologists. It is a multidisciplinary team that comes to the diagnosis.
Q. Thank you.
Last month I think you were a co-author on a paper called "Utility of Post Mortem Genetic Testing in Cases of Sudden Arrhythmic Death Syndrome" published by the Journal of the American College of Cardiology and the principal author is Nadim Lahrouchi MD.
A. Yes.
Q. I wonder if you could be passed a copy of that if that is possible, please. (Handed)
A. Yes, I can refer to it.
Q. Can I just clarify, first of all this draws upon data from various places, including New Zealand, Denmark, the

UK and Holland and the data from the UK includes your own hospital --

A. Yes.

Q. -- st George's, as well as other hospitals around the country.

If we continue through the paper, we have a section called "Results" on page 2137. In fact a very handy graphic with pie charts, et cetera, overleaf.

A. Yes.

Q. Could you just talk me through the principal results and --

A. Without going into detail is that there was a molecular diagnosis, that means on genetics, but they actually used a very high level of genetic testing because we all have mutations in our system and it is actually being specific getting an expert on genetics to say, "This is responsible", because some mutations are not responsible so it is a whole area of expertise which has developed over the last 20 years.

So molecular in 11 cases, just 13 per cent and there was a higher yield --

Q. Can I just pause there. The 13 per cent?

A. Of the 300, of the cases examined, of the families, as you say validated SADS with suitable DNA.

Q. Is that 13 per cent, the 13 per cent of the molecular

pathology that was conducted on the deceased?

A. Correct, where they had clinical follow up on the family and they had material from testing in the proband in the person who died.

Q. Assuming that that is a statistically valid percentage, does that follow that you will expect to find a positive molecular result or some form of genetic problem in 13 per cent of cases?

A. Yes, you will. It varies in different studies but it is generally between 13 and 30 per cent.

Q. I think you said that the science has evolved over the last three years even, 20 years but over the last three years you have been able to conduct a certain type of testing, is it still in --

A. It is still developing, an interpretation, because everybody thinks one test is all you need to make a diagnosis when we now know it takes testing and interpretation of what is found by the -- because they can do whole gene testing and we all have mutations, we all have abnormal genes, and it is interpreting the abnormal genes as to whether they are not causing the disease.

This is the skill required. We have actual clinical geneticists who are experts in cardiac disease only.

Q. Are there some recognised forms of channelopathy that

one cannot yet test for?

A. Oh yes, in Brugada syndrome, if it is clinically diagnosed in the family or in the person, only 30 per cent get an answer that has a mutation, the other 70 per cent of Brugada is negative. It is well known, it is a lower yield than the long QT syndrome or CVPT.

Q. Do you think it is possible that further channelopathies will become apparent?

A. I think so, with better testing, better interpretation, more genetic testing, a larger panel that we will be finding new mutations, but it is going to take considerable expertise and time. Although now it is going ahead so rapidly it may not be a major issue, but it still takes expertise and interpretation.

Q. Just taking it in stages, in terms of male/female breakdown, do you see any difference between the two?

A. Huge. Male 3 to 1, compared to female, for sudden cardiac death, not cardiac death -- yes, even sudden cardiac death, males die much more than females with all cardiac disease.

Q. Why is that?

A. We don't know. Women are protected, for cardiac coronary artery disease we are protected by our hormones, during pre-menopause, after the menopause unfortunately we females catch up with males. For

sudden cardiac death for instance, this sudden adult death or arrhythmia death, it is well known that males die way more than females and we simply can't explain it.

Males have more muscle in their heart, but they don't have more channels.

Q. They have bigger hearts?

A. They have a bigger heart, so are they more liable to sudden death because they have a bigger heart? We simply don't know.

For instance, sudden death at adolescence is common in males when the growth spurt with the heart, in cardiomyopathies is very, very common in young males, but not in young females we think it is the growth and the muscle bulk, but we really cannot be definitive about that.

Q. That again raises another point about the age, taking males, for example.

A. Yes.

Q. It appears from the data that has been gathered that most of these deaths occur under 30?

A. That is not correct. We find when I examine all cases, most are under 35 but that is because that is where the majority are, but half of my database is in people over 35, so it is as common in over 35 as under 35. But

I think it is misdiagnosed in older patients because they attribute the death to coronary artery disease, they will see a coronary artery with a bit of atheroma and that is attributed to the death, whereas I have a very, very precise way of judging a coronary artery and I am quite conservative, but I find half my cases are over 35. In this study, again, median age was 24 but there was quite a few people over the page of 35 in this.

Q. Yes, just looking at "Most were of European descent", and "235 died before or at the page of 35 years", which is 78 per cent?

A. Yes, so the other 32 per cent were over 35.

Q. Your data you are saying is --

A. My database of my own cases that I get from all over the country, half of my referred sudden adults arrhythmia deaths are over 35.

Q. Thank you.

Just going back to the issue of the misdiagnosis of coronary artery disease, will you usually expect to see an infarcted --

A. No, you may not see an infarct, you may not see damage. Pathologists and corners also accept the fact you have just coronary artery disease which is just blocking the artery.

Q. You need a block though as opposed to --

A. I am sure the coroner will agree with me that it depends on what the pathologists describe as a blocked coronary artery, it is done by naked eye and you judge as "blocked" or "pinpoint" or "significantly narrowed" are the terminology used by different pathologists. In this case, there was no question of coronary artery disease blocking or being pinpoint or causing any obstruction. This man's coronary artery was non-significant, all three pathologists agree on that.

Q. Thank you.

In the results section of the paragraph I am still looking at on page 2137, bottom left, there is reference to exercise. It says:

"The most prevalent circumstances of death were during sleep, 43 per cent, or rest, 29 per cent. With death occurring during exercise or extreme emotion in 10 per cent or 1.5 per cent respectively."

So 10 per cent of deaths on exercise?

A. Yes, and that reflects in our studies my database 10 to 15 per cent of my cases of sudden death with exercise.

Q. Does that have an age-related element to it as well?

A. Generally, from the point -- well, not -- well it depends on what you are dealing. Age related when it is coronary artery disease, coronary artery disease cause

of sudden death is very common with exercise, let's say marathon runners, middle aged men taking up marathon running, they are the most likely people to die during a marathon.

Age related cause would be older, but in the younger it is channelopathies under 35, or cardiomyopathies.

Q. Overleaf there is a series of graphics?

A. Yes.

Q. You can see at piechart B, exercise is 10 per cent there and it shows the relative comparable activities or non-activities?

A. Yes.

Q. C has a large three-quarters of the graphic is taken up by "no prior symptoms"?

A. Yes that, is the tragedy of this entry, is that the majority of patients are utterly asymptomatic until they die suddenly. Which is very tragic for the families, it is a dreadful shock to everybody.

Q. By asymptomatic, do you mean a period of time beyond the --

A. Or beyond prior to death, that they are, they have been well, but then they get symptoms an hour prior to death.

Q. That is the suddenness, is one hour?

A. That what the definition of it is.

Q. Thank you. I think, if there is no one observing

them --

A. Or if it is unwitnessed death, it is last seen alive 12 hours before, but that can vary sometimes, a body may not be discovered for two or three days, so it is a bit variable as to the definition.

Q. Looking at the same chart, chart C, seizure 13 per cent, so that is fit of some kind?

A. Yes.

Q. Implying a brain malfunction of some sort?

A. Yes, but quite a percentage of people with a cardiac arrest have a seizure, deprivation of the blood supply to the brain. Labelling it as epilepsy is incorrect, because that may be the very first and last seizure that person has because of the cardiac arrest.

Q. It is carefully worded "seizure" rather than "epilepsy"?

A. Yes, it is seizure it is not epilepsy. That is a clinical diagnosis prior to death.

Q. What is syncope?

A. Syncope means a collapse, a blackout, where if I am talking to you I suddenly collapse but I recover, syncope. It is a very dangerous what we call symptom, it is a red flag symptom.

Q. A brief period of unconsciousness?

A. Loss of consciousness, correct.

Q. Palpitations?

A. That means irregular heartbeat, where you are aware yourself of your heartbeat going rapidly or slowly and you are aware of it in your chest.
Q. Chest pain, straightforward?
A. Pretty straightforward.
Q. Although can be non-specific in heart cases?
A. They can be due to any of many multiple cause.
Q. Lastly shortness of breath?
A. Yes.
Q. In 1.5 per cent of cases?
A. Yes.
Q. Some of these could be analogous to the type of experiences one gets when you are about to have a normal heart attack?
A. Yes, all these can occur with a heart attack, yes.
Q. Thank you.
Overleaf if you would, please, another set of diagrams, just to understand, I don't need to take you through them in detail because much of the medicine is too complicated for these purposes. You can see on the chart A, "Yield of genetic testing", your pathogenic and likely pathogenic, is that what makes up the 13 per cent, so 6.5 plus 6.5 and "likely pathogenic"?
A. Yes, this reflects the grey zones that pathogenic has been shown that in the past that mutation is definitely

causing the disease.
"Likely" means they are almost 99 per cent certain it is but there is a 1 per cent possibility it is not.
Then variance, "VUS" is variance of unknown significance, means there is a variation but we don't know what it means. Like we all have variations in our genes.
Then no rare variant, 44 per cent which is nothing found in 44 per cent.
Q. Presumably the variance is going to be the focus of considerable thinking and research for years to come?
A. Absolutely.
Q. Below there is a Venn diagram where one can see the 13 per cent on the left-hand side in blue. Then on the far right-hand side you have 14 per cent, is that the follow up with the family on --
A. Yes, that is clinical, when they follow up immediate blood relatives, you screen the siblings and parents of the deceased to look for the electrical abnormalities that will diagnose the channelopathy.
Q. For example the 12 --
A. Correct.
Q. In between, the bit that is the coincidence part, 8.5 per cent?
A. That is the combined, where you do the genetics with the

clinical and bring them together.
Q. That is just shy of 40 per cent or is that --
A. Yes, 39 per cent.
Q. Yes. Is that when you refer to the paper that is going to be published soon, is that the kind of figure that you are looking at?
A. Absolutely. Our latest papers confirming this, that we will find a diagnosis in 40 per cent of families.
Q. Is that drawing upon new data?
A. Yes, new data. More, bigger, larger numbers again.
Q. Is there anything else arising from either this paper or the pertinent papers on SADS which is relevant in the context of this Inquest?
A. Not that I am aware of.
Q. Thank you.
As far as the types of channelopathy are concerned, it may be that given the preponderance of diagnostic investigations are in life or clinical signs or clinical investigations by ECG, am I better directing my questions to Dr Wilmshurst on that?
A. Absolutely, because to emphasise the pathological examination will not be able to tell what type of channelopathy it is, it just says the heart is normal and then it is up to the cardiologist to look for the different types. We cannot tell.

Q. Just to establish your conclusions, you have been clear in your evidence that you have excluded any structural problem or disease in Mr Perepilichnyy's heart?
A. That's correct.
Q. I think you will be aware that Dr Homfray commissioned genetic research which ruled out certain conditions but I think what you are based on the percentage expectation that we have looked at, there is a huge number of conditions that cannot be ruled out genetically at this stage?
A. That's correct.
Q. A large majority of conditions, in fact?
A. 60 per cent.
Q. Can I just ask you to look at your joint statement, please, which you may have loose leaf as well. It is in the bundle, it is tab 96, right towards the back, page 846.
A. 96 is it? That will be in folder 3, I think.
Q. Yes.
A. So 93?
Q. Yes. Tab 93, I am going to use the internal pagination because I do not have one which has the other form, so page 8, please, and it is paragraph L.
A. Page 8?
Q. 96 for those that need the other pagination.

