SCOTTISH HOSPITALS INQUIRY

Hearings Commencing
19 August 2025

Day 4
22 August 2025
Kathleen Harvey-Wood
Dr Samir Agrawal

CONTENTS

9:34

THE CHAIR: Good morning. I think we're ready to resume with Ms Harvey-Wood.

MR CONNAL: That's correct, my Lord.

THE CHAIR: All right. Good morning, Ms Harvey-Wood.

THE WITNESS: Good morning.

THE CHAIR: Now, you have given evidence previously in the Inquiry, I think on 18 September of last year.

THE WITNESS: That's correct, yes.

THE CHAIR: So you'll be broadly familiar about the way we proceed. I think, to begin with, you're prepared to affirm.

THE WITNESS: That's correct, yes.

Ms Kathleen Harvey-Wood, Affirmed

THE CHAIR: Thank you, Ms Harvey-Wood. Now, we're scheduling you for the next hour and a half, broadly speaking, but if at any time you want to take a break, just give me an indication and we'll take a break. Now, Mr Connal.

MR CONNAL: Thank you, my Lord.

Questioned by Mr Connal

Q Good morning and thank you for returning.

A Good morning.

Q You produced for the Inquiry what's been described as a consequential witness statement. Can I just ask you the formal question: are you content to adopt that as part of your evidence?

A Yes, I am.

Q Thank you. Now, I think I've been advised that you've got in front of you a copy of your own witness statement on which you've made a few notes of one kind or another. Now, I certainly have no difficulty with that, and I suspect his Lordship will have no difficulty with that either.

A Thank you.

Q So feel free to consult it as we go. Now, it's possible you may feel that some of the questions that you'll be asked are returning to questions you have been asked before----

A Okay.

Q -- but that's, I'm afraid, a consequence of the way matters have developed over the period of time. So, your consequential witness statement obviously sets out a fairly long

association with Yorkhill. Is that correct?

A Yes, it is.

Q About something over 30 years there?

A Yes, I was at Yorkhill for 32 years, and then when Yorkhill laboratory moved to the QEUH, 2012, and I was there for-- from 2012 to 2023. So most of my career was at Yorkhill Hospital.

Q So you were still dealing with Yorkhill prior to the fact that the Schiehallion Unit moved in----

A Yes.

Q -- to the new hospital?

THE CHAIR: Should I take from that-- I'm sorry if I'm being slow about that. You were physically at Yorkhill Hospital until 2012.

A That's correct.

THE CHAIR: 2012, the labs moved and you moved with the lab to what I think now is the Queen Elizabeth campus. Is that right?

A That's correct, yes.

THE CHAIR: Right, and you remained in post between 2012 and 2023. Is that right?

A Yes, at the QEUH site in the new laboratory building.

THE CHAIR: Right.

A In the three years between 2012 and 2015, till the hospital moved to the campus site-- in that three years, the laboratory was no longer on site at Yorkhill, but we went back daily to Yorkhill Hospital to attend ward rounds and give advice. We also had a hot lab at Yorkhill which did urgent samples. There was a biomedical scientist there as well doing urgent samples on the request of the clinicians to prevent delays of samples being transferred across, especially through the Clyde Tunnel, from Yorkhill to the campus at QEUH. So there was a hot lab for three years as well.

We were given mobile phones which enabled us to be in contact with Yorkhill staff at any point in time, because not being on site we felt was a bit of a change in the service provision, because if you're on site in a laboratory, you can just walk across and go into the ward. If you're off site, you know, the-- the geographical distance isn't far, but if you're in your car and you're travelling, it can take a-- a bit of a journey. So we always went-- At one point in the day, the microbiology staff who were transferred from Yorkhill would go back to Yorkhill at different points in the day. Sometimes in the morning we would go there first thing and then travel on the QH site. That was for the

three years.

THE CHAIR: So despite the move in 2012, you're continuing to provide support to, among other services, the paediatric haemato-oncology service----

A Yes.

THE CHAIR: -- which is still located in the Yorkhill Hospital site----

A That's correct.

THE CHAIR: -- until 2015?

A Yes.

THE CHAIR: Thank you. As I say----

A So they-- they'd moved the lab before they moved the hospital, because the lab building was ready in 2012.

THE CHAIR: All right. Thank you.

MR CONNAL: Am I right in thinking, in fact, that your focus was entirely on paediatrics?

A Yes, it was. I have-- I haven't done any microbiology in-- in the adult speciality. My training was specifically for paediatrics, which I retrained even after the move to the-- the new site at QEUH.

Q Thank you. We find a lot of that set out very helpfully in your consequential witness statement. I mean, I might just call it a witness statement, make it less of a mouthful. Can I just ask you about one thing that you mentioned, just to make sure that we're understanding what you are saying? In paragraph 1.12, which electronically is on page 20 of your witness statements, page 3 of the hard copy, you talk about something called a "spring bloom." Now, you say that it can lead to an increased risk of infections. Now, first of all, what's a spring bloom?

A It-- it was an increase in the bloodstream infections that we would see in the early months of the spring as the-- the weather got warmer, and it was just like a-- an annotation, like a-- We just-- It was like a phrase we used in microbiology, and there is literature which-- I've referenced one, to say that it does happen, you know, in the environment when the-- the weather becomes a bit warmer, the temperature changes, that the bacteria start to grow.

So sometimes we would expect small peaks in the spring months, but they were never of any concern and I don't have any recollection of having outbreaks of environmental organisms or anything during these kind of what we called spring blooms. It was one of my colleagues that used to use the term, and so it was just a kind of-- It's difficult to explain, just-- just a

terminology we would use in microbiology, just, “Oh, there’s a wee bit of an increase in blood cultures.” It was never of any clinical concern.

Q Well, that’s what I just wanted to check with you, because in paragraph 1.12 you say that it’s something that can lead to increase of infection, but in 1.13 you said it wasn’t investigated as it didn’t have a detrimental effect on patients at Yorkhill.

A Mm-hmm.

Q Is that----

A That’s correct, yes.

Q Now, let me move on a little bit and use your time at Yorkhill to advantage. I think you’ve been asked to consider the suggestion that there were considerably higher numbers of cases of environmental concern in Yorkhill compared to the new hospital, and you’ve expressed a view on that, I think, in paragraph 2.1 of your witness statement. When you say you don’t accept that, is that your position?

A Yes, that’s my position.

Q That’s based on presumably some considerable contact with what I might describe as what was going on in Yorkhill over the years. Is that correct?

A Yes.

Q Can I ask you to look at another document? Because things are always moving on. As soon as we think we’ve pinned them down, they move again. Can I ask you to look at bundle 44, volume 5, at page 50?

Now, this is a more recent production than some of the other comments, and I’m not going to ask you to discuss the epidemiology in any great detail, but you see the graph and you see there’s the red line and there’s a pink wider line, if I can call it that in a very non-technical way. Now, if that was to be understood as indicating that there was a trend which was perhaps slightly down perhaps somewhere around 2015, and then rose to a peak somewhere around 2018, before dropping off after that point, would you recognise that pattern as something that matched or did not match your experience?

A I think that would match our theory and, also, with referring back to the graphs I presented at my last oral evidence, you can see that there is an increase with the smooth linear line over 2000 and starting 2017, end of 2016, up until 2018/19. Of interest for me, actually, is the negative or zero points in the black dots, which are the incidence rates. You can see that prior to the move there was quite a few low or zeros,

and then after the move, in the first year post-move, again, a larger number of points at zero; and then for me the most interesting thing here is as well that, after all the interventions and mitigations, in 2020, you can see zeros appearing again and more appearing so they're closer together. In 2022, after the refurbishment of the Schiehallion Unit and the patients being back into the unit, the trend is downward in Yorkhill. There isn't that increase that you see with the GAM fit on the smooth line during the period of Yorkhill analysis and data.

THE CHAIR: Sorry, it's just for my noting, Ms Harvey-Wood, you said what was interesting was "after the mitigation and interventions." Now, what are you taking as the period of after the interventions and mitigations?

A I would say you're looking at sort of where you see the line falling down, but when it falls down and then kind of becomes more straight. So I would say 2020. If you look at the graph at 2020, it's when the lines cross as well. If you see that's about the point where the blue and the pink line sort of cross in 2020-- Do you see that, where they sort of join together again?

THE CHAIR: Yes.

A I would say around about then, and also this is the same time as when you see the negative where you've got zero incidence rates.

THE CHAIR: Yes. Now, having established the point that we're talking about, you drew attention to the number of zero points, which I take to be represented by the dots coinciding with the x-axis of the graph.

A That's correct, yes.

THE CHAIR: Right. Thank you.

MR CONNAL: Thank you, my Lord. You mentioned the materials you produced previously. I'm going to put these back up again just in a little while, but if you can bear with me for the moment while I ask you one or two other things just in passing. I'm not going to ask you about everything that's in your witness statement, purely for practical reasons. Can we look at 225 of your witness statement, page 25 of the electronic version 8 of the hard copy? Just at the top of the page there.

Now, you seem to be taking a view about the use of "normal" to describe a particular bug, which I'll, as usual, not pronounce correctly, Acinetobacter. You see what you're saying there?

A Yes, I have.

Q And what's your point, so we understand it?

A Well, what I was doing was responding to the statement in the HAD Report, page 96, where they said that, "...however this pattern was the norm at Yorkhill, suggesting this may be usual presentation in this population," which I thought the comment really wasn't appropriate because they are comparing it with sporadic cases at QEUH. Acinetobacter should never be normal, so I don't know why they use the word "norm," the abbreviation as well, rather than normal. Obviously from their data they have shown there was cases of Acinetobacter at Yorkhill, but it would never be considered a normal infection.

Q And can I just ask you about that? If there was an infection of that kind in Yorkhill, was it dealt with?

A There was occasions when we had an increase in infection, and if it was thought to be of clinical concern and the clinicians had raised it as well as Infection Control, then we would investigate it, and I have put an example in my statement of a Pseudomonas outbreak in the paediatric ITU. So there was instances where we would investigate it, and as I said in my statement, a very close relationship with the infection control nurse at Yorkhill, who used to come to the lab every day and we would discuss any concerns. So it's probably difficult to prove this with documentation historically, but if we were aware of a "problem" or a concern, then it would have been raised with Infection Control at the time. And in the context of this, the National Infectious Control Manual wasn't available for part of the time at Yorkhill. I think it was 2012 it was introduced.

THE CHAIR: That's our understanding.

A So if you're looking back before 2012, the statuary infection control guidance wasn't in place.