A. I am here on 94, is it 96 you are looking at.
Q. So I am told.
A. 96, yes, here it is, yes, joint statement.
Page number?
Q. Internal 8 is probably the easiest.
A. 8, yes.
Q. That is probably the easiest reference, I think.
You say there:
"In families where someone has died of SADS, about 4 in 10 families show no sign of inherited heart disease."
A. I think it is more, 4 in 10. I can say from our studies, this is from SADS, this is about to be published so we now have upped it to that, 40 per cent, in our latest study which will be published. You can say 60 per cent do not but 4 in 10 will have, where is it again?
Q. I was just clarifying, is it the wrong way round?
A. Yes, but I mean 4 out of 10, this is from a study that was previously done by Elijah Behr on an earlier study but small numbers. Now that we have larger numbers.
Q. Yes, just to clarify is this statement correct as a representation of what Dr Behr has found?
A. Has published on, in about 2006 though, it is nearly 10 years --
Q. I thought you were saying earlier that the basic

proposition is that 4 in 10 do show signs of a problem?
A. Yes. Four in 10 do, but 6 don't.
Q. Here it says no sign of inherited heart disease?
A. Yes, 4 in 10 show no sign -- no, not of inherited heart disease, we find in other words they look and they see and they do an examination and the family have nothing wrong with them. If genetic testing is done it is negative, but you cannot rule out the possibility that they have something, an underlying unknown channelopathy, or inherited channelopathy. It is a bit of a challenging area.
Q. It is but just for clarification, based on your latest research, if one is to test a family for genetic channelopathy of some kind, what is the percentage expectation that you will find an abnormality?
A. Electrical -- it is 40 per cent.
Q. It is now 40 per cent?
A. It is now 40 per cent, based upon our latest numbers. It is evolving as we are saying and getting better with time. In another year it may be better again, or two years.
Q. Thank you.
You had a discussion with Dr Wilmshurst and during that discussion you came to a conclusion about the cause of death. Can I just show you the joint statement

again, which I may have handed down.
A. Yes.
Q. Thank you.
On internal page 10, please, paragraphs 27 and 28.
A. Sorry, I think -- this is the second one. This is in 10?
Q. The joint statement.
A. Yes.
Q. Yes.
A. Paragraph 28, yes.
Q. 27 and 28.
A. Yes.
Q. The first sentence you say is:
"The findings on genetic testing have no significance, even though no gene abnormality linked to channelopathy was detected."
That is because the expectation is so low that you are going to find something, 13 per cent?
A. Correct, yes.
Q. "Therefore in our opinion if the court decides that Mr Perepilichnyy did not die from trauma, catastrophic acute medical illness, overt heart disease or poisoning, the exclusion criteria will have been satisfied in order to make a diagnosis of death from SADS."
A. That is correct.

Q. You in fact can rule out overt heart disease, you have done?
A. Yes.
Q. That includes a structural abnormality and disease process.
The rest is for others, including the coroner?
A. Absolutely. Yes.
Q. From your perspective, if those things are ruled out to the satisfaction of the court, then this is a sudden adult death?
A. Yes, if the others are ruled out.
Q. Without going into the detail, which I think we will have to go through with Dr Wilmshurst, you go on in paragraph 28 to say:
"Negative findings at post mortem examination and on toxicology are consistent with both SADS and channelopathy."
A. Yes.
Q. And failure of the molecular autopsy to demonstrate a pathogenic gene mutation does not alter that?
A. Correct.
Q. Is there anything you would like to add today to that conclusion?
A. No.
Q. I didn't ask you at the start, but do the conclusions

that you reach both in your answer to Dr Ratcliffe, in your short written answers I took you to and in the joint written statement stand as the evidence that you want to give to this court?
A. Yes.
MR SKELTON: Thank you.
THE CORONER: How long are be going to be, if it is possible to carry on so that we can finish and the Professor can go --
A. I am quite happy.
THE CORONER: -- I would like to do that, shall we.
MR MOXON BROWNE: I will do my best to cooperate --
THE CORONER: I am sure you will.
MR MOXON BROWNE: -- and to enable Professor Sheppard to get away.
Questions from MR MOXON BROWNE
MR MOXON BROWNE: Professor Sheppard, at the beginning of your evidence you were stressing that the heart that was sent to you showed no abnormality that had been with Mr Perepilichnyy all his life, no anomalous arteries or anything of that sort. What I think you didn't perhaps mention or emphasise particularly was that as well as that finding, you also found that there was no damage to the heart that might be associated with a fatality?
A. No, there was no damage to the heart.

Q. Yes. If somebody has a heart attack for example because they go marathon running, there is massive muscular damage --
A. No, because people can die rapidly without damage evolving, when they get a clot, for instance you get a clot in the coronary artery, you don't need to have muscle damage in the heart, you can instantaneously die of an arrhythmia.
Q. But you would find --
A. I would hope to find a clot, sometimes you don't -- well generally you do find a clot or a significant blocking of the coronary artery.
Q. The coroner has heard evidence in this case, that he will make up his mind about, of a middle aged man running up a hill?
A. Yes.
Q. Mr Perepilichnyy. Whether he was just a middle aged man puffing up the hill or whether he was in some distress will be a matter for the coroner, but I think I am right in saying that a death associated with that sort of activity tends to result in what we call a heart attack?
A. If it is due to -- we call that due to coronary artery disease, is what I label as a heart attack because it is given out for -- even the sudden death of that recent footballer was due to cardiac failure it was claimed,

but the autopsy doesn't label it as "heart attack", it can be due to any cause.
Q. Yes, but that is the type of death associated --
A. The most common, the most common. Linked to coronary artery disease.
Q. Perhaps I could finish my question, is something that you would detect at autopsy either because --
A. We would detect, the pathologists, the coronary artery disease, or the damage in the heart if there was damage there.
Q. I hope it is not misleading or an oversimplification to say that the number of SADS deaths associated with exercise is quite small, 13 --
A. Compared with other causes, yes, it is small.
Q. Although the fact that Mr Perepilichnyy was exercising when he met his death may be relevant for other reasons, it doesn't for you have a particular resonance for a SADS death?
A. No.
Q. I think that you have had a lifelong interest in SADS and in particular the proper recognition of SADS and its statistical prevalence. You have been, if I may say so, a bit of an evangelist, but it has been under-reported and you are trying to improve that?
A. Yes.

Q. I think you have certainly, with colleagues, a national reputation as a leader in that field. Indeed I have read papers from America and Japan, all of which seem to come back to your research. You are an international expert in this field?
A. Yes.
Q. A lot of those papers go back to work you did in the early 2000s, culminating in a paper in which you identified, or did your best to identify, from existing data, the prevalence of SADS. The process, which was to some extent an epidemiological approach, was to look at and define sudden cardiac death, which is someone who dies unexpectedly within 12 hours of anything happening, and then to take out of that all those patients who at autopsy or when their history was investigated, you could put another cause to it.
You went down, down, down, down, down, and you came up with 4 per cent out of those sudden cardiac deaths which you could say, "Well, we cannot find anything that might explain this, so we will call that a sudden cardiac death"?
A. Yes.
Q. That is --
A. That was the initial project, yes.
Q. What you were looking for and what your list in that

paper shows is that principally you were looking for what I would call co-morbidity, that is to say something wrong with the person. That is right, isn't it?

A. Yes.

Q. For example if they had a history of epilepsy then they would be out automatically?

A. Correct. In that study, we now include epilepsy in our studies.

Q. Because that is a special thing, I understand.

There were I think at least a couple of conditions that would cause you to exclude a SADS death which I don't think could properly be described as co-morbidity. One of them is Down syndrome, which I don't think is a morbid condition, but somebody who has Down syndrome you are not going to count them as SADS because there is a lower expectation of life in any event.

A. Down syndrome can have congenital heart disease, but now in my larger study, we don't find an excess of Down syndrome with sudden death with a normal heart. Not now, maybe in our earlier study but it is not significant now as an entity causing sudden arrhythmic death.

Q. It is more the principle of the thing, that we are not necessarily looking for illness. The other example

about your approach -- at any rate in 2006 -- which was to exclude people altogether if they had Down syndrome or if they had a history of excessive drinking, even if there was no evidence that that caused the death?

A. At that time I was just learning my pathology with Professor Mike Davies who trained me and trained me very well, but then we have altered our diagnosis now but at the time, when he was the first person to lead on this, he excluded them yes to actually get down to a core of people where there would be no argument from anybody, he really got down to a core. Since then we have expanded it, I suppose.

Q. That is very helpful.

I just want to establish the approach.

We have endlessly heard the definition of SADS, that it is appropriate if there is no cardiac cause found at death, the toxicology is clean and no other cause is found.

I think that isn't really the complete picture, is it? If you were presented with a body that was found in a skip in a bin bag, obvious evidence of third-party involvement. I think even if the body was, as it were, clean of any evidence of third-party involvement, the mere circumstance in which they were found would be enough to exclude SADS?

which I would like to just put in your mind is people who drink excessively but there is no damage that can be observed. You would exclude someone in that category from a SADS death, not because you could find damage at autopsy but simply because there was evidence that they had drunk excessively?

A. Normally with an alcohol, if it is toxic, obviously the death is alcohol related.

Q. Yes.

A. If it is non-toxic levels there is an entity of sudden unexpected death with alcohol abuse, which we now accept as an entity. Where the person is either non-toxic levels or no alcohol in their system and have died suddenly and all we find at autopsy is a fatty liver, generally middle aged males. I don't think there was any evidence of fatty liver in this case.

Q. I think you may be missing the point of my question.

Back in 2006 anyway, you would exclude a diagnosis of SADS if you had a history or were told of a history of someone who drank a lot?

A. No, no, we would not exclude someone drinking a lot does not mean -- my goodness, obviously it depends on what you mean by "drinking a lot", I mean if there was a toxic level of alcohol found at autopsy.

Q. No, that is not what I am talking about. I am talking

A. Yes. If you are found in a bin bag, obviously there is a third party involved in whatever the circumstances are.

Q. I think the definition that we have is, for understandable reasons, not quite complete.

Similarly, if someone like Sleeping Beauty was found in the morning, perfect body, no sign of any cause of death but a note to the coroner saying, "I have decided to do away with myself", you would hesitate to ascribe that as a SADS death?

A. Yes, but obviously depending on the note and who wrote it --

Q. Of course.

A. -- and the proof of that naturally.

Q. It would have to be proved, I am just trying to get the principle of the thing, yes.

Can we just take a slightly more nuanced approach. You agree with me that in those cases the SADS diagnosis would not be appropriate. What if it was a little more nuanced and after the death a note was found, "If I die in the next two weeks, please inform the coroner or the police that you should be making inquiries in X, Y and Z direction". In other words someone who seemed to have foreseen the death. Would you ascribe SADS in such a case?

A. I would just say that there is sudden death with morphologically normal heart. It is up then to the other investigations to eliminate. I won't label it as SADS until -- and I always do that in my reports to the coroners and pathologists. This is a diagnosis of elimination once other investigations are completed, and then the family is referred --

Q. I think you were good enough when you were considering this with Dr Wilmshurst to agree that amongst the matters that you need to look at is the possibility of third-party involvement?

A. Absolutely.

Q. Yes. If I can just press the analogy with the suicide case, if you had not a note to the coroner but a history of depression and consulting the GP with suicidal thoughts and then the pristine body found, again you would hesitate I suggest to ascribe SADS?

A. Yes, but interestingly we have a higher than average instance of sudden cardiac death with psychiatric patients --

Q. That is an interesting observation.

A. -- even with or without suicidal tendencies there is a higher incidence --

Q. A topic which will be of great interest to my clients but not I think to this coroner in this case, but

certainly a very interesting explanation.

Just looking at SADS, I think you have mentioned Brugada and long QT syndrome, I think it used to be thought that long QT syndrome was the commonest but I think Brugada has come in on the outside now and --

A. Has overtaken. In the UK based studies Brugada now dominates.

Q. That is improved diagnostic techniques?

A. We think improved diagnosis.

Q. We can call those two the commonest, I appreciate long-QT encompasses a number --

A. In most studies they are the two commonest, but CPVT is also coming up -- I think it is for the cardiologist to comment on that.

Q. I understand. Dr Wilmshurst will give evidence but I think it is according to the literature often described as a disease of children and young people and whether and to what extent it is prevalent in an older age band, Dr Wilmshurst will help us?

A. Yes.

Q. Those three are really the commonest?

A. Yes, generally.

Q. If you would look at your joint statement, at paragraph 43.

A. 43. Yes.

Q. You are asked about the probabilities in relation to those three disorders and a number of factors are set out as to why Mr Perepilichnyy may not have died as a result of LQTS, your conclusion with Dr Wilmshurst was that, on the balance of probability, he didn't die as a result of LQTS?

A. Yes.

Q. As far as Brugada is concerned -- perhaps that should have been put logically first because it is the commonest -- again, on the balance of probability, he didn't die from Brugada and the same -- not the same --

A. All the entities.

Q. Not the same, CPVT, the question is what is the likelihood of CPVT causing Mr Perepilichnyy's death as a health 44-year old male without symptoms? You say, "We don't know, it occurs mainly in children". Is that as far as you could go on --

A. And also in adults. We cannot say it is exclusive to children. It is not exclusive to children.

Q. I understand that and I think Dr Wilmshurst in particular has some evidence that people over the age of 40 are occasionally affected.

Good.

Molecular testing, genetic testing, that is complex and I think we are going to hear evidence from another

witness, but I think you can agree with the general proposition that the yield from genetic testing varies according to the channelopathy in question?

A. Yes.

Q. I think for example Brugada has a comparatively low yield -- I say comparatively -- LQTS has a comparatively high yield?

A. Yes.

Q. I think you were not asked, but I will ask you, you are aware of course that Mr Perepilichnyy's tissue was tested for a genetic disorder and none was found?

A. Yes.

Q. That is --

A. I was aware of that.

Q. You were aware of that, yes.

Can I finally ask you this. You are obviously aware that insurance companies have vast data banks, dealing with questions of how long people live and why they die and what sort of questions one ought to be asking them in order to find out what the risks are. If, and I put to you this as a hypothesis, I am not asserting it as a fact, there is no evidence of that, it is an if question. If you or one of your PhD students were satisfied that there is a statistical link between overinsurance or high levels of insurance and early

mortality, would you put overinsurance on your list along with Down syndrome, excessive alcohol and the rest? If you were satisfied of that?

A. If you could statistically prove it, I would have to say it is there but I am not aware of any of my cases having over -- this is the first case I have been involved with insurance. It is not a risk factor that we have been -- interesting and all as it is, I am not aware of it and I am sure the cardiologist will comment the same.

MR MOXON BROWNE: It was an if question.

Yes, thank you very much.

Questions from MR STRAW

MR STRAW: Professor Sheppard, just a few questions from your report which is at tab 96. Do you still have that open?

A. Yes, I do.

Q. Bundle 3, tab 96.

Question 17 --

A. File 1, yes?

Q. It should be bundle 3.

A. Yes, file 3 and it is under 96.

Q. Tab 96.

A. Yes.

Q. Question 17, please.

A. It is on page 17, is it?

Q. It is internal page 5.

A. Internal page 5? Yes.

Q. You say there that the approximate portion --

A. Which paragraph?

Q. I'm sorry, it is number 17.

A. 17, yes.

Q. You say there that the approximate proportion of all sudden cardiac deaths which can properly be attributed to SADS is about 45 per cent.

A. Yes. But it can vary, depending on the age you study, depending on the group you are looking at, young athletes, sedentary people, it can vary from let's say Denmark to the Netherlands to the UK. But in our studies, yes.

Q. Question 21(m), so this is internal page 8 and right at the bottom of the page there is (m).

A. Yes.

Q. Is it right that 7 out of 10 people known to have long QT syndrome have mutations of known identified genes?

A. This is Dr Behr's booklet, prior booklet, so it is Dr Behr you will have to ask. I cannot confirm that. With more studies it is variable, put it that way but you get a higher yield in long QT but I cannot comment, it is a genetic question really for a geneticist.

Q. You have commented today that the overall rates from your studies by which molecular autopsies identify mutations in genes thought to be responsible for SADS is about 13 per cent.

A. Yes.

Q. In the case of the Manchester laboratory, where Ms Henchcliffe tested Mr Perepilichnyy's genes, can you tell us what the rate of detection there is?

A. I believe, Dr Wilmshurst may confirm, 30 per cent.

Q. 3-0?

A. 3-0.

Q. That is what your report says, 30 per cent.

A. Yes, but it depends on the group you are studying. If you are very confident it is long QT clinically you would get a higher yield than if you were not so clinically confident, it is the clinical combined with the -- and also the panels have increased from a few genes to multiple genes now. It is a very complex area. But, as I say, it is still in the majority, we do not find a significant at the moment mutation.

Q. Internal page 13, please number 46, towards the bottom.

A. Yes.

Q. Is it right that you and Dr Wilmshurst concluded that no individual channelopathy can be shown to be more likely than not the cause of death?

A. Yes. We have no evidence that favours one over the other that we have clinically, pathologically or genetics wise.

Q. The last area I would like to ask you about comes from over the page, item 50, where you are asked: is it agreed by the expert that a sudden unexplained death (SUD) cannot properly be attributed to SADS unless other possible explanations for the death have been totally excluded?

A. Yes, you have to exclude the others.

Q. In this particular case, the possibility of a poisoning would need to be totally excluded before the death can be attributed to SADS?

A. Yes, but you have to find your poison, haven't you?

Q. So the coroner, with the assistance of the toxicologists, would need to totally exclude the possibility of a poisoning before coming to the conclusion of SADS?

A. There has to be positive evidence of a toxic substance I would imagine, not a presumed toxic substance.

Q. Sorry, the possibility would need to be totally excluded before the conclusion of SADS could be come to?

A. Yes, in every case of SADS the possibility of poisoning or of forensic must be considered in every case and toxicology has always to be done to exclude that.

MR STRAW: All right, thank you.
Questions from MR BEGGS
MR BEGGS: Just one matter, you were taken to paragraph 17 in your joint report, which is internal page 5.
A. Paragraph 17, yes. Yes, here.
Q. Mr Straw, the gentleman behind me, took you to it where you said the proportion of sudden cardiac deaths attributable to SADS was 4 to 5 per cent. Doing the maths, that would appear to be something in the order of 1,200 to 1,500 people a year in the United Kingdom?
A. I think I gave you 800 originally --
Q. You did.
A. -- that is a conservative estimate.
Q. Yes, but in your report we get rather nearer to 1,500, don't we?
A. Yes, I believe that is the true incidence.
MR BEGGS: Thank you very much.
THE CORONER: Anything else?
Thank you very much, Professor, thank you.
MR SKELTON: Sir, after the lunch break we will hear from Dr Wilmshurst. We also need to address the Buzzfeed issue, which we may do shortly before Dr Wilmshurst gives evidence.
THE CORONER: Shall we do that at 2.30 or thereabouts?
MR SKELTON: Yes.

THE CORONER: Thank you very much.
(1.28 pm)
(The Luncheon Adjournment)
(2.46 pm)
THE CORONER: Yes.
MS HILL: Sir, you indicated you wished to hear some submissions or have some discussion about the issue of the Buzzfeed article, perhaps I can call it that but I know it has been widely reported by other news sources.
THE CORONER: I think you said you wanted to make some. It doesn't matter much, either way you are going to make some.
MS HILL: Are you content to hear submissions now, sir?
THE CORONER: Yes.
MS HILL: They will be very brief.
Submissions by MS HILL
MS HILL: I said that because I have had some discussions with your learned counsel and I understand it is on your radar, sir.
We have given thought to what the proper response to this article is and I am sure, sir, you have read it.
The key parts of the article for our purposes are the parts where the article quotes American intelligence sources, so I don't wish to address you on any other

part of the article but the key parts of the article as far as we are concerned are that it is reported that there are several, not just one but several, different American intelligence officials who have given an account to the journalist writing this article to the effect that there is in existence high level or high grade intelligence that has led to a report, I understand, by the Office of the Director of National Intelligence in America, with assistance from the CIA and the NSA which asserts with the, and the quote is, "high confidence" that Mr Perepilichnyy was assassinated on orders of the Kremlin.
The second significant factor about the article in our submission is that it is said not only that that report was provided to the British intelligence agencies, but that MI6 officers who agreed with its conclusion were apparently silenced is what is reported.
The issues it seems to us are that this, on the face of it, is highly pertinent information, it is intelligence that comes on the reported evidence from not just one but several different officials within the US intelligence, it is perhaps unusual for people to have spoken in such a way but it is potentially highly significant evidence that has been referred to.
The report appears to be based on reliable

intelligence, and if there is indeed in existence a report the nature of which is referred to, which I understand the BBC have made some inquiries about, I understand there was some reporting last night, sir, from the BBC which suggests they may have independently verified the existence of this report, I do not have that report in front of me but I understand it is not necessarily Buzzfeed alone that has reported it.
THE CORONER: One has to be so careful about that, I don't know what Dr Ratcliffe had seen when he said he had been reading all the papers. The Times, which I read this morning repeated a lot of this. I mean -- you mentioned the BBC but as you say the real point, I don't know whether that is independent or whether they are merely repeating this.
MS HILL: My understanding is there is mention in the BBC article, we are trying to find it now, or there was mention in the BBC report of the existence of the ODNI report, if I am wrong about that I will be corrected. My understanding is that the BBC ran with the story late last night and reference was made to the existence of this report.
The issue it seems to us, sir, for you to determine is what steps need to be taken in response to this.
The steps that we would invite you to consider are,

firstly, to consider a fresh Schedule 5 notice, sir, to the British authorities. Of course you will know that in April of last year a Schedule 5 notice was sent by the previous coroner to the British security agencies requiring them or asking them to provide all material within certain categories. The drafting of those categories was deliberately focused, but it did include a request for any evidence about effectively third-party involvement in the death.

A similar request should now in our submission be made, because there is a basis for thinking that there is in existence this report that it was provided from the Americans to the British.

Separately, sir, we would invite you to consider making a request to the American authorities to provide a copy of this report. I think there is room for discussion about the proper way in which these requests would be made. The previous coroner has taken the approach of issuing a request for information first, followed by the Schedule 5. It may be that that is sensible. We appreciate of course that there are difficulties in enforcement of any such Schedule 5 in relation in particular to overseas authorities, but given the potential significance of this report, and that it bears directly on the issues that you need to

determine, we do invite you to make those requests.

It seems to us that given the potential significance of this evidence, that it would be appropriate to make those requests as soon as possible and it would be appropriate to have regard to the potential impact of the answers to those requests as far as your conclusions are concerned.

Sir, we don't at this stage apply for any adjournment of the evidence or anything of that nature. It seems to us that we can press on and hear the medical experts but it does mean, in our submission, sir, that it would be inappropriate for to you return any conclusions on the Inquest until you had a sense of the likely response to those requests for information.

We have made other suggestions I think informally to those who assist you but we only press those two options. Essentially it is a request for the British authorities to turn over what they have in this regard and the American authorities too.

I should add that it is not terribly clear from the reporting when it is said this report was written and when it is said this report was provided to the British authorities. If in fact it was in existence and with the British authorities as at April of last year, then there is obviously a concern about why it wasn't turned

over in response to the earlier Schedule 5.

Obviously, sir, you have had sight of the matters that fall within the PII material, you have given a decision that nothing in the PII material is material for this Inquest. It seems to us therefore that this ODNI report cannot be within the material that you have reviewed, because it would be impossible in our submission to conclude that if this report does include intelligence that this was an assassination at the behest of the Putin administration that that was immaterial to the Inquest. We are working on the assumption that this report was not provided in response to the earlier Schedule 5, and, as I say, without knowing the date of the report, assuming it exists, it is impossible to know at this stage whether there is a concern about that or not but I simply put a marker down that if in fact it was in existence and was with the British security agencies prior to the receipt of that Schedule 5, then there is a concern.

Equally, sir, as you know, the PII process ran in this case, with the Government's involvement, from around April last year to -- I will be corrected if I am wrong -- around October/November last year when we got the judgment from Mr Justice Cranston.

One would have hoped that even if the report was

provided between April and October it was turned over as part of that PII process, but as I say that is a separate point perhaps, the immediate issue is to ensure in our submission that you have access to the material and that no conclusions are returned until you know whether you will receive it and if you are going to receive it, that the interested persons have sight of it if possible.