MR CONNAL: I don't know whether you can help us with this or not, but one thing you might take from the statement in the HAD Report was that this population, i.e. this cohort of paediatric haemato-oncology patients, had a kind of norm of this particular bug. Do you know whether that was found in the new hospital?

A Acinetobacter cases?

Q Yes.

A We did see it, yes, but I would say of all the environmental organisms it probably wasn't the predominant one, but it was seen, and they'll have the information in the HAD table as well. But, yeah, we did see it,

but it wasn't one of our concerning organisms, and as you'll see in my statement it was more of a concern in the paediatric ITU, actually, rather than the Schiehallion patient group.

Q Thank you. I've been asked to ask you some questions about paragraph 230 of your witness statement and what follows from that. In paragraph 230, you explain that you were in touch with Dr Bagrade because you wanted to bring to her attention what you describe as the first case of colonisation with Stenotrophomonas maltophilia, isolated from a sample which was, in what you say here, not part of the norm, and you were told, "Not interested, don't look for that." The way you've expressed that, were you expecting a different response from Dr Bagrade?

A Yes, I was expecting some note of interest because we were looking at the microbiology for the new unit, if you call it that, the refurbished unit. We were very interested in what would happen when they opened the new unit, when the patients were moved back in. We were concerned, would colonisation happen again? Was the environment safe? So if anything at all from our microbiology results was isolated that

we thought Infection Control should be aware of, then we were emailing them and looking even more closely at the monthly blood culture statistics as well to look at, was there any increase in infections post-move back to the refurbished unit?

Q But you were told not to look for it?

A Yes, we were.

Q Would that be what you were expected to be told?

A No. I would expect that they would want to know about Stenotrophomonas maltophilia, because it's one of the key organisms, and I think I said that before. It's one of our key organisms that we use-- Remember, like a trigger point. It's one of the organisms that when you see it, it kind of says, "Oh"-- It's like an alarm bell, "Oh, this shouldn't be here," sort of thing. So, any environment of concern, like Stenotrophomonas, is something we would want to bring to attention of Infection Control and also inform my line manager at the time as well, who was involved with the email too.

Q If you're asked not to look for it, does that impact on the reporting of this organism?

A Well, that's exactly correct, because when you examine a

microbiology specimen you're looking for the bacteria in the sample that are infectious or infections, rather-- You have normal bacteria in any clinical sample, but you also have the bacteria that cause infections, and we were screening faeces samples from the haemato-oncology patients to look for colonisation with potential environmental organisms that could cause infection, and this wasn't a new specimen type. We did this process of screening, I think it is a better way-- We screened certain samples of haemato-oncology patients at Yorkhill as well to look for colonisation, to have an alert, an awareness, of anything that might be concerning.

So we used to do oral samples, stool samples. Each patient had a screen to monitor their potential infections, so the faeces sample was part of that. We weren't looking for faecal pathogens. It was a separate code in the laboratory. So we weren't looking for pathogens that cause gastroenteritis; we were looking for organisms in the faeces that would be a potential source of infection.

Q I mean, you can't speculate beyond what you were told, but were you told why you were not to look for it?

A No.

Q Now, you go on in paragraph 2.31 to quote part of the HAD Report, and then you offer a comment on Stenotrophomonas maltophilia in paragraph 2.32. Just so we're clear, what's the point you're making in 2.32?

A Well, 2.32 just sort of falls on what I just said. It's recognised as an important pathogen and it can have a high mortality rate if it's found in the bloodstream in a bacteraemia, so it requires close liaison with Infection Control. So this, you know, is why I gave the example of the screening sample with Stenotrophomonas, because it could have referred back to a comment made in the HAD Report that said that it rarely causes infection in immunocompromised patients.

Q You've made various comments on the statistics and the comments on the statistics that were put to you for the purpose of this statement, and you accept that there were environmental organisms found in Yorkhill. At the foot of the page we're looking at at the moment in your statement, you say:

> "Yorkhill environmental BSI infections did not have the same consequences or concerns at the

time regarding patient morbidity or safety."

You're putting quite a lot into one sentence there. What's the message you're trying to get over?

A I think the message is any hospital-- If you look back at one of the other reports by Mr Mookerjee, he compared different hospitals, paediatric hospitals, like Cardiff and Leeds and Great Ormond Street. They also had environmental infections in the bloodstream. So all hospitals do have environmental infections, but the difference here in the QEUH was the diversity and the complexity of the infections. We did see infections from time to time in the environment at Yorkhill, but clinically they didn't have the same concern. And I think, to refer back, if that's okay, to Professor Gibson's witness statement, she also had the same impression, if that's the word to use. She also, as the lead of the haemato-oncology unit, was not concerned that the infections were as serious, whether that's the word to use, or in the numbers close in time. So what we were seeing at QEUH was a lot of infections at the same time with different organisms, whereas at Yorkhill it was more sporadic. We would see infection from time to time, but they weren't clustered into a shorter period of time and lots of them, if you see what I mean. The timelines were very different.

Q Okay, thank you. Now, earlier you drew his Lordship's attention to the materials that we looked at briefly. Now, I say briefly on your previous appearance because we did look at them, but we didn't look at all of them and we didn't take a great deal of time on them. So I'd just like to touch on them again as we have you here. That's bundle 19, page 143. Now, I'm going to look for a moment at some charts, but what we probably didn't do on the last occasion was note specifically the document that this is part of, which you were part of preparing with Dr Peters. First of all, who was it presented to?

A This was presented at the haemato-oncology unit, would have that information.

Q And that's, what, the clinicians?

A Yes.

Q Just taking it generally, first of all, do you remember what reaction you got when you went through that material for the clinicians in this presentation?

A They----

THE CHAIR: Sorry, just so that

I'm keeping up. When you say this presentation was made to the haemato-oncology unit, is that the paediatric----

A Yes, sorry. Paediatrics, yes.

THE CHAIR: So the Schiehallion Unit?

A Yes, uh-huh.

THE CHAIR: I beg your pardon. Mr Connal?

MR CONNAL: Sorry, I'm just trying to take from you very generally. This is obviously-- As we will see in the moment, it's a presentation with lots of graphs and so on and so forth. It's topped with a narrative and it's tailed with a series of conclusions. Were you getting shock, horror, agreement? Can you remember?

A I would say agreement. I think they weren't surprised when the data was put into this format. I think it was what everyone was seeing, so I think it confirmed everybody's concerns that there was problems with infections and that we were being told by management that there was no infection concerns. And the CLABSI group also produced graphs and data, and that also was very similar to the presentation here or report, and in the actual PowerPoint presentation, I think there's a graph from the CLABSI work as well. So they also-- The CLABSI team, which I was part of the CLABSI team as well, their data and this data here were supporting each other. They were showing the same thing.

Q Just for completeness, because we probably didn't take it last time, particularly because we weren't focusing on this, I see, although this is produced by Dr Peters and yourself, you also have another note there saying, "Pharmacy data provided by Ysobel Gourlay, pharmacist." Now, does that cover things like antibiotics?

A Yes, that was for the daily dose, the DDD. So to look at the different antibiotics in the graphs and the usage of the different antibiotics, we needed to access pharmacy information and their databases to look at the amount of antibiotic was used in the Schiehallion Unit for several different antibiotics over the time period of this report.

Q Thank you. Now, can we just----

THE CHAIR: I mean, just to repeat back that answer. So that means, in October of 2018, there was data held by the pharmacy----

A Mm-hmm.

THE CHAIR: -- which was specific in relation to the particular antibiotics being administered in the

Schiehallion Unit?

A They'll have data on all antibiotics, all drugs administered. I mean, obviously, antibiotic is one group of drugs. There'll be all the chemotherapy drugs, additional drugs that-- you know, pain, whatever, pain relief. So the pharmacy departments there's-- within Schiehallion, they have their own pharmacist who are trained in haematology-oncology specifically. Ysobel Gourlay was one of the lead pharmacists for the hospital. So she would have got the information on the data on the antibiotic usage from the database from the Children's Hospital paediatric pharmacists. So those pharmacists are trained in paediatrics as well as adults, a bit like microbiology. They're specialised in paediatric pharmacy and also in haematology-oncology pharma, they're very specialised in that area too.

MR CONNAL: Yes.

THE CHAIR: Thank you.

MR CONNAL: I think the point his Lordship is trying to ask you is, was it possible at the time to extract information which focused on antibiotic use in the Schiehallion Unit?

A Yes, yes.

Q And is that what you----

A Yes, indeed.

Q -- as one of the participants, in this arranged to be done?

A Yes.

Q Thank you. Now, I'm not going to ask you about all of this because time wouldn't allow. What we'll do is we'll just move through it and I'll pause on one or two of the graphs. So we just go on to the next page, please. Now, that simply shows overall numbers of blood cultures, in case there was any question about that. 145, number of patients with blood cultures. 146, that's increasing rates of positivity. That may show some things, but we'll probably see it more clearly in some of the later graphs. 147, what's being shown there?

A So, for some reason, the green bar chart on the graph, I wonder what's happened to it. Oh, sorry. Sorry, the green's a line. Sorry, I can see it now. Sorry. The green's a line. Sorry, I thought it was another-- like another graph we had before. Sorry. So it's just showing-- I just-- the number of patients that had blood culture taken and the number of patients with positive blood cultures. So it's just showing that there was a downward trend in the number of positive blood cultures that patients had in the period of 2015 to '16, but

the number of blood cultures taken remained the same.

Q Thank you. 148, I want to come back to the polymicrobial question with you a little later on. 149, that's a much more complicated graph because that contains details of a whole range of individual organisms. Is that correct?

A Yes.

Q What we have there is a graph which has a list of organisms down the right-hand side of the graph, which are represented by colours on the graph. Is that correct?

A Mm-hmm.

Q And again, we see totals, but we also see a breakdown in this case of the----

A It's breaking down the different types of microorganisms into groups.

Q Thank you. Then 150, ratios we're looking at there. Again, there are quite a few graphs which show similar things.

A Similar things, yeah. That's just presented as a linear graph as opposed to bar charts, yes.

Q All right, thank you. 151, that's just a note, 152, that's gram-positives, so we've moved away from gram-negatives for the moment, and again, a list of the individual organisms appearing, or abbreviations for them appearing down the right-hand side.

A Yes.

Q 153, that's just a note again, 154, and let's just pause here because we're talking about gram negatives, not exclusively, but quite a lot in this Inquiry. Now, just very briefly, what is this showing about what's happening to numbers?