Submissions by MR MOXON BROWNE

MR MOXON BROWNE: Sir, if I could just say briefly, we have looked at this article and it seems to us possibly three sources of information that you might think it would be useful to pursue. Subject always to the PII certificate that has already been issued and your sight of that material. We don't know what is in that, it may be that what I am saying is otiose or has been overtaken by that.

First and foremost, because it appears to be what I might call the low hanging fruit which would be easy to identify and comparatively easy to get hold of, is the US Congress report. It is said in the Buzzfeed article that ODNI were approached and their spokesman said no comment beyond confirming that we prepared the report for Congress, so there appears to be --

THE CORONER: Who was the spokesman?

MR MOXON BROWNE: I'm sorry?
THE CORONER: Who was the spokesman?
MR MOXON BROWNE: It doesn't say. There is some evidence that the document does exist.
THE CORONER: Who was the spokesman who says -- where is it?
MR MOXON BROWNE: It is on page 5 of 16 in the version I have. The paragraph starts, "US and UK officials say ..." and it is near the end:
"... US intelligence officials told Buzzfeed that the report produced by the Office of the Director of National Intelligence with assistance from CIA ... asserts with high confidence. An ODNI spokesperson said the agency had no comment."
It sounds like an official response, on the face of it.
We would suggest there are two steps you could take.
First of all to ask the Americans, I am well aware that your writ does not run in Washington or whenever wherever this is, but since it appears that the Americans were taking the view or are publicly saying or are saying to journalists criticisms of the police investigation, now of course everybody can be satisfied that a full and thorough investigation is being undertaken under you, and it would appear to lie ill in the mouths of those who have that report to withhold it

from you, if what they are encouraging is a very full inquiry. At the very least, even if you didn't get the report, you would have a basis for saying, "Well, I tried and those who were criticising the UK authorities for not pursuing this properly declined to give us the information that they apparently had".
There would be some utility in it, notwithstanding that you have no, as I understand it, no ability to enforce any order or suggestion that you might make.
Of course as far as the UK authorities are concerned, it is different because they are within your jurisdiction.
Ms Hill has elided the information that I have just referred to with information that comes just before that in the report:
"Buzzfeed News has confirmed that British spy agencies secretly received intelligence from the US ..."
She is I think assuming in her submissions to you that that is the same as this Congress report and that therefore there is evidence -- I am sorry, she doesn't say that.
At all events there is there what would appear to be a second lot of documents which relate to information that has been received by British intelligence, which has come from America which connects Mr Perepilichnyy's

death to the Kremlin.
If the matter is not covered, as it may well be, by the existing PII certificate then we would ask you to either make or renew a request for that material. One of the problems that we have had throughout in relation to PII, it has been very difficult to ascertain from our standpoint what the immunity was being asserted in respect of. Not only did we not know what the material was, we didn't know what the relevant requests were.
That brings me to the third area, which is on my page 6 of 16, just above a paragraph that starts, "Medical checks acquired ..."
What it says is:
"He fled to Britain and blew the whistle in 2010, handing evidence to Swiss prosecutors and sources say becoming a prized asset for Western intelligence agencies investigating the flow of money."
Now "sources say" of the three that we have been looking at is the weakest and most vague and therefore in a sense the least promising, but it does relate, it is why I am making this submission, to material that we have previously asked for and which was the subject of a ruling by the senior coroner for Surrey.
What we asked for, very specifically, was documents in the possession of Surrey Police, and I stress Surrey

Police rather than the intelligence agency, which would suggest that there had been contact between Mr Perepilichnyy and British intelligence. He made an order -- as I am sure you know from your knowledge of the history of this case -- that that information be handed over. But at some stage and in circumstances which were never made very clear to us it was asserted by either Surrey Police or the Government -- that wasn't clear -- either that this material was subject to public interest immunity or that it was irrelevant.
In the end, the coroner made the ruling which for a long time was not published but which at our request was published and is now on your website saying that having reviewed the gist which related to PII, he was satisfied that this information was not relevant. In the end, as we understood it, it went off on the basis of relevance as opposed to public interest immunity and I have to say that we were left feeling that this matter had not been dealt with in a particularly open and frank way, because we were left simply confused. I don't know whether my current request that you look at this again is covered by what you have seen or whether it is not. If it is not, and if you feel that it would be worth pursuing, then I would invite you to make a Schedule 5 order. If it is said, as it might well be, that this is

something that attracts public interest immunity, then that can be identified and can be tried out.

But in our submission, at the moment it looks as if this question would be worth asking and I stress, and perhaps I can just read you from what Mr Travers said:

"I have already made requests for evidence to the Secretary of State for the Home Department and the Secretary of State for Foreign and Commonwealth Affairs which included inter alia any material held by the Security Service and Secret Intelligence Service respectively pertaining to ..."

Then there are a number of categories, and then he goes on:

"Whilst any material appertaining to issues identified in the questions in the paragraph above would be relevant to the Inquest, evidence as to whether Mr Perepilichnyy was or had been acting as a British -- [probably a word missing] agent would not in itself assist me in answering the question how did he come by his death.

"Consequently, in the light of the request for evidence that I have already made and the summary gist that I have seen arising therefrom, I do not consider the further requests for evidence which I am asked to make [that includes the category that we were seeking]

to be relevant to my investigation and as such they will not be made."

We were left a little bit confused as to exactly what the basis for declining to pursue that matter was, but in our submission it is obviously of absolutely central relevance to the issues that you have to decide. If Mr Perepilichnyy was in contact with British intelligence prior to his death, then you can see, obviously, what consequences and arguments might follow.

If that isn't plain I am very happy to say more.

Further submissions by MS HILL

MS HILL: Just to come back on it, I now have in front of me an informal albeit a transcript, the reference to the BBC is a bulletin that went out at 6.00 yesterday on Radio 4, and I am just reading out:

"Intelligence sources in the United States have confirmed to the BBC that they believe a Russian businessman ... who was found dead was assassinated."

This does appear to suggest there is some independent verification from US intelligence sources to the BBC as well as to Buzzfeed. I should make clear and I am sorry if I didn't before but my suggestion is that the wording of any Schedule 5 is, as the senior coroner before did, the subject of discussion and negotiation and submissions if possible because it would be

appropriate as Mr Moxon Browne has suggested to ask for not only the report but the underlying intelligence.

I am sorry if that was not clear from my submissions.

THE CORONER: What I am interested in is who is the ODNI spokesperson? Perhaps Buzzfeed can help us, because it says that the agency has obtained a comment which is confirming that a report has been prepared. That is obviously not a -- that is a comment apparently, if this is accurate, that was someone was happy to make but I am just wondering who the spokesperson is.

A MEMBER OF THE PRESS: If I could help, that would have been a request that went straight to ODNI press office. That would have been a formal on-the-record comment --

THE CORONER: A formal on-the-record --

A MEMBER OF THE PRESS: Which would have went to the ODNI press office. So we don't know, it is just a general press officer.

THE CORONER: Right.

Submissions by MS BARTON

MS BARTON: Sir, may I just say this on behalf of those who I represent. We have no knowledge of or possession of any of the documents referred to in that article.

It is perhaps regrettable that the article attributes, or doesn't attribute, the comments it makes

to any identifiable individuals.

Submissions by MR BEGGS

MR BEGGS: Sir, first of all we had no notice of the nature of the submissions that were going to be made. We haven't been privy to any back channel discussions, so we are not in a position to give an informed or considered response but there are one or two things we want to say.

First, I adopt what Ms Barton has just said.

Second, sir, I hope you will indulge me if I say that if we get to the end of this Inquest, we will be commenting upon the media coverage, which started a few weeks after Mr Perepilichnyy's death. We will be commenting on the nature and style of that coverage, we will be commenting on the likely sources and the purpose of those who may have been creating deliberately certain types of coverage.

Most importantly, of course, flowing from those observations, we will be asking you to consider very carefully, as indeed it is obvious you already are, the impact upon such coverage on what is meant to be an independent judicial forensic process, to what extent we may be asking has the media been primed with various theories which are then subtly able to gain what objectively might transpire to be entirely spurious

traction by repetition and adoption, I use adoption in inverted commas, into evidence.

To give you a quick example, Surrey Police will perfectly diligently report something that is told to them, as indeed any police officer would have to do, that is then replayed to the media as if the source is somehow Surrey Police or that they are acknowledging the accuracy of the report when they are doing nothing more than recording the fact of the report.

Indeed, I confidently predict that this very exchange is now going to be reported as lending even further credibility to some of the more florid media reports to date.

I remind the media, perhaps we become the solitary voice on this, that on the current state of the evidence, one very respectable viewpoint, one might think the overwhelmingly respectable viewpoint if we base our court proceedings on evidence, is that there is zero evidence of third-party involvement and yet there is credible evidence of channelopathy being the likely cause of death.

I am equally confident in making that assertion that that will not make the newspapers, because, of course, "Wealthy Russian dies through tragic genetic mutation" tends not to make headlines.

I am not going to make any comments on the suggestions made by my learned friends Ms Hill and Mr Moxon Browne, because as I said at the beginning I wasn't privy to the nature of their requests and I have to consider it properly with my client but might I make this request. If you are going to make any request and I could understand why without prejudice to anything I have said, you might consider you needed to do so, you might also be interested in asking Buzzfeed -- not that I expect them to cooperate with you -- but you might ask them when did they receive these stories. In other words, when were they placed with Buzzfeed and who placed them?

We have our theories but I am not going to indulge in the very thing I criticise others for doing. We have our theories as to the timing, we observe that Mr Browder's evidence got very little publicity for reasons to do with the coincidence of the general election, and we observe that shortly thereafter an alternative route to publicity was secured via Buzzfeed.

So that when you are making investigations, you will be as alert to the possibility, we say based on cogent circumstantial evidence which is so beloved of others in this case, that there may be forces, well-funded forces,

out there deliberately manipulating the media.

Indeed we have ourselves heard what we consider to be cogent rumours that that is precisely what is going on and that you are quite improperly being placed under pressure by skilful media manipulation.

Sir, that's all I say for the moment, but I may come back later.

Submissions by MR SKELTON

MR SKELTON: Sir, may I just limit my remarks to these. You will proceed carefully and with caution whenever you respond to media reporting which does not relate to evidence that has been proffered before this court by any of the IPs or any other persons, including the British Government and after consideration of those articles and any responses to those articles, including it seems further information that comes from the BBC.

May I say this in respect of the PII just so there are not any misapprehensions, and really I am repeating what was said in front of Mr Justice Cranston based on the statement by the then senior coroner who was then in charge of the Inquest. He said in his statement that there were two Surrey Police documents that were included within the PII application because the equity in those documents as far as the public interest was concerned rested with the Government. In this case it

was the Home Secretary who brought that application.

The response to the two requests made by Mr Travers, which went to the Home Secretary and the Foreign Secretary, responsible respectively for the Security Service and the Secret Intelligence Service and the confidential gist, the aim of which was to corral in summary form the information that the Government gave in response to the senior coroner's requests.

In making that clear in his statement Mr Travers also adverted to the fact that the senior coroner had made clear publicly that the Government had indicated to him that it had searched more widely for relevant material pertaining to Mr Perepilichnyy's death.

That indication was given in order, it was hoped, to neutralise any speculation or assertion about Mr Perepilichnyy's contacts with British intelligence and the like in the index period prior to his death.

Sir, as you are aware, it is an unfortunate stricture of public interest immunity that one cannot say what one reads even where it may not be relevant and where it is relevant, one is limited in its use. I cannot say to this court what is contained within that material, beyond what you have indicated publicly that it does not materially assist you in determining how Mr Perepilichnyy has died. Nor can I say the period in

which it covers or anything more beyond what has been said publicly.

As far as I can assist you today, sir, I think that is the limit, really.

THE CORONER: Yes.

MR SKELTON: I would suggest that careful consideration is given now to what you have heard.

THE CORONER: Yes. Well I think that is the way forward.

Ms Hill, Mr Moxon Browne and Mr Beggs I have heard what you have all said. Not everything you have said, Mr Beggs, because you have explained that there might be more but I have listened to that and I shall consider everything you have said.

MS HILL: Thank you, sir.

THE CORONER: Right.

MR WASTELL: Sir, turning back to the evidence in this case, we have one further document to read, it is found behind tab 27, that is page 133 of the core expert bundle volume 1, it is a post mortem report from a Dr David Rouse, a fellow of the Royal College of Pathologists, a forensic pathologist indeed. It is dated 17 June 2016.

It deals with his own, indeed the second forensic post mortem.

THE CORONER: Yes.

MR WASTELL: Sir, you will see it is a post mortem from Dr Rouse, on the second page, dated 17 June 2016. I think it is uncontroversial that the post mortem itself was the date at the top, 3 December 2012.

Sir, you can admit this on the basis it is unlikely to be disputed, in the usual way, and I have dealt with the full name, the nature of the evidence and of course you must announce that any interested person may object and is entitled to see a copy, which they already have.

THE CORONER: Yes, thank you, I confirm all those things.

MR WASTELL: Dr Rouse writes this 3 December 2012 -- I should say before I start, a minor health warning there is for those not used to post mortems, some of the details are vivid or graphic.

THE CORONER: Yes, thank you.

Statement of DR DAVID ROUSE (read)

MR WASTELL: "Acting on instructions from HM Coroner for Surrey I attended the Royal Surrey Hospital mortuary Guildford where I performed a second post mortem examination on the body of an adult male identified to me by the mortuary staff and the appropriate namebands as being that of Alexander Perepilichnyy, stated age 44 years. Those present included mortuary staff.

"Prior to the examination I was shown the following: report of Dr Ratcliffe.

"Subsequent to the examination I was shown the following: statement of Dr Fegan-Earl dated 10 February 2014.

"The body was that of a middle aged Caucasian male, 186 centimetres in height and weighing 93 kilograms at the initial examination. The body was naked at my examination. There were changes of marked decomposition consistent with the post mortem interval. There were the usual post mortem incisions.

"There were abrasion to the outer third left eyebrow, left cheek prominence and left corner of the mouth, together with some possible abrasion on the left side of the nose. There was a band of abrasion to the left knee, 5 centimetres in length and a 1 centimetre area of abrasion right knee.

"There was a 1 centimetre abrasion lower left forearm, (left ulnar styloid process). Venipuncture mark was present to the left antecubital fossa (related to the previous cannula).

"Internal examination. All organs had been dissected to current standards. The heart had been submitted to Professor Sheppard for expert opinion. Samples had been submitted for toxicology. There was no obvious odour to the body other than that associated with decomposition. No obvious ulceration was noted to

the mucosa of the upper gastrointestinal tract. Resuscitation type fractures were noted to the ribs (left third to fourth ribs and sternum).

"Opinion (1) the body was that of a middle aged male with no evidence of obvious natural disease which may have caused or contributed to death.

"(2) there was no evidence of significant blunt trauma to account for the death.

"(3) I have been informed that there were no significant toxicological findings.

"(4) no samples were taken at my examination.

"I note and would agree with the opinions expressed by Dr Fegan-Earl.

"Dr Rouse, dated 17 June 2016."

MR SKELTON: Sir, Dr Wilmshurst.

DR PETER WILMSHURST (affirmed)

Questions from MR SKELTON

MR SKELTON: Dr Wilmshurst, will you please state your full name to the court, please.

A. Peter Thomas Wilmshurst.

Q. You are by profession I think a consultant cardiologist?

A. Correct.

Q. How long have you had that post?

A. Well I have been a consultant cardiologist since 1987. I was at St Thomas's then but I have moved. My current

post, 1991.
Q. Do you hold any other specialist qualifications?
A. I am accredited in general medicine and intensive care.
Q. Thank you.
You were originally instructed I think by Mrs Perepilichnaya; is that correct?
A. Correct.
Q. Having had your report proffered to the court, the then senior coroner who was in charge of the Inquest adopted you as an expert of his own and you are now effectively an independent expert giving evidence to the court?
A. Yes.
Q. Can I confirm that your previous instruction has not in any way influenced your opinions?
A. Yes, that's right. It hasn't influenced me.
Q. Thank you. I think it is fair to say that you are you are not someone who is afraid of giving independent views, in fact you have a reputation of being someone who has done so in the past publicly and to considerable effect?
A. Yes, I suppose so.
Q. By which I mean you have been involved previously in whistleblowing for example?
A. Correct, yes. Yes, I have reported doctors to the GMC and they have been struck off and so on. So, yes.

Q. You produced several documents for the court. I think if you go to file 2 of the expert bundle, you should see under tab 55, page 419 your initial report.
A. Tab 55?
Q. 55, page 4189.
A. Yes.
Q. That is dated 4 August 2015?
A. Yes.
Q. That was your report, originally commissioned by Seddons for Mrs Perepilichnaya?
A. Yes.
Q. You then produced some answers to questions which were approved by the senior coroner. They are found under tab 56 at page 439, dated 25 October 2016.
A. Yes.
Q. You recall those?
A. Yes, yes.
Q. Yes. By that stage you had changed into the coroner's expert, as opposed to an interested person's expert?
A. That's correct.
Q. I think you also produced what I am going to term a supplementary response, because I think that is the phrase that you use, which is under tab 65, page 546, which is dated 20 December last year.
A. Yes, that is because I independently had some additional

information, on minor points but I thought it would be useful for the coroner.
Q. Yes. You also met with Professor Sheppard to discuss jointly cardiological and cardiopathological issues recently?
A. That's correct.
Q. As far as the facts within those reports are within your direct knowledge, do you stand by the truth of those facts?
A. Yes, I would point out that each of those has been at different stages when I have had different sets of documents. When I gave my original report, I have listed the documents that were seen.
Q. Yes.
A. Subsequently I was asked -- in fact that report was a slightly unusual report in that it asked a series of specific questions rather than a general, "Look at these documents, tell me the cause of death".
Q. Yes.
A. Then subsequently I had other documents given to me, particularly before the joint meeting with Professor Sheppard. So I was given another bundle, some of these documents were new to me, so when you say I stand by those, you have to take into account that each of them were true at the time that I had the

documents on which I based them, if you see what I mean.
Q. Absolutely. It is fair to say that your professional opinion in response to the information given to you and your analysis of that information has evolved over time during the course of your instruction?
A. Yes.
Q. Would I be right in concluding though that the joint statement, which is the most recent expression of your opinion, is really the representative opinion as at today?
A. Yes. I would also point out that that was an expression of responses to questions --
Q. Yes.
A. -- which of course is not necessarily entirely the same as an opinion, you know, if you say: what is your opinion on this particular thing?
Whereas if you provide a series of leading questions, that is not entirely the same as your overall opinion.
Q. Yes, but you are not saying though that you have been somehow sort of corraled into a position which you are uncomfortable with?
A. No, I am not.
THE CORONER: You are just saying there may be, beyond the specific questions, other things that you can say?

A. That's right.
MR SKELTON: If there are such matters and you feel they are significant and I don't elicit them or indeed those who speak after me or ask questions after me don't elicit them, will you please identify them?
A. Yes.
Q. I hope that we do cover all the salient points, but if there are some things burning then please do say. Are there any from the off that you would like to mention?
A. No, not particularly.
Q. Thank you.
Questions really about the presentation of someone that dies from a sudden cardiac cause. You will be aware of the evidence of fact, and ultimately it is for the coroner to decide whether he accepts or rejects that evidence but if a person presents while running as struggling, grimacing, hand across the abdomen or stomach, looking unwell, white in the face, giving the impression of a lack of fitness, and then drops down to the floor, what from a cardiologist's perspective are you thinking is likely to be going on?
A. I think that is very, very difficult to say because of course when I run I look pretty unwell, you know, so I think that it is rather depends on how unwell you look, I guess.

I mean it is, you know, if someone is very breathless and pale it may be that they have a cardiac problem, if they have got their hands on their chest it might be that they have a cardiac problem, pain and discomfort, but it could just be that they are very breathless running up hill.
My understanding is that that is based purely on observation of someone driving past in a car who says, "This chap doesn't look too well". If I made a diagnosis on that basis in, you know, in a hospital, I would be before the GMC in no time I think.
Q. Yes, I mean we don't know for example if Mr Perepilichnyy was suffering chest pain, which would be more significant?
A. Yes, you cannot say from that for sure.
Q. There are certain patterns of chest pain which are also significant, you can get chest pain with radiation into the arm and so on which for a cardiological perspective can be a sign?
A. But it is very variable, it's very variable. How can you know where someone's pain is if unless they tell you, you know, so I don't think you can say for sure.
Q. You cannot draw any reliable conclusions from those matters that I quoted to you or elicited to you?
A. No, except that he was thought to look unwell. I think

that is all that anyone could say.
Q. Can you say that they are consistent with an arrhythmic event manifesting itself?
A. Well, if they are consistent with an arrhythmic event, they are -- I don't know how far he managed to go, run, beyond that point, but if you look at some people who have had arrhythmic events, if you have a cardiac arrest you will collapse in 4 seconds. There was the footballer at Tottenham who suddenly stopped, staggered for a couple of steps and collapsed. A cardiac arrest is not consistent with running another 50 or 25 yards.
But you could get some arrhythmic event, such as ventricular tachycardia, where the heart goes into a ventricular arrhythmia but is not pulseless and is very inefficient. The blood pressure would drop, you would get breathless, you would feel very unwell but you would be able to stagger on. In fact some people walk into hospital with ventricular tachycardia and then we shock them out of it. So you can have ventricular tachycardia.
That will often deteriorate into something called ventricular fibrillation, which would cause you to become unconscious within a matter of seconds.
Q. Is there much information or research on the preceding symptoms and signs before a sudden death from a cardiac

cause of cause unknown? Not your classical heart attack from a blocked coronary artery, but the kind of thing that what we are looking at in this case where someone may have an occult channelopathy, are there a set of signs and symptoms that classically one looks for?
A. In people who die without preceding history of more minor symptoms, such as transient loss of consciousness, there isn't very much data.
The very limited data -- well, in fact there is, I would say, no data. There are cases reported where people have had transient loss of consciousness, for example blackouts or palpitations and we put on them a 24-hour tape or implant a reveal device which monitors their heart. Most of them in fact don't have any problems, but some of them occasionally die while the tape is on.
Occasionally, this is a very small number of people reported, you can see arrhythmias before the fatal arrhythmia, but some of them go instantly into a fatal arrhythmia. Sometimes, for example, you would go into ventricular tachycardia and that may be sustained for minutes before you go into ventricular fibrillation and the heart stops. Or, conversely, your heart may slow, slow, slow and then you go into asystole, so you would be conscious and then the heart would stop. If you had

a progressive conduction defect your heart is getting slower and slower and then stops.

That is less common than it going faster, faster, faster and then so fast that there is no output.

Q. There is some suggestion in the evidence that Mr Perepilichnyy was faintly breathing after his collapse into the road.

What does that allow you to infer with any degree of reliability about his heart at the point where he is still breathing?

A. Nothing. I mean you can -- I mean it is very common when people die, and you see in the hospital, in the coronary care unit, people die, you either give up, resuscitation, you have been attempting resuscitation and they will make what we call agonal breaths. This is sometimes distressing for relatives, we often actually don't let the relatives into see the body until we are sure that agonal breathing has stopped. They are actually dead but there is a bit of neurological activity which makes them gasp, so the heart could have stopped five minutes before and you give the occasional breath, so that doesn't actually mean anything at all.

Q. Falling down without protecting yourself implies a loss of consciousness, cerebral consciousness?

A. That's right, yes.

Q. Vomiting?

A. No. I think being unconscious you relax the sphincters, so you -- well if it is active vomiting, it is actually more likely regurgitation of what is in your stomach rather than, you know, contraction and vomit. I mean it is more regurgitation than vomiting.

Q. What causes the regurgitation after such an incident?

A. You are lying flat so your stomach is no longer lower than your mouth, that is one good reason. Just lying flat you have done away with the gravity holding the food in your stomach.