A Well, it's showing two things. It's showing the increase in number, total number of the gram-negative microorganisms. It's showing that, in 2015 to 2016, there was no environmental organisms. HI is Haemophilus influenzae. That's----

THE CHAIR: Sorry, could you just give me that again? It's showing that in 2000----

A 2015 to 2016, the gram-negative organism that was isolated was not an environmental, because if you look at the key at the side, you can see the different colors. And ENT is green and ENT/ENV is purple.

THE CHAIR: So ENT will be enteric?

A Yes.

THE CHAIR: And ENT/environmental is an organism which can have an enteric source or an environmental----

A Yes, maybe like

Enterobacteriaceae, for example, and then the kind of turquoisey-blue line is ENV, is environmental. So I think to look at it you need to look at these two colours together, that's the environmental and the environmental enteric, so that is the sort of turquoisey-blue colour and the purple. So you can see post-move that the amount of gram negatives in that category increases in time to April '18 to June '18, where we've already mentioned that there was a peak in April 2018. So, you have two things in that last bar chart. You have the highest number, which is-- and you also have within that the largest amount of the blue and the purple colour, which indicates that you have more of the environmental and environmental enteric organisms.

If you look back then to just after the move, you can see that there is no blue or purple, and also in October '15 to December '15, January '16 to March '16, again, there is no blue or purple. We first see that colour in the chart in April '16 to June '16, and that is when I remember saying that I began to see the organisms appearing in the blood cultures, and to me that was when things first started to change, and it's shown there in that graph.

THE CHAIR: Sorry, again, my

fault for not just hearing that. When did you say that you saw a change?

A Yes. So in the chart here, if you look at April '16 to June '16, this column, thank you, is the first column post-move that is showing environmental organisms in the blue and in purple, environmental and enteric organisms, and the small amount of green is the enteric-only group.

THE CHAIR: Sorry, Mr Connal.

MR CONNAL: Thank you. Well, I think we have your explanation of that graph. Can we go to 155? Now, what's different about this one is that this is just environmental?

A Yes, so the other previous graph included different-- other different types of gram negatives. So it was titled, "Gram-negative organisms."

Q Yes.

A So what Christine did was we teased out from that just the environmentals, and that's the three groups of organisms that I discussed in that previous graph, that the ones we were concerned about-- the ones we wanted to see in more detail, so we took the other data out so it was more obvious what was happening.

Q Also, there were gram positives here. Is that right? We see

the green.

A Yes, there was some green. That's right. Yes. Our environmental and all our organisms included gram positives, as you'll see later in the presentation, but, yes, this is-- we kept the gram-positive environmentals in. So the previous graph, if you want to go back and look at the title again, just for clarification, was, "Gram-negative organisms," okay? So we then looked at environmental organisms, and from the legend at the side, you can see that we have environmental gram positives as well as the environmental gram negatives. So it's a slightly different presentation, including some gram-positive environmental.

But what you can see from that is there wasn't as many environmental gram-positive organisms as there were gram negative. And I've got a-- quite interest-- In the graph, if you look back at Yorkhill, we didn't have any environmental gram positives, and the chart shows that the numbers, the total numbers, the height of the bar charts is much lower than post-move and quite striking for me, if you look at that graph is the gap where there's no organisms which, again, refers back to what you saw in the HAD Report.

For me, it's always interesting to look at when there's nothing, when there's no organisms, when there's no infections and refer that to when there is. What's happened? What's the change? Why has it gone from no organisms to organisms being isolated? And why has it gone from a lot of organisms and a concern of environmental organisms? And then you see that they have gone away again. So it's a bit of a cause and effect there. So, for me, the three different time periods post-move, you can see there's nothing on the graph. So there wasn't any environmental organisms.

Q So that's when the hospital is, as it were, brand new?

A Yes. Yes. The little red arrow in all the graphs is just an annotation to remind people when the move took place. So it's just a visual representation of, that's when the hospital moved in 2015, and that's why it's June, because it moved 2015 in June.

Q That's migration of patients rather than any contractual handover date, is it?

A Yeah, it was the actual patients' move, yes. The actual move of-- yeah, the actual move of the Schiehallion-- because the patients didn't all move in the one day.

Q No.

A Yorkhill moved from the site at Yorkhill to the QEUH site and the patients went in wards or groups or specialities, then the clinicians would go with them. So they moved in groups of specialities into the ward that they were given in the new hospital. So the move was kind of-- and the Schiehallion was one of the last wards to move.

Q Thank you. Let's just finish this. 156. Let's not delay on that one. 157, I'm not sure what that's intended-- What's that intended to show at 157?

A This is cases of VRE, which is Vancomycin-resistant enterococci.

Q Right.

A This is a gram-positive enteric organism. The concern with that is it's called Vancomycin-resistant because it's resistant to Vancomycin and it's an organism that is quite easily transmitted from faeces. So it's found in the gut, colonising, and the concern is that Vancomycin is one of the antibiotics that's used to treat gram-positive infections in the Schiehallion oncology cohort of patients. So it was just looking at the spikes of cases of VRE along with the blood cultures.

I think it was-- As Christine described, I think it was just looking at other environmental factors and other concerns, as well as the environmental blood cultures. It was just referring to the fact that, for some reason, that number of VRE cases seem to kind of mirror slightly the percentage of positive blood cultures, and I'm----

THE CHAIR: Sorry, positive blood cultures?

A Yeah, so the red line is the percentage of positive blood cultures, and the blue line is the cases of this resistant organism found in the faeces, which can cause outbreaks, and I think Professor Gibson referred to that as well as Rotavirus. But I'm not sure why-- what we're seeing is we saw this phenomena, but we're not sure why the blood cultures would peak at the same time as the cases of VRE. It was just an incidental finding.

MR CONNAL: Well, I just want to ask you about one or two more things in the time we have available. One of the issues that you covered in parts of your presentation was focused on antibiotics. Is that right?

A Yes.

Q Prescribing and so on. Can we just go on-- We'll turn each page, but I want to pause when we get to antibiotics. 158, leave that. 159, yes. This is daily dosage figures. Is

that right?

A This is the information that we had requested from the pharmacy. This was information that Ysobel Gourlay provided from the antibiotic usage within the Schiehallion Unit.

Q Yes. If we go to 160 and 161. Now, tell me what this graph was designed to show.

A It's quite a detailed graph, so you've got your environmental organisms as a bar chart. So superimposed on that is the antibiotics. So you're looking at three antibiotics: ciprofloxacin, meropenem and Tazocin, which are highlighted in different colours as a linear presentation.

Q What do you take from that? Do you take any particular point from it?

A Well, it just shows that, you know, the lines go up and down and up and down. There is, if you want to call, a small peak of Meropenem use in the beginning of 2015, then again in 2018. This may have been due to organisms that were resistant to Tazocin. You can see the Tazocin line is not going up, so it's sort of staying static, up and down. But then as the meropenem goes down in 2015, quarter 4, the Tazocin goes back up again. That's the usage.

So these two antibiotics are used for different reasons. So if Tazocin is a first-line antibiotic, so when the patients first become unwell, they're given Tazocin plus or minus gentamicin. Once we get the results back from the laboratory, if the organism isolated is resistant to Tazocin, then the antibiotic is switched to meropenem. So you can see that the lines cross over each other as the antibiotics are switched or used depending on what organism is isolated. So the antibiotic use is tailored very much to the laboratory port with the antibiotic sensitivities, and they're guided by that on the choice of antibiotic.

The next three lines are dashed lines, and they're showing when the organisms were resistant. So, the dash of the yellow line and the dash blue line are organisms isolated from blood cultures that were resistant to the antibiotic. So if the antibiotic is resistant, then it switched. Is resistant to the antibiotic, then it is switched.

THE CHAIR: Sorry, my fault entirely, I think I failed to follow the point you make on dotted lines. Could you maybe give me that again?

A Yes. The dotted line is organisms that are resistant to the

antibiotic. So the-- the dotted lines are actually organisms, not antibiotic, so the thick line is the antibiotic usage.

THE CHAIR: Sorry, the solid line is----

A Yes, uh-huh.

THE CHAIR: Yes.

A The dash line for meropenem and Tazocin is organisms----

THE CHAIR: Which are----

A Resistant to that antibiotic.

THE CHAIR: -- resistant----

A Yes, and the blue bars are the total number of organisms.

THE CHAIR: Right, and I get the resistance from the letter R.

A Yes, that's correct.

THE CHAIR: Right, okay.

A So when you get resistance, you-- If you refer that back to the solid line, you'll see that the antibiotic usage changes with the resistant organisms being isolated. So if you look at, for example-- So if I give you an example, if you look at 2016-2 and to 2017-1, there is a slight increase in the dotted Tazocin line, so that means we were isolating organisms resistant to Tazocin. So if you go up to the same point, 2016, quarter 4, you can see that the solid Tazocin line fell because we weren't using it to treat infections because we had resistant organisms.

THE CHAIR: Right. So the policy on antibiotic administration follows on the results showing resistance to particular antibiotics.

A Yes.

THE CHAIR: Thank you.

MR CONNAL: When this was displayed to the Schiehallion Unit clinicians, do you remember, did you get any concerns, disagreements?

A No, I think-- I think it was what they were seeing. The concern was the Tazocin resistance, in that it's the first-line antibiotic. So you can see there's sort of two peaks there. 2016, there were----

Q These are organisms----

A Yes.

Q -- which are resistant to what would normally be----

A First line----

Q -- the first-line antibiotic.

A Yes, but with that-- the caveat with that is that they use gentamicin as well. So we use what we call double cover, and that's why we do that, because it gives you a broader cover against bacteria that might-- that are in the blood culture. We don't know yet what the sensitivity pattern of that organism is. So to give cover, quite often they start two

antibiotics we call double cover, and then when you had the results back, you tailor it to the results from the lab. So you may change one, you may stop one, or you may continue. That's where microbiology clinicalisation with the Schiehallion Unit was very important.

Q Gentamicin is not on this graph?

A No, it's not on this graph. No.

Q Thank you. Well, I'm content to move on from that to 162. Now, just in general, why are you particularly looking at meropenem?

A I think this is because there was a question – and I think it's been addressed previously – about meropenem usage driving resistance or driving resistant bacteria, and so there has been audits performed on meropenem usage. It can mean two things. It can mean that the increased usage was due to resistant organisms being isolated that we couldn't treat with Tazocin. Then the question is, do-- by doing that and using another antibiotic, do we select out more organisms that are resistant to meropenem? It's cyclical. That's what happens when you switch antibiotics, but there's-- there's been quite a lot of work around this issue, and from our microbiology perspective, we didn't see a driver for selecting out resistant organisms. The big example here was obviously Stenotrophomonas, which is resistant to meropenem.

Q What that graph shows is not numbers of meropenem doses, it shows a rate of meropenem use per gram-negative----

A Yes, exactly.

Q -- blood culture.

A Yes. So it's related to the-- the number of blood cultures and the organisms that we isolated from the blood cultures, what antibiotic we would use to treat, and this is-- this is just teasing out from the previous graph the meropenem usage against the number of bacteria, the gram-negative bacteria.

Q The comment there is that the rate is stable since the third quarter of 2016.

A Yes.

THE CHAIR: Sorry, just give me that again. The----

MR CONNAL: I'm reading from the top of the page, my Lord:

> "[The] Rate of [Meropenem] use per gram [negative] blood culture [is] stable since [the] third quarter [of] 2016."

THE CHAIR: All right. Can I ask

you, you said that quite a lot of work has been done on this. Is that work that you were involved in?

A No, there was an audit performed by one of the consultant microbiologists on meropenem usage.

THE CHAIR: Sorry, there was an audit?

A Yeah, performed by Dr Alison Balfour, Microbiology Consultant. We also looked in more detail at the use of meropenem in the DDDs, and then there was the questionnaire response which I responded to, and Christine Peters did, that I received----

THE CHAIR: That was the recent----

A Yes, the recent-- Yes. It was part of the Public Inquiry. This has already been addressed, I think, yes.

THE CHAIR: But the hypothesis that meropenem use was driving the results had been raised in 2018. Is that right? The hypothesis? I'm just looking at your graph and assuming----

A Yes, yeah, that's probably correct. That's why we would have looked at it, yes.

THE CHAIR: Yes, right.

A In the timeline of things, that would-- that would have been around the time we had the peak of organisms. I think it would have been looked at then as part of what we called the increased incidence or water-- But they'd have looked at different parameters of concern regarding the increase in gram negatives.

THE CHAIR: Is that chart a reflection of Dr Alison Balfour's work or your and Dr Peters' work?

A This is Dr Peters' work in conjunction with Pharmacy, this particular graph.

MR CONNAL: So the Pharmacy information that's used to determine how much meropenem had been used and so on and so forth, that comes from the pharmacist that you mentioned earlier?

A Correct, yes.

Q Thank you.

A We requested that information from Pharmacy-- or Dr Christine Peters requested that information from Pharmacy.

Q I'll need to move on from your presentation shortly. Just going to 163-- Well, I suspect we can probably leave that one. 164, I see it's similar. 165, another bar chart, and then do we get to the end at 166? What you now do is you basically summarise what you take from the various charts that had been set out

above under various headings: “Epidemiology,” “Antibiotic Use” and “Resistance Patterns.” So, is this material that would go to the clinicians, these summaries?

A Yes.

Q And what was the reaction to these summaries?

A As I’ve said before, they-- they were not surprised at the information. I think it was helpful for the clinicians, because they also-- from a clinical perspective and seeing the patients and the infections in the patients around about 2018 when obviously this data was collected and analysed-- that it supported their concerns, and I think they thought it was helpful, in that we were showing in graphical form what they were seeing in the ward and the patients. Because, remember, the clinicians aren’t seeing all the results coming at the labs. They’re not collating the data. They’re just seeing individual patients and looking after that patient and that patient’s infection, but if you put it all together in a graphical presentation, it-- it shows that there was a pattern and that there was-- there was an incidence of gram negatives, and I think they found it helpful.

Q Thank you. Well, we can leave that document. Thank you very much. I’m just going to ask you one or two more, and then we’ll have a short gap while I check if anyone else has questions. You’ve made various comments. We have obviously all this material whether you deal with it orally or not, and you’ve pointed out that there were lots of interventions started basically in 2018 onwards, and you’ve set out a lot of these in your witness statement, and also your view on whether you were seeing exogenous or endogenous infections, and your view was that there was an increase in exogenous. I’ve been asked to ask you about a different part of your witness statement: paragraph 4.11 which is on electronic page 34, hard copy page 17. I think the question you’re discussing in these sections is the use by the HAD Report of material collected about municipal drinking water. Is that a useful exercise, in your view, in this context?

A I wasn’t sure, as I said in-- in my comment there, of the relevance of it, because the actual purpose of this publication was to look at resistance genes in-- in the water. However----

THE CHAIR: Sorry, just to clarify, “the purpose of this publication.” Are you referring to the Khan paper?

A Yes, is this-- Sorry, are you referring to that, sorry? Am I----

MR CONNAL: I think the-- Yes, the Khan paper that's referred to in turn by HAD.

A Yes, the HAD Report referred to this publication with regard to environmental contamination-- or environment-- in water systems, and as we've heard through the evidence this week, water is not sterile. There is organisms in the water, but they were looking at tap water from Glasgow residences. What we have is patients in a hospital where there is a tank, a big storage tank, of water. It's slightly different. I-- I--

My opinion is that it's different water. It's hospital water. It's not tap water. There's plumbing systems in hospitals, there-- there is stagnant water in hospitals. The children in hospital which we're concerned about and we're discussing here, immunocompromised. They're more vulnerable to infection, their immune system has been compromised or immunosuppressed. In the public water supply, you're giving water to the general public. So, I wasn't convinced that the relevance of this publication referred to an outbreak, because they used it in the context of discussing an outbreak.

THE CHAIR: Have you read the Khan paper?

A Yes, I have.

THE CHAIR: Right. The reference in the report by Dr Agrawal and his colleagues, if I recollect correctly, draws attention to the Khan paper, which I think was maybe published in 2014, but the reference simply says the identified micro-organisms were the most commonly found. Now, what was the Khan paper's purpose?

A Well, that's-- They said in it the aim of the study was to look for resistant genes. What they were looking for was bacteria in the public water supply that had resistant genes. The relevance of that to a hospital and hospital infections was what I was challenging, because I-- I wasn't sure why that reference was used in that context.

THE CHAIR: When you use the expression "resistant genes," is that resistant to antibiotic?

A Correct, yes.

THE CHAIR: Right.

A Resistant genes, antibiotic resistant genes. Bacteria carry on their-- I don't know how to put it. They carry resistant genes. They can----

THE CHAIR: I think it's probably

enough for my purposes to understand----

A Yes, uh-huh.

THE CHAIR: -- that it's about resistance----

A Rather than go into the terminology, yes.

THE CHAIR: -- as opposed to how their resistance----

A And plasmids and all that sort of stuff.

THE CHAIR: -- arises. Right.

A I just wasn't sure-- I just-- I mean, it's a-- the HAD Report is a very big report, but that-- it just-- when I was reading it, it was just something that-- it kind of stood out. I said, "Oh, I'll read this reference. It looks very interesting, 'Environmental bacteria'"-- They used a statement, "Environmental bacteria occur widely in the general environment," and I went, "Oh, that looks interesting." So then I went and-- and looked at the reference and read it, and then I made my comments on it because I couldn't quite connect the relevance of this publication to what they were-- they were referring to it within the-- the section of an outbreak.

THE CHAIR: Mr Connal?

MR CONNAL: I think I possibly only have two more points to take from you, and I hope to be able to take them both fairly briefly. When we were going through your charts – I'm just calling them your charts; I know you were a co-author – we kind of skipped past one about mixed blood cultures. Now, you take the trouble to set out your comments in narrative form, I think in paragraph 5.3, which is at page 35 of your witness statement, and you highlight the fact that you had 11 mixed blood cultures in the first year after the move to 36 in 2016/'17 and 40 in 2017/'18. Now, you say this was of concern. Why was it of concern?

A Because a bloodstream infection or a bacteraemia is normally caused by one organism that colonises the line or gets into and enters the bloodstream. This was infections with more than one. You could call it polymicrobial, which is what-- the heading of the question that you gave me, "Polymicrobial." So that is a concern because there is more than one organism, it's a mixed infection, more difficult to treat because you need to use quite a few antibiotics to treat it because there's more than one organism. This usually means that the line has to be removed, because treatment of a mixed or polycrobial(sic) infection is much more difficult.

Also, it alerts you to the fact that

there's something not quite right. Why are patients getting, you know, an increase? So you're getting-- As you pointed out, you're getting an increase in the number of mixed blood cultures, you know, from 11 up to 40. That's a lot of blood cultures in a year that had more than one organism. It's also a lot of work in the laboratory processing this sample, looking at all of the organisms that you've isolated. So it does bring it to your attention, and it does, for me, suggest there's some source or there's some problem, because most blood cultures are of a single organism.

Q Thank you. You were asked a not untypical question in the questionnaire. Basically, "Have you anything to add, any views on the impact of the environment?" and you've set out a lot of detailed views which I'm not going to get you to repeat orally, and they appear in section 8 of your answers. I just want to ask you one question, and it's just because it jumps out as slightly different from everything else, and that, right down on page 40 of your witness statement, under "Current ongoing issues", at c), you say:

> "Staff have had to use hand gel as the water was not safe due to high levels of Chlorine dioxide."

Am I right in saying you just picked that up in the newspaper?

A No. Two things. I have referenced it as a newspaper, but I do have colleagues that I know this has happened to. They were told not to use the water and to use hand gel because-- I think it was due to the increase of-- The chlorine dioxide levels was unsafe, and for a certain period of time, they were asked not to use the water, and this is quite recently, in the last year.

MR CONNAL: My Lord, I have no further questions for this witness.

THE CHAIR: But you would wish the opportunity to check with the room.

MR CONNAL: Just in case.

THE CHAIR: Yes. As you may recollect, Ms Harvey-Wood, what we do is check with the legal representatives, if there's any further questions. So could I invite you to return to the witness room? It should be no more than 10 minutes.

A Could I ask Lord Bodie, is it okay for me to make a comment about something that I've not been asked about?

THE CHAIR: Well, I think in fact, you may or may not have been invited to do so, but it is all right for you to

make a comment.

A Is it appropriate to do it now or do you want me to do it at the end?

THE CHAIR: You may wish to do it now so that the legal representatives have the opportunity to respond. I should say if I feel you're perhaps going too far away from your central area I might intervene, but if you have a comment, please make it.

A Yes. It was regarding the oral evidence yesterday by Dr Drumright. She was discussing a graph on page 51, which is from bundle 44, volume 5, graph 51.

MR CONNAL: You were shown page 50 by me this morning, which was the environmental. This is the non-environmental.

A Yes. These graphs were part of my bundle of documents to read to prepare for today. But the reason I'd like to raise an issue is that it was stated that the non-environmental organisms, their infection rates may be due to the lab not working and the laboratory contamination of samples. I would dispute that fact, and I feel that this comment was made without any reference to any information or any details of laboratory practices or the working of our laboratory, or had Dr Drumright been in our laboratory to see how we process samples, our SOPs, our guidelines. So I just feel that that comment was a bit unfair.