Q. Resuscitation?

A. When you resuscitate you are pressing so sometimes -- also, if you are doing for example mouth-to-mouth resuscitation you are blowing in, often when you are blowing into the mouth, you are trying to get by tilting the head and jaw to get the gas into the lungs, but you almost invariably get gas into the stomach and the abdomen will get inflated if you are doing mouth to mouth.

That is why, in fact, the ambulance crew in this case put an endotracheal down to protect the airway.

(1) so they could ensure that the gas they were putting in, the oxygen was going into the lungs.

(2) because it is a cuff tube, you know you put down

the tube and it has got a cuff down it so you put air into the cuff so it seals it, any regurgitated fluid from the stomach is not aspirated, because that is a big problem on the ITU when people have recovered, if they recover from this, the big problem is the acid they have regurgitated into their stomach gives them lung damage, which is a problem. It is hard to place any reliance on that.

THE CORONER: Are you saying that, as it were, if you were not an ambulance man, in doing mouth to mouth you could inflate the stomach, I just wanted to follow, but were you saying that because of the some of the equipment that the paramedics used they obviated that risk?

A. Yes, but he had --

THE CORONER: He had before, but that is part of the reason for them using what they use?

A. That is the reason, they do it.

THE CORONER: Yes.

A. That is the reason they put an endotracheal tube into your, you know, your oesophagus when you have an operation. It is why, of course, when you have an operation they say we don't want -- we want you starved for six hours, because they know when they anaesthetise you, you will be unconscious, laying down and you will regurgitate into your lungs and acid in the

lungs causes a lot of harm.

MR SKELTON: The regurgitation which was described by one of the lay witnesses during his attempt to give CPR was consistent with cardiac arrest and as it were the relaxation of the preventative sphincters and so on that would stop that from happening?

A. It is consistent with unconsciousness for any reason.

Q. Thank you.

In terms of other pathological signs you deal in the joint statement and I think before in your evidence with pulmonary oedema. There is no reason to go into the complex physiology of that, but is pulmonary oedema a sign which is consistent with an arrhythmic event leading to arrest?

A. Yes, it is consistent with a great many things leading to death.

Q. Yes.

A. Pulmonary oedema, as I explained in the joint statement, is fluid coming out of the alveolar capillaries into the alveoli. The thing that keeps fluid in the alveolar capillaries is albumin, which has a hydrostatic attraction for water. If you increase pressure in the alveolar capillaries, fluid will come out into the alveoli. That commonly occurs in people who are walking around in the street who have heart failure, they get

pulmonary oedema, a bit of pulmonary oedema, and they are breathless. Because the heart is inefficient and the filling pressures of the heart are elevated so the alveolar capillary pressures are elevated beyond that which keeps the albumin will keep the fluid in the alveolar capillaries.

Of course, when you have a cardiac arrest and someone is doing massage, Mr Perepilichnyy did not die when he collapsed. He died when they gave up -- legally when they gave up cardiac massage. For 45 minutes or so he was in heart failure. Heart failure is not the heart stopping, that is a cardiac arrest. Heart failure is inefficient contraction of the heart. That usually occurs because the heart is damaged, you know the people I see in my clinic with heart failure have damaged hearts but here if you are having someone keeping your circulation going by compressing your chest, you are effectively in heart failure, you are mimicking exactly the situation. Your alveolar capillary pressure will be about 40 in that circumstance, at cardiac arrest.

Normally if you have a normal albumin of 40 grams per litre, you go into pulmonary oedema when your alveolar capillary pressure exceeds 24.

Q. Can you say whether pulmonary oedema is more or less consistent with other forms of death, like for example

poisoning which is of course a critical issue for this Inquest?

A. Sorry?

Q. Is it more or less consistent with poisoning compared to some form of arrhythmic arrest?

A. Well I am not a toxicologist, so I wouldn't like to stray outside my area but I do know that some poisons cause the heart to pump inefficiently and some cause cardiac arrest. So both of those could cause pulmonary oedema. I also know that some poisons damage the alveolar membrane, so fluid can pass out of the alveolar capillaries into the avails by that mechanism, and I am sure the toxicology experts can say more but essentially, many poisons kill you by stopping your heart or making it work inefficiently, so it is --

Q. It is often associated with heart failure, the cause of the heart failure is to be determined?

A. Yes.

Q. But it can be caused directly by a poison and certain types of poison, but you are not a toxicologist?

A. That's right.

Q. Thank you.

You heard, I think, and were in court throughout Professor Sheppard's evidence?

A. Yes.

Q. Is there anything you would like to add to her comments about the macroscopic and microscopic examination of Mr Perepilichnyy's heart and the conclusions she drew from those?

A. I am not a histopathologist and I accept her expertise, really.

Q. Do you also accept, and I think you agreed on all points in the joint statement, her definition of sudden arrhythmic death syndrome or SADS?

A. Yes, it is -- well, the definition that applies here is you die within that one hour of onset of symptoms. There is an alternative in that you are found dead having gone for example to bed well, but in this situation you are found dead within one hour of being asymptomatic, with no evidence of trauma, with no evidence of a life threatening medical problem, such as cerebral haemorrhage, pulmonary embolism or massive haemorrhage into the gut. That there is no cardiac cause, no toxicological cause and then you say the cause of death is sudden arrhythmic death syndrome.

I think that is what she said, is that correct?

Q. I think in a nutshell it is pretty close, yes.

A. Yes.

Q. As far as ion channelopathies are concerned, how commonly have you seen those in your practice over the

years?

A. Well, they are quite common. Most commonly I see them in people who have come to me because a close relative, usually a first degree relative has dropped dead suddenly. Usually they have done so without warning. That is their first event, their relative is not known to have a channelopathy and has dropped dead suddenly and then other people, family members, want to be tested, quite reasonably.

Q. Again I think you heard the data that was put to Professor Sheppard about the percentage of diagnoses that are made through molecular genetic testing post mortem being around 13 per cent?

A. Yes, well I mean I wrote and had quite a lot of discussions and correspondence with the Manchester lab with Dr Eaton(?) there and got them to audit their data, and their finding was about 30 per cent.

Q. That is purely on the genetic testing?

A. On the genetic testing of people who die suddenly, who are thought to have sudden arrhythmic death.

30 per cent they pick up a channelopathy gene that is known to be pathogenic, in other words it is called class 5 where it is definitely in other people, in other families, has been known to cause sudden arrhythmic death or is thought to be highly likely to be

pathogenic. That is to say it is not known for certain that it causes sudden arrhythmic death but the position on the gene, from that they can work out the confirmational changes in the protein that the gene codes for, which suggests it would have a similar pathogenic effect on the particular ion channel. That is about 30 per cent in Manchester.

In fact in Oxford they have not found any so far on a sudden arrhythmic death on a molecular autopsy that was positive.

Somewhere between 0 and 30 per cent pick up is what I am saying.

Q. Yes, well it may be that that explains the figure which was drawn from international data of 13 per cent, because as I put to Professor Sheppard it was based on cases from Holland, New Zealand, Denmark I think, and United Kingdom and a wide cohort, the widest cohort I think so far?

A. Yes, but I think probably not Ukraine or Russia, and there is considerable variation between countries in prevalence of particular genes because these are inherited. So where you have particular features, in particular countries, inherited features, less so now when we have globalisation but 50 years ago if you had gone to Scandinavia, you would expect to see most of the

population with blond hair or red head, because they lived in communities and the particular gene for hair colour was in that particular community.

Some particular genetic abnormalities are particularly common in say the Mediterranean parts of Europe but not common in northern parts of Europe. Thalassaemia you get in Greece or Italy but you don't get that in northern countries, except for people who have migrated.

Q. Is there any reliable data available about Ukraine? Mr Perepilichnyy as we said born in West Ukraine, lived in Russia, I don't know if he is ethnically Ukrainian, if that is an ethnically identifiable grouping?

A. I am not an epidemiologist, but as far as I know there is none.

Q. From what Professor Sheppard was saying in fact the collection of data is less impressive outside of the UK for reasons which she explained, partly from the coronial system there are investigations of any sudden adult death in the United Kingdom which leads to a better data set?

A. Yes. Yes. I would say that is almost true.

Where it doesn't happen, of course, is in elderly people. Elderly people who die quite suddenly, people don't look. Even if the heart is normal, they just

assume -- in fact I had a personal experience last year when my next door neighbour collapsed and died and her husband rang me and I ran round with my wife, who was a cardiac ward sister, we tried to resuscitate her, we were unsuccessful and the ambulances came, she was in VF and could not be resuscitated. The post mortem was entirely normal and the coroner just said well it must have been hypertensive, so she had a sudden arrhythmic death which, you know, the coroner did not refer.

Q. Without getting caught in a down an epidemiological foxhole, as it were, is it the case that if those sorts of deaths were investigated -- so the 80-year old who dies peacefully at home if that death were investigated you were likely to find a cause or are you saying ultimately that is going to be by exclusion categorised as sudden death?

A. Well it is sudden death, and --

Q. It is sudden but if you undertook pathological investigation of an elderly person would you be likely to?

A. No, there was a pathological investigation at post mortem, which was normal including the heart.

Q. I was not talking about your individual, I was talking about the population in general.

A. What I am trying to say is -- I am saying that

I actually think the figure -- you said because of the coronial system we pick these up, I am saying --

THE CORONER: You are saying there are still some that are not picked up?

A. I am sure there are not, and I think that people don't look so hard for causes the older you get, the 95-year old who collapsed, "Well, old age, isn't it", in fact you can put "Old age" on the death certificate.

Q. Beyond genetic testing --

A. Yes.

Q. -- if one assumes that you don't have any reliable clinical data beforehand, which we will come on to, from the person who died. What can you do post mortem to diagnose ion channelopathy?

A. Well you can only do the panel of -- well, sorry, you can do the panel of tests on genetic material from the deceased and you can do tests on relatives. You can look at particularly first degree relatives and they will tell you about dominantly inherited diseases, but there is a problem if you are dealing with recessively inherited diseases, where the deceased is homozygous, has two copies of a recessive gene, because their first degree relatives are likely to be unaffected. Or rather there is a 1 in 4 chance of any sibling being similarly affected, statistically.

Q. Professor Sheppard said that you can do the genetic testing on the deceased but that in her view is unlikely to get more than a 13 per cent probability of finding a positive condition, although in Manchester that may be higher for reasons which are not entirely clear but we can ask Dr Homfray about.

You then have the family testing which, combined with the patient testing, takes you up to about 40 per cent?

A. Yes, and that picks up things that are obvious on an ECG.

Q. The clinical data, one might have gone to see a cardiologist beforehand or even your GP, and they have done a basic ECG, which in retrospect you can see has picked up something?

A. Yes, but I was actually thinking about the relative. If the relative goes for an ECG, an echocardiogram, for example, you might see on the ECG in a first degree relative there was a long QT, which would influence you to thinking that that relative has long QT syndrome.

Therefore, if it is in a first degree relative, there is a 50 per cent chance exactly of the deceased having the long QT syndrome, if they have Brugada syndrome, similarly the ECG would tell you the deceased had exactly a 50 per cent chance.

The pick up rate of course is dependent on how many first degree relatives you have and how many are tested. If you have no surviving parents, no siblings and no children that doesn't work. If you have got, you know your parents are alive, you have six surviving siblings and, you know, eight children, then the chances of picking something up is very high.

Q. But still less likely --

A. No. If the abnormality is present, but yes, but still --

Q. You are still unlikely to get the answer?

A. Oh, you are still unlikely to get the answer, sorry.

Q. Yes.

In terms of identifying -- we will come on to the individual channelopathies and I would like to understand briefly your analysis of each of those as they apply to Mr Perepilichnyy. As a matter of generality, is it possible based on post mortem data without genetic testing of that person or their family, to say which channelopathy someone has died from?

A. On the balance of probability, no.

Q. How could one approach that task beyond the genetics and the clinical information, which we don't have in this case?

A. Well that is all you have, but on the balance of

probability, if someone died from a channelopathy, the chances are you would not identify which the channelopathy, the sudden arrhythmic death, that is the evidence we have.