THE CHAIR: You listened to Dr Drumright yesterday, as did I and everyone else in the room. Now, my recollection is that at that stage in her evidence, she was discussing what-- the expression that was used were counterfactuals, in other words, possible. And my recollection is that she made it quite clear that she was discussing-- My recollection may be wrong, but this is just my recollection, that she was discussing counterfactuals, other possibilities, and I think there was a list of about maybe six of these.

A Yes, there was.

THE CHAIR: So as I understood it, she wasn't saying that this occurred as a matter of fact; she was putting it forward as a possible explanation for the events that were seen, the rates of infection. And again, this is my recollection. She excluded lab infections as unlikely. That's my recollection of her evidence, but I'll bear in mind what you've said, and it's part of your evidence, and as part of reviewing the evidence I will be going back to the transcripts anyway.

So I think I understand the point,

but in fairness to Dr Drumright, my recollection is that she was just putting this forward as a possibility, not so much as a possibility of what might actually have happened but as an analytical method of considering how particular events may have arisen, but thank you for making that point.

A Thank you. Thank you, Lord Brodie, for considering that.

THE CHAIR: Right. So, as I say, we should be able to get back to you in ten minutes.

(Short break)

MR CONNAL: Apologies, my Lord. Practical issues involving changeover, just intervening a little there. But I can announce that there are no further questions for the witness.

THE CHAIR: Right. We'll invite Ms Harvey-Wood to come in, and if there's any housekeeping to be addressed, we can do that afterwards. Ms Harvey-Wood, there are no further questions, which means that you're free to go. But before you do, can I thank you for your attendance today, which is the second time you've given evidence to the Inquiry, but also the preparation that went into not only your previous evidence but your evidence today, and in particular responding to the new material. But thank you very much. You're free to go.

THE WITNESS: Thank you, Lord Brodie.

THE CHAIR: Right, with apologies for breaking into the obviously important discussion. I mean, are there any-- What I would propose is that we take an early coffee break and sit again at quarter past eleven, but is there anything we need to decide before then?

MR CONNAL: Not as far as we're aware, my Lord.

THE CHAIR: Right, okay. Well, we'll have coffee and see each other again at quarter past, where I understand Mr Mackintosh will take over.

(Short break)

THE CHAIR: Now, Mr Mackintosh, are we able to resume with Dr Agrawal?

MR MACKINTOSH: Yes, my Lord.

THE CHAIR: Good morning, Dr Agrawal. Now, as you understand, you're about to be asked questions by Mr Mackintosh. But first of all, I understand you are prepared to affirm.

THE WITNESS: Yes, your Lord.

THE CHAIR: Now, sitting where you are, could I ask you to repeat these words after me?

Dr Samir Agrawal, Affirmed

MR MACKINTOSH: Thank you, Dr Agrawal. The timetabling for today will be that we will plan to sit until one o'clock, take an hour for lunch, resume at two, and proceed through the afternoon. We usually try and finish by four, but your evidence is important and it may be that we go a little beyond four. However, and I wish to make this very clear, if at any stage you want to take a break----

THE WITNESS: Yes.

THE CHAIR: -- for whatever reason, just give me an indication and we will take a break. So please feel that, as it were, you're in control of the situation.

THE WITNESS: Thank you.

THE CHAIR: Now you speak, to my ear at least, very clearly. Could I ask you, nevertheless, maybe just to speak a little louder----

A Louder, okay.

THE CHAIR: -- maybe with a little bit more projection than you would in normal conversation because the room has to hear you. The microphones are intended to help but from the perspective of someone wearing hearing aids, I'm very conscious of the need to be clear.

A Okay.

THE CHAIR: Now, Mr Mackintosh?

Questioned by Mr Mackintosh

Q Thank you, my Lord. Dr Agrawal, I'm wondering if I can take your full name?

A Samir Gupt Agrawal.

Q Thank you, Dr Agrawal. Your qualifications appear in the HAD Report in bundle 44, volume 1, at page 184. We'll just put that on the screen to start off, page 184, please. We can obviously read them and we can read your publication, what your particular service interests and your current posts involve, but really just to sort of set the scene, what's your current post?

A So I'm a senior lecturer and honorary consultant at St Bartholomew's Hospital and Queen Mary University of London.

Q And what's your current role there? What does it currently involve?

A So I'm a consultant haemato-oncologist. So I work in the haemato-oncology unit at St

Bartholomew's Hospital where we manage adults, people 18 years and older, with the full range of haematological malignancies and we perform the full range of treatments available for those disorders, including so-called bone marrow transplantation.

Q Thank you. Now, within that, what's your principal specialism? The area you feel, as it were, is the core of what you do?

A So I think it would be fair to say within-- I have a reputation for being the expert, particularly in various forms of leukaemia, in bone marrow transplantation, and specifically and particularly for the purposes of this Inquiry, infection management in people with these disorders.

Q Now, I wonder if you could expand on – I can tell you it is important to this Inquiry – what you mean by infection management in people with leukaemias.

A Okay. So I think it is very important to be clear that I'm a haematologist, I'm a haemato-oncologist, my academic qualifications are in the immunology of cancer and the immune response. I am not an infection disease specialist or a microbiologist, so my expertise in infection management is to do with the clinical application of how we manage

protocols, how we try and prevent infection, how we investigate, and ultimately how we treat either proven or suspected infections. Within that, I've developed an expertise and international reputation specifically in the management of invasive fungal disease, which is one of the issues for the Inquiry. So in other words, airborne infections.

Q If we can just step back a bit, we heard some evidence on Tuesday from Professor Gibson who heads the paediatric Haemato-oncology Unit at the Royal Hospital for Children in Glasgow, and she talked about, I think if I understood her correctly, the idea that, depending on a treatment and depending on the child, one might have an anticipation of how long a period of neutropenia will last, how long, what form it will take, how deep it will be, and one will then plan the treatment and the management of infection around that knowledge. Are we talking about the same sort of conceptual idea? That you know about the patient, you know about the condition, you know about the treatment, and then you react or you plan for infections on that basis?

A Absolutely. So, I mean, in broad terms for Professor Gibson, I completely agree with everything

you've just told me. It's-- There are two broad things happening here. One is, what is the underlying malignancy? And the second thing is, what are the treatments being proposed for that malignancy? That will give us----

Q And how those impact on immunity and----

A Absolutely.

Q Right.

A Yes.

Q Now, we'll come back to invasive fungal disease, I'm sure, later on. You've obviously described your role in infection management, but what would be your working relationship with microbiologists in this context?

A So if we just go back a little bit, let's give some perspective. So in 2005, I started – and this has now become the largest meeting in Europe – an annual meeting addressing questions of clinical mycology. So that's fungal infection, and that's something we've been doing now for 22 years, and that's really given me a certain expertise in that area. In terms of the day-to-day working in the unit, we've developed what's called Antimicrobial Stewardship Programmes, and that's a key part of how we interact with our microbiology colleagues and infectious disease colleagues, and my input into

that was I was the one who instigated that approach----

Q So what is antimicrobial stewardship?

A So antimicrobial stewardship is really regarded as being a requirement for all NHS institutions, principally to manage antibiotic prescriptions with a view to trying to limit antimicrobial resistance, which is a major global health concern. What we do is, with microbiology colleagues, infectious disease colleagues, and pharmacy colleagues and myself, we perform what's referred to as a AMS antimicrobial stewardship round, looking at all of our inpatients with respect to their infection prophylaxis, infection treatment, if they're on treatment, and diagnostics.

Q So, at a very high level, you're trying to not make the problem of antimicrobial resistance worse by the treatments you're applying to the patients?

A Absolutely.

Q Right. Now, did you watch any of Dr Drumright's evidence yesterday?

A I saw some of it. I was travelling in a train to the----

Q You were standing in a train?

A No, I was travelling via

train to Edinburgh, so it was limited.

Q It would help me to understand when you came in, as it were.

A In the afternoon.

Q So, after two o'clock?

A Yes.

Q Right, well that's useful for me because there's points I'm going to come back to. Now, before we do ask questions, I need to identify your reports and ask if you formally adopt them as far as your evidence. Now, I have in mind six documents----

A Okay.

Q -- and I wonder if we can just identify them. There is this document we're in here, which is what we've called the HAD Report, which starts on page 5 of this bundle, and that was produced by you and Professor Hawkey and Dr Drumright in July of last year.

A That's correct, yes.

Q Then we have a short note about your calculations for chapter 8, and that's in 44, volume 2----

A Yes.

Q -- at page 107. Yes. Then we have what we've come to know as the HAD response document, because you were asked by us to respond to various reports we got into your report, and that's 19 July, and that is bundle 44, volume 5, at page 20.

A Just to confirm, this is a so-called first response. This is-- HAD 1 was our response to HAD----

Q No, this isn't the questionnaires. I'm about to come to the questionnaires. This is the standalone document that sits alongside the second questionnaire.

A Oh sorry, yes.

Q Yes.

A Thank you. Yes.

Q Then you have an earlier report which you produced for NHS Greater Glasgow in respect of Ward 4C in 2021, which is the same volume at page 103. Now, can I just check with you: you've presumably seen this bundle?

A Yes.

Q Did you apply any of these redactions?

A No.

Q No. So the Inquiry applied these redactions, yes. Then we have the responses to two questionnaires we asked you. So it's back to volume 2 of 44, at page 12. So quite soon after you and your colleagues were-- instructions were transferred to the Inquiry, we sent you a questionnaire which asked some sort

of initial questions----

A Yes.

Q -- and you responded.

A Yes.

Q Yes, and then a second questionnaire is in 44, volume 5, at page 4. Now, are you willing to adopt all these documents and responses as part of your evidence?

A I am.

Q Thank you. Now what I wanted to do is then to look at your letter of instruction, because that's in 44, volume 1, document 3, at page 234. It's dated 21 November 2022. Now, it seems to be in exactly the same terms as Professor Hawkey's.

A Yes, I believe so.

Q How was it you came to be instructed in this project? What was your first awareness of it?

A If my recollection serves me well, I think [redacted] must have contacted me, since we'd already been in contact with respect for the previous report----

Q Into 4C?

A Yes, that's right. So with respect to ventilation and infections pertaining to ventilation, with the request that, would I be willing to get involved in what was a broader investigation, in other words, this Scottish Hospital Inquiry, in terms of infections not limited to paediatric and adult haemato-oncology, possibly on a wider scale, which Professor Hawkey had been engaged, and would I be happy to do that with him.

Q So Professor Hawkey had already been involved before you?

A Yes, I believe so. Yes.

Q Yes. So what was the original nature of this investigation as put to you first?

A Well, as it says, I think, in the letter of instruction, that certainly when we were discussing this with the CLOs-- Is it----

Q That's the name of the agency, yes.

A -- to refer to the CLO, that we were told that the scope of the Inquiry was potentially broadening. So that was one aspect of it, and they wanted to have an independent opinion on, "What were the actual infection rates occurring within the Queen Elizabeth University Hospital and Royal Hospital for Children in terms of the period of time since these institutions opened?" with the background that there had been concerns around potentially patients acquiring infections because of failures in the environment of the hospital. I think that's broadly how it was put to us.

Q So, ultimately, your investigation into both bloodstream infections and Aspergillus became focused on haematology-oncology patients?

A Yes.

Q But it seems from your initial conversations, the look was at the whole hospital.

A Well, the impression certainly I had – I'd assume my-- Professor Hawkey had as well at that stage – was, it wasn't necessarily restricted to the haemato-oncology population, but in terms of trying to address the question from a practical perspective, it was reasonable to look at those patients who potentially would be most affected by failures of the environment with respect to infection prevention----

Q This is your idea that these patients would be the ones having intensive chemotherapy----

A Yes.

Q -- and, if I get the acronym right, HSCT?

A That's correct, yes.

Q That is what's known as bone marrow transplant.

A Yes, colloquially. Yes, yes.

Q Right. I think narrowing might be the right word: this is narrowing from the whole hospital to that group.

A Yes.

Q Did that come from you or from those who instructed you?

A If I'm frank, I actually can't remember if-- which direction that came from. From my personal perspective, it was a sensible approach to make the task manageable. The task already seemed to be potentially a very challenging one because of the scale of what we might be dealing with. In terms of data, I mean, in terms of information.

Q Yes. Thank you. Then if we just step back to Professor Hawkey, had you dealt with Professor Hawkey before?

A Not in any capacity I can recall, no. So, neither professionally nor in any other capacity.

Q Right. What I wanted to look at is in the letter. If we go to the second page-- Well, firstly, go to the bottom of the first page. There's the background-- So 234. We start at the bottom of 234. There's a narrative description in this letter of instruction which runs over the page and makes reference to various things that have happened in the hospital.

A Yes.

Q Then the instructions at the bottom of the page state that:

"All of these infections have been the subject of intensive investigations, both by NHS GGC, and external bodies..."

What I wanted to understand is, I think it's the case that you haven't reviewed these other investigations. Have you?

A No.

Q Can you help us understand why?

A Yes. So when we were discussing this, the question was put to us, "In our opinion, how could we best answer the questions being put to us?" And essentially, the question being put to us was, "If there was-- If there were failures in the water system at whatever level, in the ventilation system, and that was leading to infection in patients, patients requiring infections because of those failures, how would that manifest itself in terms of infection rates clinically? What would we expect to see?"

Q And do we actually see that from the top of page 236 in broad terms?

A Possibly, yes. Yes.

Q Yes. Now----

A So, to answer your question-- So that was-- So Professor Hawkey and myself discussed what was the best way to try and answer that question. We'd been presented with, as it says in the letter of instruction, the fact that a lot of information had already been considered, looking at these issues, and what had not been done, was there was no, if you want, baseline and a comparator to see what were the infection rates and were these within any expected limits or not. That was very much how we focused our approach.

Q So you were looking for a comparator?

A Yes.

Q Might it be fair to say that, in a sense, you're testing the hypothesis that if you have a comparator or indeed a patient group that moves from somewhere to inside the hospital, there will be a difference-- or will there be a difference in the rates between either the comparator and the hospital or the place before the move? Is that effectively what you were doing?

A Well, we felt there was an obvious comparator, and that was, before the QEUH and the Children's Hospital were open, the same population of individuals were being

managed in-- at other sites. So looking at the infection rates at those sites would offer us some form of comparison as to what then occurred when that population of patients moved.

Q It has been suggested that the weakness of that approach is that the places where the patients-- if we stay with the haemato-oncology patients, the haemato-oncology patients were located beforehand, were to some extent older buildings, some Victorian----

A Yes.

Q -- and therefore a comparison between the hospital environment in an old building and a new building is not entirely fair.

A I can understand the logic of those statements and I accept that. I mean, we're also faced with the reality of, "What is a reasonable comparator and how can we aim to get the-- the best data to give us, one, a baseline of what was actually occurring, and does that represent data of concern or not?" So I would put it to you that it was a reasonable comparator to take – not the only one – but I know in other reports that perhaps we'll discuss, other comparators were done by other experts who've given evidence to the

Inquiry. So there were other approaches to take.

Q Well, I mean, one of the options exists which I suppose is for the adult haemato-oncology patients-- would a suitable comparator be in your unit at Barts?

A Potentially, yes. Yes.

Q Why didn't you look at that as an approach?

A It wasn't a-- Well, I mean, it was a question of deciding which approach to take, within a timescale that wasn't entirely defined, plus resources. So the resources we were working with at the time were those of the CLO, and the data available in the Health Board's system.

Q So you had access to-- You could have asked for any information that Greater Glasgow and Clyde held, could you?

A Well, I assume so, yes.

Q Yes, okay. The other comparator point that is made-- Well, two other points made. One is made, and it's made against Mr Mookerjee's exercise and also an exercise carried out by HPS briefly in October '19, is that the danger of comparators is that they will be too different. They'll either be too big or too small or too new or too old or the population will change. Do you feel that by having simply, in a

sense, one comparator for each patient group – the place they were before – that you run the risk that, I think, the concept is confounding, that other factors will come in which will muddy the waters, literally, in the design of the study?

A I think everything you've said there is valid and it-- it raises the challenge of having the most suitable comparator. I mean, another issue that it is impossible to – unless statistically it can be done, but that's not a question for me – address chronology. Because if we're looking back in time, we're looking at a period of time where things were necessarily different and medical management evolves, but that's the nature of making comparison. I think we accept the limits of what we can do. So I don't disagree with you, but we're also making an attempt to have some sort of comparator in order to make sense of what we're looking at.

Q What was your state of awareness of what was being done by the Health Board to address what was seen by some, including some people in the Health Board, as inadequacies in the ventilation system of the building?

A Well, that takes me back to the report that I'd prepared earlier in

2021 with respect to a specific question around the ventilation system for the Adult Bone Marrow Transplant Unit, and----

Q Sorry, you looked at 4C?

A Yes.

Q It's not the Adult Bone Marrow Transplant Unit----

A Oh, sorry.

Q Did you look at 4B?

A I'd have to check my report, sorry.

Q Just check in here. When you say the Adult Bone Marrow Transplant Unit at Queen Elizabeth, what ward is that located in now?

A Again, I'd have to check, I'm afraid.

Q Well, I mean, is it the ward you were looking at when you wrote your report in 2021?

A Without-- I can't answer that question.

Q Let's go and look at the report, which is 44, volume 5, document 3, page 103. So I don't particularly want to go into why the report was prepared, over the page. We passed through your qualifications, perhaps. We've seen them before. I'm not sure I've got the right document. So I can take you straight to the right pages.

THE CHAIR: I think it probably is

the right document, Mr Mackintosh.

A I think this is the right document.

MR MACKINTOSH: It is the right document. I just wanted to jump around it without taking a long time to walk through, and of course I'm wasting time getting my copy in front of me. Maybe the place to go to is page 105, the next page, where you've done a summary, and you see at the start, "Ward 4C." So do you understand that to be the bone marrow treatment ward?

A Without checking, I'm not sure.

THE CHAIR: Have you visited the Queen Elizabeth?

A No, it's not been part of our activity, your Lordship.

THE CHAIR: No. So you've never been to the hospital?

A Sorry, no, I've never been.

MR MACKINTOSH: I suppose a question that would help me resolve this, actually-- Because I was always feel when people can't quite remember facts, what you do is try and pull at a thread to see if it helps memory. If we go into your report, you discuss your unit at St Bartholomew's, and I think you do that-- Let me just make sure I get the right page reference because I don't want to jump around too much. Well, can we go to page 116? So, do you see at 5.5, you were discussing infections? We'll come back to this in substance, but the reason I'm going to it is to mention, do you see how you're discussing a comparison with the infections in the haemato-oncology unit at Barts?

A Yes.

Q And you go on to the next page, at 117, in the discussion about material risk of infection, to make reference at (f) to the HSCT service at Greater Glasgow Health Board.

A Yes.

Q Was it your understanding that Ward 4B carried out what is colloquially known as bone marrow transplants?

THE CHAIR: Do you mean 4B or 4C?

MR MACKINTOSH: 4C carried out bone marrow transplants.

A I actually can't remember any----

Q Well, what I think I'll ask you to do is we're going to get-- We've got lots to do today.

A Yes.

Q So when we come back after lunch, I wonder if you might see if you can-- I know you brought your

notes with you.

A Yes.

Q I wonder if over lunch you could just check your notes, because I'd like to come back to this at two o'clock.

A That's fine.

Q What I'll do is I'll go back to where I was and----

A Can I just clarify something?

Q Yes.

A So point (f) there, saying HSCT service. I know HSCT is our abbreviation for haematopoietic stem cell transplants.

Q Yes.

A At St Bartholomew's hospital, we do not have a separate transplant service.

Q Yes, so----

A So when I refer to that, as it's stated there, the JACIE accreditation, which verifies whether a unit is working at a suitable level or not, is effectively accreditation for the whole service.

Q Right.

A So the point----

Q I think what I'll do is I'll put something to you----

THE CHAIR: Could I just take a detail, because I'm not entirely clear about this? The JACIE accreditation is an international minimum requirement for transplant services.

A It is a minimum requirement for, let's say, bone marrow transplantation.

THE CHAIR: But it is focused on transplants.

A It is. However, your Lordship, the JACIE process reviews the processes of the unit, and in an institution like ours, St Bartholomew's Hospital, our processes are the same whether it's a stem cell transplant or it's an individual receiving other forms of treatment. So they, in fact, cover out our standard operating procedures, regardless of whether it's for transplant or non-transplant.

THE CHAIR: But other sites might have a different arrangement.

A They may operate differently.