I think it is also worth pointing out that if someone were known to have a channelopathy and were found dead suddenly, but had a post mortem cerebral haemorrhage or a knife in their back, you would not say they died from the channelopathy because they wouldn't fulfil the criteria of sudden arrhythmic death.

Q. Yes.

Going through the individual ones that you identify in your report, one which we have already heard about is long QT syndrome, does that remain the most common channelopathy in your view?

A. Well, it depends how you work out what is the most common.

That is the difficulty. You see the long QT syndrome and Brugada syndrome have features that you can see on an ECG. Some of the other channelopathies are associated with a normal ECG. Therefore diseases which have an obvious characteristic, an easy test to find, are more often identified, even if they are less common, if you see what I mean.

Q. There is an artefact of the investigation because you

can get a positive from one but the negative doesn't necessarily mean you haven't got something else?

A. Yes, and in fact people who -- some people who have got the long QT syndrome, about a quarter of them who have the gene for the long QT syndrome do not have the phenotype for the long QT, which is to say they don't have a long QT on their ECG, they don't have any symptoms. They have got the abnormal gene and they don't have the phenotype.

It gets even more complicated with CPVT, because even more people have the genes, you know they have the gene because the child has the gene, but three-quarters of their parents who have the gene have no symptoms.

Q. CPVT -- I will not try and pronounce the full name because the stenographers will burst into tears -- could you describe the difference between that and the QT syndromes that you have identified?

A. It presents often as blackouts or palpitations and the ECG shows what we call ventricular tachycardia and it has to be a ventricular tachycardia that can go round the heart in more than one direction, so that is polymorphic -- "polymorphic" means more than one appearance. The ECG is polymorphic but they have a normal resting ECG. These people can go on to get ventricular fibrillation and cardiac arrest and die, but

it is actually becoming increasingly recognised that it is present in larger amounts of the population than previously recognised.

Q. If Mr Perepilichnyy had presented with signs or symptoms which were consistent with blackouts for example, if he had presented in that way. Would you start to be able to form a basis for saying it is likely to be one or the other of any of the particular ones, for example CPVT?

A. If he presented with blackouts, if he had had blackouts before the actual cardiac arrest you mean, well, you could do an ECG and you might see some abnormalities if he had long QT or Brugada syndrome, but if he had CPVT his ECG would look normal. If he had early repolarisation the ECG might look abnormal, because quite a lot of people who have transient cardiac arrest have early repolarisation.

If he presented with blackouts, you might see something on the ECG that would give you a clue, but if he had CPVT, his ECG would look normal. Unless you actually did an ECG when he had just collapsed or had palpitations, you would see a normal ECG.

Q. We don't have an ECG as far, as I am aware there isn't one in existence. We don't have a history of suspicious, it seems, signs and symptoms, blackouts and the like.

What we do have is a man collapsing while running with no genetic abnormalities found, so a very limit set of information. Is there anything you can say on the basis of the information you have seen about which of the particular conditions is really more likely than not?

A. No, you can't say. In fact we know that something like three-quarters of people who collapse and die and are thought to have died from sudden arrhythmic death syndrome, you know the coroner says they died of sudden arrhythmic death syndrome, we know that in three-quarters of them you don't find any abnormality on genetic testing. That is I think because we don't know what all the genes are.

It may be -- I mean we are finding more and more genes all the time that are responsible for abnormalities but, you know, as we go through, we find more genes that cause abnormalities.

The problem is finding the genes when the phenotype is unclear.

That is to say, for example, if someone dropped dead suddenly, and then in 20 years time one of their children dropped dead suddenly. If you want to do genetic linkage, you have to have lots of people who you recognise the disease so that you can find the gene.

You need large families where you can identify. The problem is that if, you know, one person drops dead now, because they have an abnormality that is unrecognised, when their child drops dead in 20 years with the same problem, and their grandchild drops dead in 40 years with the same problem, the chances are you will not have the genetic material from each of those three people to find out what is different about them from the rest of the population, because all of us have many, many innumerably different genes, that is why none of us look the same.

Q. Can I try and summarise your evidence, if that is possible.

It's unlikely that you are going to find a genetic marker from the deceased?

A. Correct.

Q. And one has not been found.

Most people who die of sudden adult death do not present with prior symptoms, and that was a chart I showed to Professor Sheppard earlier from the Lahrouchi paper, 75 per cent do not present.

We haven't in this case got any signs or symptoms in any event presented to us, we don't have family testing, so on that basis -- also, I think you were saying it is not reliable to draw any conclusions about the inherent

likelihood of particular conditions and individuals, because some of them may test positively but you cannot rule out other tests based on the things like ECGs and the like, you don't quite know how many people are actually suffering from these conditions?

A. That's correct, yes.

Q. Putting all that together, can one draw any conclusions about which channelopathy, if it was a channelopathy, Mr Perepilichnyy died from?

A. No, you can't.

Q. I think what you were saying is that it is possible that he died from poisoning, there is nothing you have seen which effectively makes it quite clear from your perspective it is a channelopathy?

A. No, that is right, I can't say he didn't die of poisoning, no.

Q. Is there anything that one can put in the balance from your perspective, your cardiological perspective, tips it towards one or other of those causes of death?

A. I, of course, haven't sat through the days of evidence and I don't know all the evidence, and I won't have seen it. All I can say is that if in a normal situation, normal situation of a man of 44 out jogging who collapsed and died quite suddenly, the idea of him dying from poisoning would not be raised by anyone. My

understanding is that there would be limited toxicology screening, mostly confined to testing for recreational drugs, cocaine in particular is one that causes sudden arrhythmic death -- sudden cardiac death.

In that situation I think it would normally be ascribed to sudden arrhythmic death if there was no post mortem finding and suddenly one died suddenly.

Sorry, have I been clear?

Q. Yes, no -- if one goes through the exclusion, as one properly must, to get to sudden adult death syndrome, that includes toxicological investigations as appropriate and nothing positive results, then ordinarily you conclude sudden arrhythmic death?

A. That's correct.

Q. In this case, we know now it is no longer possible to exclude certain types of poison.

A. Yes, I have no --

Q. At least that is what our toxicologists tell us in their joint statement.

A. Yes, I can't say anything about that really. I mean I accept if that is what they say, yes.

MR SKELTON: Thank you.

Questions from MR MOXON BROWNE

MR MOXON BROWNE: Dr Wilmshurst, in Mr Skelton's questions and perhaps to a lesser extent in your evidence there

has been quite a lot of emphasis on the fact that this death occurred either in the course of or very shortly following exercise?

A. Hmm, yes.

Q. I think that in general, death from a channelopathy is not something which is usually associated with exercise?

A. That's correct, it is more commonly when resting, that's correct.

Q. I think with Brugada in particular the majority deaths, by a large majority, take place during sleep?

A. Yes. Yes.

Q. I think that Professor Sheppard has told us, I think in fact you have agreed, that if you take across the board, that an element of exercise only figures in I think 13 per cent of cases, really quite small?

A. Correct. Something like that, yes.

Q. If Mr Perepilichnyy did die as a result of a channelopathy, which is obviously a possibility, the coroner might well conclude that it was a coincidence that this happened following exercise?

A. Or during, yes.

Q. Or during, yes. Thank you.

In your original report, there is a certain amount of mention of Wolff-Parkinson-White.

A. Wolff-Parkinson-White syndrome.

Q. Wolff-Parkinson-White syndrome. Just a couple of things on that that I forgot to ask Dr Sheppard. I think it is a relatively common -- I will not say "affliction", but a disorder or an irregularity. There is debate about it but somewhere between 2 in 1,000 and as many as 6 in 1,000?

A. Yes, it is very, very common.

Q. Quite common, compared with the other conditions we have been talking about.

I think the important statistic perhaps from the coroner's point of view is that of those with that disorder, it very, very seldom leads to a fatality, all sorts of other things happen but not death?

A. It usually leads to palpitations and syncope, it rarely leads to death in the people that you know about.

Q. Yes.

A. The problem is, it is like many things, when people die suddenly without warning, you cannot say they didn't have it, if you see what I mean, because it can cause sudden death and people are sometimes resuscitated and then found to have WPW, with an accessory pathway. It is impossible to exclude it I guess with absolute certainty, but it is not that common.

Q. Not that common. I would suggest that death from WPW is rare.

A. The difficulty I have is that we are talking about, if we deal with a group of people that we accept had sudden arrhythmic death, in other words the population that we are talking about where there is no evidence of poisoning, so in the general population. We know that 70 per cent you do not find evidence of a genetic abnormality on molecular autopsy.

I do not know if any of those have died because of Wolff-Parkinson-White syndrome that was previously undiagnosed.

Q. I understand.

A. Some of them could have. They have certainly died of something that is undiagnosed, so I can't say they haven't.

Q. Very well. Let's see if we can get on to firmer territory.

Long QT syndrome, as the name implies, it is the long QT interval on the ECG, is detectable on an ECG?

A. Yes.

Q. In fact that is what it means, that you have this irregularity on the ECG?

A. It is not an irregularity, it is --

Q. It is a long interval?

A. I mean the QT interval is longer than it normally is, except that a quarter of the people who have the gene

for it have a normal QT interval. They have the genetic abnormality but the normal phenotype.

Q. I think you also mentioned that Brugada is detectable on ECG?

A. Yes, but not always, sometimes you have to inject ajmaline to reveal it.

Q. I think the same goes for CPTV, you see it on the ECG?

A. No, you don't.

Q. You don't?

A. No, you don't see any, not unless they are having an attack of ventricular tachycardia.

Q. Ah, yes, that was the caveat.

Certainly as far as the common ones, LQTS and Brugadas, are concerned you would have to postulate that Mr Perepilichnyy had reached the age of 44, with obviously ample access to the best private medicine, without ever having had an ECG?

A. Yes, he didn't have an ECG but I am not quite sure --

THE CORONER: Are you not saying it wouldn't necessarily be picked up on ...

A. Why would he have an ECG if he hadn't had any symptoms? But as we have said, three-quarters of people who die from sudden arrhythmic death syndrome die without any preceding symptoms, so they don't necessarily have an ECG. Some of them who -- you know sometimes we pick

up Wolff-Parkinson-White syndrome when people come in who have no symptoms and they are 60 and they come in and the anaesthetist for a hernia repair he is 60 we will do an ECG, it is an incidental finding and they have never had symptoms.

Q. This is obviously a factual issue for the coroner but your position is that if he doesn't have any symptoms why would he have an ECG, that is in summary it?

A. Yes.

Q. Yes.

I think it is made clear in your original report, and I think I understand what you are saying, that if you have ventricular arrhythmia, down you go?

A. No. I think I said you go down if you have pulseless VT or ventricular fibrillation, but you can have VT, ventricular tachycardia, you have a ventricular arrhythmia that is not so fast that you lose consciousness.

Q. Sorry I said arrhythmia, I should have said fibrillation, I think if the oedema was collected prior to resuscitation if, then you are postulating, I think a double, two events following, first atrial fibrillation developing into ventricular fibrillation?

A. That would only apply in Wolff-Parkinson-White syndrome. It is well recognised, the problem with the

Wolff-Parkinson-White syndrome is there is an accessory conducting system or systems, sometimes there are multiple, from the atria to the ventricles. The normal conduction is through the AV node and the AV node will not conduct rapidly in most people.

If you exercise someone, it is virtually impossible to get their heart rate above 200 by exercising, but if they go into ventricular -- and at 200, you know, you feel pretty unwell, but if you go into atrial fibrillation, you effectively have a disorganised atrial rhythm which is about 600 per minute. That will not conduct through the AV node. It will often conduct at 150 and you will feel awful.

Q. I think we may be getting a little bit technical. Can we simplify it in this way, what I am suggesting, if the coroner comes to the conclusion that there was a period of distress of, I don't know, half a minute or something, leading to a fairly sudden collapse with no defensive injuries. That is not consistent with the arrival of ventricular fibrillation, it must have been preceded by something that would have allowed him to run uphill?