MR MACKINTOSH: I wonder, actually-- I think I might have found a way out of this. If you go to page 125 of the HAD Report, so it's 44, volume 1. This is in your Aspergillus chapter. Volume 1 of 44, page 125. So we'll come to this eventually, but if you look at the paragraph below the figure:

> "The Adult BMT service moved from the Beatson Institute to Ward 4B QEUH on 6 June

2015 but then returned to the Beatson Wards B8/9 on 8 June 2015. Adult BMT was then permanently placed at QEUH 4B from 30 June 2018. The distribution of cases in the Adult BMT service in the split site years of 2015 and 2018 ... was: six versus none..."

Well, that's about cases. We'll ignore the cases. Then what we do is we go back to the BSI chapter in the same volume, page 69. So this is where you're about to discuss bloodstream infections in adult patients in chapter 7. See at the top of the page:

"For adult patients, data were examined both by consultant sector, BMT, North and South, and by months and years in which consultant section was assigned to either the QEUH or all other locations."

And then it describes the allocation. So, South and BMT were attributed to Queen Elizabeth the following way:

"...the South sector became QEUH in May 2015 and remained part of QEUH through 2023 when data provided stops; BMT became part of QEUH for a period of approximately one and a half months across the months of June and July in 2015 and then from June 2018 onwards. As bed days were only available...."

And then you describe the split. So what I want to put to you, but ask you to reflect on, is the Inquiry understands that the Adult BMT Unit when it's in the Queen Elizabeth is in Ward 4B, and that's where what is colloquially known as bone marrow transplants take place. The South Sector Haematology Ward, when it's in the Queen Elizabeth, is generally in 4C and at one point it pops into 4B when the others aren't there, and before that it was in Ward 24 in the Southern General Hospital. Now, can I ask you just to check that over lunch?

A Okay.

Q And I'll come back to that at two o'clock, because it seems an important point. What I'd like to do is to return to our discussion of methodology.

THE CHAIR: Just flagging a point. At some point, I would be interested in knowing from Dr Agrawal the detail of his unit in Barts.

MR MACKINTOSH: Yes, absolutely. We'll do that then, I think, my Lord. So what I'd like-- We'll come

back to that in terms of your other report, and we'll do that all after lunch.

THE CHAIR: Thank you.

MR MACKINTOSH: So what I want to do is just go to the appendix to the letter of instruction, which is at page 237 of the same bundle, because both you and Professor Hawkey received the same appendix, and I want to have a discussion with you about the conceptual design of the whole study. Do you see how at (3) it states, "If there was 'widespread contamination' of the hospital's water system," and then there's a list of things that this might be down to, and there's a Roman numeral footnote, and you see the document at the bottom.

A Yes.

Q Have you read that document?

A I don't think I have, no.

Q Right. And then a series of questions are asked, and I suppose (5) is where Dr Drumright comes in. So would it be fair to say the process ascribed here is you are being-- Well, what is the process? What are you being asked to do before you get to the data? What are you comparing with what?

A Well, the premise is there may be infection arising in patients in the hospital from the water system in some way and/or failures in the ventilation system.

Q Right.

A And from that, what would we expect to see and how can we try and answer that question?

Q So in order to do that exercise for the water, to what extent do you need to know what is the nature of the suggested failure in the water system?

A Well, our approach was not to consider that, and the reason for that was we decided to focus on what were the infection rates that we could establish, because what we were aware of-- We were aware of concerns around apparent increased rates of infection, and so what Professor Hawkey and myself discussed was, well, how would we establish that? How could we demonstrate that that was the case or it wasn't the case? So that is how we approached trying to answer this question, and hence our desire to try and get as much data as was available from Glasgow Health Board in terms of the infections that had been documented.

Q So, just to ask the same question in terms of ventilation, in order to answer that sort of question

around ventilation, do you need to know what the nature of the perceived flaw in the ventilation system is?

A Well, I had some knowledge about that because of previous work already, but it was really the same approach, so the same approach to say, "What were the observed rates of infection?" and then attempt to see, "Does that correspond to what may be expected or was it an exigence?"

Q When you say what may be expected, did the CLO – other than by this footnote, which would tell you the answer because we've read the document – tell you that the Greater Glasgow and Clyde Health Board publicly expressed position is that by a point in 2019, or maybe a little later, but in that sort of territory, they had the water system under strong control and that intervention such as putting filters on taps and showers in all high-risk areas had been completed in March, April 2018? Did they tell you that other than by the reference in the document in your letter of instruction?

A No, I'm fairly confident this is something we've become aware of more recently. It was not something that we were told at the time.

Q Well, shall we look at the document you didn't read? I wonder if we can go to----

THE CHAIR: Really just for my note, Dr Agrawal, to understand your last answer, you only became aware recently of any information about remedial measures. Have I picked you up correctly?

A I think I need to just caveat that slightly, but the details-- I mean, I think we were aware there were many efforts going on to address perceived risk, but the details of those efforts we had not investigated. We didn't have the documents to look at.

THE CHAIR: When you talk about the details, you mean the nature of the measures?

A Yes.

THE CHAIR: And the dates when they were applied?

A The exact dates, no.

THE CHAIR: Thank you.

MR MACKINTOSH: I think probably-- Yes, it's in bundle 18, document 11, page 819. The reason I want to put this to you is because I want to suggest something else that you could have done in terms of the study design. So if we look at these, this is-- Yes, 819. This is a report by Health Protection Scotland. Are you aware of who Health Protection Scotland are?

A Yes.

Q Yes. So they're not part of Greater Glasgow and Clyde Health Board.

A Yes.

Q And they produced a summary of incident and findings of the water contamination incident with recommendations, and it's December '18. And then if we go to page 821, we have an executive summary, and you see in the third paragraph it explains that:

> "Between ... 29th January and 26th September ... 23 cases of blood stream infection...."

And then the second sentence:

> "As a result further testing of the watter supply was undertaken across both hospital sites...."

And it refers later in the decision to put "point of use filters in wash-hand basins in Ward 2A ... and other areas where patients were considered high risk"; it talks about drain contamination, and there's a long list of interventions there.

Now, what I want to suggest to you is that if you wanted to do a comparator between a-- A comparator that would have been perhaps more interesting and less risky would have been to compare before the interventions with after the interventions, because then it would be the same hospital, the same patients, but in the case of the water, with luck, the water isn't reaching patients in the state it previously was. Would that have been a helpful thing to look at?

A I think effectively we have looked at it.

Q But you don't know the date of the interventions, do you?

A No, but in the analyses performed by Dr Drumright, she has analysed and taken all the data available from the Glasgow Health Board, so----

Q Well, no. Let's go back to bundle 44, volume 1, page 237. This report was available to you when you were instructed and you didn't read it. So you had the report from HPS available to you in November '22 describing the intervention, and yet you didn't read it. And the difficulty that poses to me is a question that I'd like you to think about, which is this Inquiry has been given your report and we have been encouraged to give weight to it, and the difficulty that I'm pondering is in a normal concept with expert witnesses, you sort of expect an expert witness to have considered all the available material, to have reached one conclusion and then to-- "defend" is not quite the right word-- deal with it

in the context of other people's opinions and maybe small extra pieces of information.

What you don't expect to happen is an expert to produce an opinion without taking account of information that was available to them at the time. And so what weight should we give to your conclusions when you didn't look at material that you were given at the time that would have told you what was done to address the widespread contamination of the water system that was found by Greater Glasgow's own employees?

A The way I see this-- I think that there's separate issues here. So, one is contamination of the water system, failure of the ventilation requirements. The other is what were the impacts of those findings on patients? So we took an agnostic approach from that perspective, hence the desire to look at data over time in order to make comparisons. I realise the point being made that comparisons with other institutions may be limited by the fact those institutions had different environments; but equally, the data we looked at does include all of the time periods and time points you're referring to, and hence we can look at infection rates in relation to those time points.

Q But you didn't in the original report?

A Not in our original report. No, we didn't. But subsequent to comments made to our original report, such analyses have been done.

Q Right. Well, let's move on. I'll come back to the----

THE CHAIR: Just, again, for my notes. "We took an agnostic approach." What did you take an agnostic approach to? Was it whether or not there had been contamination or failure in the ventilation system, or the second question, which was what were the impacts?

A We did not make assumptions about whether there had been or whether there was contamination, but we accepted that if there was, what might we expect to see? And also we didn't make any assumptions of the impact of other measures that may or may not have been happening within the Glasgow institutions to try and address the problems they were seeing, because what we wanted to do was try and establish what the incidence rates were and, from that point, then try and make some statements about what was the impact of those perceived or real failures on patients in terms of infection rates. So that was, when I

say agnostic, I mean in that sense, your Lordship.

MR MACKINTOSH: But Dr Agrawal, if you wanted to look at what was the impact of perceived failings in the system, do you not need to know when they stop? If they stop?

A Well, I would suggest that we would see that in the data.

Q Well, I appreciate that but, in summary, your original report, you don't see it in the data and then you do, to some extent, see it later. So I'm wondering, is it helpful to the Inquiry to attempt to look at what's the impact of a perceived failure without knowing its temporal or geographical extent?

A Well, I think from the work that has been done, the answer to that is, yes, because the analyses that I've seen, which on re-analysis, the figures and graphs do show-- firstly, I would absolutely agree with you, our initial impression that we couldn't see a signal of increased infection rates in bloodstream infections in the paediatric population was changed with further analyses performed by Dr Drumright. I'm not aware the data changed. It was the nature of the analysis that changed, if the model was used.

Q Right. Well, I'll come back to that after that, I want to get on with some other stuff first. So I think probably the most important thing to do in terms of basic housekeeping is just to make sure I've got the division of labour between you all so I can ask the right questions in the rest of your evidence for the right person. Now, the paper consists of an executive summary and eight chapters, and Dr Drumright described it, in essence, that chapters 1-6 were very little to do with her. Part of chapter 7, so point 2, was her, and then subsequent work on Aspergillus after the report had been produced was her as well.

A Yes.

Q Would you agree with that in terms of Dr Drumright's involvement?

A Yes.

Q Right. Now, for you, working backwards, is chapter 8 effectively your work?

A It is.

Q Right. Well, we'll deal with that with you. Within the rest of the report, I have already been discussing with you conceptual design----

A Yes.

Q -- and you're comfortable to deal with that. The design of the whole study, is that something you feel

comfortable to talk about?

A Yes.

Q Yes. One of the topics addressed within chapters 1-5, or 1-4 rather, is the idea that you need to know a normal rate in order to understand when there's been a change. Is that something I can discuss with you?

A Yes.

Q Yes. Then there's the topic of whether whole genome sequencing can be used to prove the absence of a connection. Should I address that to Professor Hawkey or to you?