A. That's correct, it would have been preceded by -- if you are saying was it preceded by another arrhythmia such as ventricular -- it could be ventricular tachycardia, it

could be atrial fibrillation, which then gets conducted to the ventricles for an accessory pathway. But ventricular fibrillation is not consistent with doing more than staggering two or three yards and then collapsing.

MR MOXON BROWNE: Exactly, thank you.

Thank you very much.

MR SKELTON: Sorry to interrupt, I wonder if we ought to have a short break for the stenographer.

THE CORONER: Yes, certainly.

(4.06 pm)

(A short adjournment)

(4.20 pm)

MR SKELTON: Sir, some interposed housekeeping if I may.

Dr Perry, who has kindly say through today's evidence, was due to give evidence but given the hour we have released her and she will return tomorrow morning.

THE CORONER: Thank you very much.

Yes.

MR STRAW: Thank you.

Questions from MR STRAW

MR STRAW: Dr Wilmshurst, was there positive evidence in the medical records, witness evidence or pathological finding either supporting or refuting the cardiac cause of death?

A. No. Neither confirming nor refuting.
Q. Also no positive evidence supporting or refuting the channelopathy as the cause of death?
A. That is correct, yes.
Q. A couple of questions about the relevance of Mr Perepilichnyy's history, if I may. Had you seen the insurance evidence in the bundle that was given to you?
A. I have seen some insurance evidence that was given to me; I don't know if I have seen it all.
Q. If it helps, have you seen some fairly detailed questionnaires where he is asked a number of questions about his prior medical history?
A. I have seen some questionnaires.
Q. Also some examinations by medical practitioners, taking his pulse and things like that?
A. Yes.
Q. Which show that his pulse is low and his blood pressure is ordinary; is that fair?
A. I remember his blood pressure was normal, I can't remember what the pulse was.
Q. Just a couple of pages then from the bundle if I may, can you have a look at bundle 1, the expert bundle 1.
A. I have 2 and 3.
Q. Tab 15 within that, please.
A. Tab 15.

Q. 15?
A. Yes.
Q. It is the top right-hand corner we are looking at, page 63.
A. Yes.
Q. Does that appear to be a note from a medical practitioner?
A. Yes. I don't know if I have ever seen this actually but yes, yes.
Q. Top right, it has "115 over 76", presumably the blood pressure?
A. Yes, that's correct.
Q. Then "P: 53", would that appear to be pulse 53?
A. I think it probably is, although if you said to me would I swear it wasn't 63, I wouldn't --
Q. Something around that sort of --
A. Yes.
Q. Okay.
I am only going to go to two other entries in the records but could you go please then to tab 16, page 81. Do you have that?
A. Yes.
Q. Do you see section 4:
"Have you had any of the following illnesses or symptoms, (a) shortness of breath, ankle swelling,

palpitations, heart murmurs, high blood pressure, heart attack/rheumatic fever."
He ticks "no", do you see that there?
A. Yes, correct.
Q. A bit further down, (d), it includes, and I emphasise this, fits or faints, epilepsy and so on. Is it correct he also ticks "no" to that?
A. Yes.
Q. The last page, please, tab 19, page 99, another questionnaire. Do you see in box 10, he is asked:
"Do you have or have you ever had chest pain, palpitations, irregular heartbeat ..."
A. Sorry, page which?
THE CORONER: 99, top right.
A. 99, sorry, yes.
Q. Item 10:
"Do you have or have you ever had chest pain, palpitations, irregular heartbeat ..."
He ticks "no" to that?
A. Yes.
Q. Finally, box 11:
"Have you ever had any blackout, numbness, dizziness ..."
He also ticks "no" to that?
A. Yes.

Q. Can you help us, what is the relevance of that history or the significance of that history in Mr Perepilichnyy's case?
A. He has nothing in the history to suggest prior heart disease.
Q. To what extent does that help us as to the likelihood of him having suffered a sudden adult or arrhythmic death in this case?
A. It doesn't, because three-quarters of people who suffer sudden arrhythmic death have no prior cardiac history.
Q. The prior history of those sort of symptoms doesn't really point to the cause of death or not?
A. No, if they had been present, that might persuade you a bit, but they are very non-specific anyway, for example a lot of, you know -- you have to explain to people what you mean by breathlessness sometimes, I usually say "undue breathlessness" because we all get breathless sometimes when we run for a bus.
Palpitations, you have to say -- I mean you get a fright and you get palpitations or you have an interview for a job and you have your heart beating fast.
I was just going to say, even if you have those symptoms, it doesn't necessarily help you anyway.
Q. In Tatiana Perepilichnaya's most recent witness

statement, she says this:
"Alexander's mum mentions to Mrs Perepilichnaya that when Alexander was a child he had two to three episodes when he collapsed and lost consciousness for a few seconds. They checked him over afterwards but nothing was found."
A child collapsing and losing consciousness for a few seconds, would it be right that that could have any number of causes?
A. I am not a paediatrician but from my general medical knowledge, that is correct.
Q. Doing the best you can, I appreciate you are not a paediatrician but seeing large amounts of blood, hypoglycemic episode, hyperventilation, there could be any number of non-cardiac causes of a couple of seconds' collapse. Would that be fair?
A. Is that what she said, a couple of seconds' collapse?
Q. Yes, she said:
"... when he was a child he had two to three episodes when he collapsed and lost consciousness for a few seconds."
A. It is difficult to know, speaking as a non-paediatrician I know that children collapse sometimes when they are very young with breath holding, children faint, although that is more common in the teens and it is more common

in girls than boys. Yes, it is very difficult to draw any definite conclusions, but it may or may not be relevant. I don't know.
Q. Presumably that evidence with then from at least Mr Perepilichnyy's evidence on the insurance applications, no further faints or blackouts that he was declaring for the rest of his life. Would this have any significance in pointing to a particular cause of death?
A. No.
I think it is very difficult to say, to be honest. It depends when we are talking about in childhood, he may not have even known, if he was two years old and you had a couple of blackouts from breath holding, when you are 40 would you even remember that?
I mean I don't know, I suspect not.
Q. Thank you. Your evidence on pulmonary oedema, would this be a fair summary of it. It doesn't take us anywhere, it doesn't tell us whether one cause or other of death was more likely?
A. Yes, that's right. Yes. When you say one cause or other, you mean did the heart cease because he had a sudden arrhythmic death or due to a channelopathy or did he have a sudden arrhythmic -- the heart stopped because of a poison. No, of course not.
Q. Last area of questioning, please, is your conclusions.

Can I take you to your report, please which is in hopefully expert bundle 3, tab 96. Then in the internal pagination 14, looking at question 50, you note there you are asked:
"Is it agreed by the expert that sudden unexplained death cannot properly be attributed to SADS unless other possible explanations for the death have been totally excluded?"
You answer yes to that.
A. Yes. I would say excluded to the satisfaction of the court, I guess is what I would actually say.
Q. For example in the specific issue we have here, poisoning, the possibility of a poison would need to be excluded to the satisfaction of the court with the benefit of toxicological evidence before SADS is an appropriate conclusion?
A. Yes.
Q. I think you fairly say you are not a toxicologist and so whether the poisoning can be fairly excluded is for someone else, for the toxicologists?
A. Yes, that's correct.
Q. Is it right at question 54, you essentially say you are not in a position to give an opinion about the likely cause of death when the possibility of undetected poisons is raised because it is outside your expertise?

Is that essentially your evidence?
A. Yes, well, yes. I mean my position is that I am here to advise the court, I don't hear all the evidence, I can just give you an opinion based on what I have heard and that is right. Yes.
So it has to be excluded to the satisfaction of the court.
MR STRAW: Thank you.
MS BARTON: No questions, thank you, sir.
Questions from MR BEGGS
MR BEGGS: Just three brief matters.
In the same spirit of inquiry that Mr Straw behind me put some child episodes to you from the deceased, we also know that post his death testing was done on members of his family. It was discovered that his sister and his daughter have mitral valve prolapse. Does that in any way change your opinion on anything?
A. Mitral valve leaflet prolapse is quite common. It rarely causes sudden death, but would always be found at post mortem. I have to accept Professor Sheppard's evidence that she excluded it.
Q. Is the answer to my question that fact is not going to assist this coroner?
A. I think it is not going to assist.
Q. Thank you.

Could we go, please, in the bundle to your original August 2015 report, I don't know what the tab number is but the page number is 429, I am afraid I can't tell you if that is the first or second.

A. Second.

Q. Second, thank you very much.

Tab 55 I'm told, 429.

A. Thank you.

Yes.

Q. Do you see paragraph 14, just about halfway down that page, please?

A. Yes.

Q. I am just checking a few aspects of your original report and whether you adhere to the propositions therein.

Do you see about halfway down paragraph 14 -- you are talking here about long QT syndrome -- you say:

"Exercise and sudden shocks are reported to cause ventricular tachycardia or ventricular fibrillation ..."

You give the percentages and the chromosome references. There you appear to be saying that exercise is one precipitator of this particular syndrome?

A. It is with long QT type 1. There was a number of different genes, in fact there are more than three genes, so --

Q. Yes.

A. With long QT type 1 you can get ventricular tachycardia and ventricular fibrillation on exercise.

Q. Thank you.

Then at paragraph 15 you make a similar reference in relation to the other group of channelopathies, CPVT.

A. Yes.

Q. You say that is typically triggered by exertion and stress?

A. Correct.

Q. Thank you. In that same lengthy paragraph, if you look at page 430, almost exactly halfway down the page, you speak of a June 2015 annual meeting of the British Cardiac Society and you report that a:

"Dr Till reported at that meeting that CPVT is increasing [I think it is meant to say 'increasingly' isn't it, rather than 'increasing'] recognised in people older than 40 years of age who have arrhythmias triggered by exercise, and in the majority of those cases no mutation is identified which leads to the conclusion that an unknown gene or genes are responsible."

You adhere to that proposition?

A. In fact more so, because I saw Dr Till last week at the British Cardiac Society and spoke to her and also I spoke to other cardiologists and they all say, you know, Andrew Grace in Cambridge I spoke to him and they are all saying that CPVT, they are increasingly recognising it as a cause of ventricular arrhythmias in older people, but the gene is not recognised but they have the phenotype. By that I mean they have the physical features, that is to say if you do an ECG when they have ventricular tachycardia it goes in more than one direction, it is polymorphic.

Q. That particular proposition, which I read completely, you not only adhere to, you think that if anything the evidence is starting to intensify in its favour?

A. Yes, and in fact I think in our joint report Dr Sheppard says that she is starting to think that CPVT is much more common than we previously thought.

Q. Thank you --

THE CORONER: Your general point is the sentence at the start of paragraph 14, is that right, that you say there are some channelopathies more likely to cause fatal arrhythmias when the person is resting or relaxed and others are more likely to cause death during exercise or if the person has a sudden shock?

A. Yes.

THE CORONER: That is the general point?

A. Yes.

MR BEGGS: Thank you.

Turning over the page to 432, please, paragraph 21, I just want to pick it up about seven or eight lines down, perhaps nine, where you say:

"Patients with this condition, known as Wolff-Parkinson-White syndrome, can develop fatal ventricular fibrillation if their heart rate is very fast, such as occurs if they develop atrial fibrillation which can be triggered by exercise and stress."

A. Yes, atrial fibrillation can be triggered by exercise and stress.

Q. Thank you. When you signed off that report, admittedly before the further exhaustive lines of inquiry with toxicologists and so forth had been pursued. Nonetheless, as a matter of historical record, at that stage your instinct was that the deceased suffered arrhythmia as a result of a cardiac ion channelopathy. That was your instinct at the time?

A. Yes.

MR BEGGS: Thank you very much.

THE CORONER: Nothing else.

MR SKELTON: Sir, I think that concludes today's questions.

THE CORONER: Thank you indeed, thank you.

A. Thank you.

THE CORONER: All right.

Thank you all very much.

10.00 tomorrow.
MR SKELTON: Yes, sir.
(4.40 pm)
(The Inquest adjourned until 10.00 am the following day)

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