A I can make general comments, but I think Professor Hawkey-- that is his area of expertise. He is the person who has written that section of the report, so I would have thought he'd be----

Q Well, we'll do that briefly and see how we go.

A Okay.

Q Then there's the topic which Dr Drumright was keen to defer to the two of you was whether there was an issue with antibiotic resistance. Is that something you can help us with?

A Yes.

Q Right. Then chapter 6, ventilation that would be something we can discuss with you?

A Yes.

Q And did you write most of that?

A I did.

Q Right. So we'll do that after lunch as well.

A Okay.

Q And then within chapter 7.2, do you feel comfortable addressing interpretation questions around the BSI results?

A Yes.

Q Right. Now, can I ask you this question? Who amongst the team-- Well, actually, I'll address it to you. What experience or expertise do you have in paediatric haemato-oncology?

A Through my training as a haematologist, so as a registrar in haematology, I worked at the paediatric unit at the Royal Marsden Hospital, St George's University Hospital, which provides the PICU facilities to the Royal Marsden Hospital, managing children with haematological malignancies. As an adult-- As a consultant, I would not regard myself as an expert in the management of paediatric haematological disease.

However, I do regard myself as knowledgeable about the infections

that individuals acquire, as we discussed earlier, who have haematological malignancies and certain-- and transplants and intensive chemotherapy, and the principles of those issues are the same, I would suggest, in adults and children.

Q In terms of the practicalities, when did you last treat a patient under the age of 16 with a haematology-oncology condition?

A That would be as a registrar, so that would be many, many years ago.

Q So when did you become----

A In the 1990s, 1990s.

Q In the 1990s, and that would be when you last worked in the paediatric haemato-oncology unit?

A Yes.

Q It's been suggested by the CNR expert panel in their rebuttal that none of the three of you have experience sufficient to be an expert witness in paediatric haemato-oncology. How do you respond to that suggestion?

A Is that rebuttal suggesting that we should be experts in managing haematological malignancies, or is it suggesting we should be experts in managing infections that such individuals may get? Because I'm slightly unsure of what the suggestion is.

Q Well, it doesn't say that, and I suppose that the question of whether you are an expert is ultimately a question for the Inquiry, not for Professor Stevens or anyone else. So the way I reflected on it was this, and so I'll put this to you. We've heard evidence, which I don't think is challenged, that paediatric and haemato-oncology patients are not little adults. Their size, the way they move around, what they do, how they behave and how they can't be expected to maintain the same level of hand and line cleanliness as an adult makes them different. Would you accept that's something that might be true?

A That is true.

Q Yes.

A But the implication of that is that they are at greater risk.

Q Well, that's what I wanted to come to.

A Yes.

Q The same thing that might imply the same thing is that because they're smaller and the diseases they suffer from last longer, are sort of more systemically harmful, are they actually a lot more vulnerable than adult patients?

A I'm not sure I could agree with that statement. I mean, in adult haemato-oncology today, we are dealing with an increasingly older population that we are now treating in a manner that we wouldn't have done 10/15 years ago.

Q But when----

A As the individuals get older they have significantly increased risks associated with their management, particularly infection. So I think that balance of who is most vulnerable is not so easily defined.

Q But what should the Inquiry do when faced with evidence from two or three Glasgow consultants, including Professor Gibson and Professor Stevens, who see the paediatric haemato-oncology patients as singly more vulnerable than their adult equivalents, partly because of their physiognomy, partly because of the diseases they suffer from, partly because, well, they're children and they play and they do all these things.

So when they tell us that, and you say in your report-- and we'll find that in the text, which is-- if we go, I think, to this bundle. I'm pretty sure it's on page 61. You make a statement at the bottom of the-- this is in chapter 6, and I'm assuming that "would this become evident" as a reference to "would a problem with the ventilation system," in the heading?

A Yes.

Q "Would this become evident by way of an increased rate of infection in the patient population as a whole or are there particular vulnerable patient groups where this may be more evident?"

And then there's a discussion in here, and what I noticed is that you list in this section the sort of people who might be most vulnerable in the bottom of the paragraph:

> "The highest-risk populations is the group of patients with haematological, and other, cancers undergoing intensive chemotherapy and/or HSCT."

Then over the page we move to chapter 7, but if we go to the executive summary on page 6, is it your suggestion that we would see any increase in infections in both the adults and the paediatric groups?

A Yes.

Q And so the difficulty I'm putting to you is that if they use people who are currently treating the patients in the hospital and Professor Stevens, who used to be head of the Bristol unit,

are telling us that the children are more vulnerable, then the use of the word “both” in that construction might be problematic and, therefore, it might be possible to see a signal in the children-- yes, this is a problem with glasses, in the children, but not the adults. I want to put that to you, but I’m conscious that I’m putting it into the context where you’re not a paediatric haemato-oncologist. So how am I supposed to deal with that problem?

A Well, my answer would be I’m not the right person to definitively answer that. My experience in terms of clinical care is with adults, and the nature of the defects that render this population of individuals, be them adults or children, at high risk of infection are principally the same. I understand that the behaviours of individuals may be different, but that’s to ignore the behavior of adults and assuming that adults are not putting themselves that risk in other ways that, in fact, children are not exposed to.

Q Because I appreciate that----

A So I’m going to come back to that balance thing. I’m not sure when my colleagues say what they say, what that’s-- how they would justify their own statements. I accept

there are differences in children and adults, absolutely, but I’m not sure you’d be so definitive about the difference in risk of infection.

Q Well, the reason that I’m asking you the question is you’ve been definitive about saying we will see the signal in both, and I think it’s fair to say that at the end of Dr Drumright’s recalculations, to some extent, and that matter is a matter for debate there is a signal in the paediatrics, but by no means is there anything similar in the adults.

A Yes, I’d agree.

Q So if that is the case, then this “both” word becomes quite important. Are you telling us that we should expect to see-- if there is a widespread contamination of water system, we should expect to see the signal in both and, therefore, the fact that we only see it in one means we can exclude that? Is that what you’re saying?

A I would absolutely expect to see a signal in children and adults in these circumstances.

Q And you’re comfortable that you have the expertise necessary to give that opinion?

A Well, I’m certainly comfortable I would expect to see it in adults. Yes.

Q Right. Now, we'll return to the adults and the placement and their exposure to these water and ventilation systems after lunch, but what I want to do now is to move on to the concept of the normal level of risk, because we've had a lot of evidence and opinion about using the word "normal" and "unusual," and trying to work out what they mean in different contexts. So I wonder if we can go to the executive summary, we're on it already, and it's paragraph 5 and it describes what is an outbreak in the summary. I'm assuming you stand by the words in the executive summary?

A Yes.

Q Yes, and the paragraph before is, "To assess if there is an increased risk of infection, a normal level of risk must be established." What do you mean by "a normal level of risk"?

A We are all at risk of infection and this patient population is at great risk of infection. There is an inherent-- It's not just risk, there's inevitability that, given the treatments, given the immune suppression, given the damage that occurs to individuals based on the treatments they get intravenous lines for access, that infections will occur. So, "normal" implies what would be expected in

those circumstances and, hence, to talk about increase above what may occur because of the circumstances of the treatment of the individual and their underlying disease. So, I regard "normal" as being what is, in a sense, inevitable and an outbreak then, as follows in section 5 there, is something that implies something is happening that would not be expected.

Q Because one of the things that's been confusing me is that I wondered if "normal," this concept of "normal level risk" has different meanings at different scales, and what I mean by that is I take it from what you're saying that if you think about the general risk of infections associated with lines, are you saying there would be effectively a certain inevitability there will be some? Is that effectively your position for line infections?

A Line infections and other bloodstream infections, yes, absolutely.

Q So, in general terms, if you take a collective group, there will be a normal-- there's inevitability about it to some degree.

A Yes.

Q You can push it down but, eventually, you can't push it any further.

A Yes.

Q Right. If you take an individual organism, one I want you to look at is Mycobacterium chelonae. The reason it comes up is because it's quite an interesting scenario in the hospital which I'll just mention to you. So there's a case, 2018, in the children's unit, and there's a case in 2019 and it's only, in the Inquiry, that we realised there's a case in the system in 2016 as well, but no one at the time notices. All the evidence of those involved in that treatment and, indeed the Inquiry experts, is that there isn't really a normal rate for Mycobacterium chelonae. It is something that should not happen. Would you accept that for an individual organism? You can drop another one into that definition if you feel it's not an organism you know well.

A Well, I'm slightly unsure about when you say, Mr Mackintosh, it shouldn't happen. I mean, things do happen.

Q Well, I appreciate that's what I'm trying to explore. So if we sort of make it very vague, to make it the concept, there's been lots of examples of members of the infection control team and the clinicians in the unit saying, "We came across unusual infections."

A Right.

Q And the way they mean that is that someone mentions patient has got infection A and they go, "Goodness gracious me, I've never seen this before, I have to go and look it up."

A Yes, yes.

Q That's basically the definition they're working on. And the same people largely then say, "There can't be a normal rate for something that's so rare I had to go look it up."

A Yes, yes.

Q And that's obviously at the level of one organism. Would you disagree or challenge that particular conceptualisation of normality in that context?

A I don't challenge it. But the question I would ask is----

Q Or how helpful is it?

A Yes.

Q So how would you make it helpful or give it meaning? Would you have to increase the scale?

A Well, I think-- well, I think that's extremely difficult. So in the context of, are we trying to establish that this very rare organism that doesn't normally cause bloodstream infections, even in the very vulnerable population, but we're finding it in an individual; yes, that would certainly

cause me to think, “Well, why? What’s happened?”

Q Yes.

A And then we go to, has it come from the water? Is it related to the air, where does it come from? I can’t really see a way of addressing that without having some techniques such as whole genome sequencing, but basically establishing that the clinical isolate is the same as can be found in the environment or wherever. We felt the organism----

Q So you would feel that the normal rate concept you’re describing here in paragraph 4 isn’t really relevant to this single organism?

A No.

Q No? Right.

A No, it couldn’t apply to that.

Q Okay. Let’s go back to paragraph 5 and the idea an outbreak. You’ve defined an outbreak as:

> “...an increase beyond the normally expected numbers of infections due to a specific bacterium. Relatedness of these infections in time and space provide circumstantial evidence for cross infection. A strict definition is the occurrence of two or more cases of infection caused by genetically indistinguishable micro-organisms.”

Is that how you managed your own patients? Would you wait for the whole genome sequencing before deciding you have an outbreak of a particular bug?

A May I give an example?

Q Please.

A Okay. So, three years ago, we had a case in our unit of a bloodstream infection with what’s referred to as a multi-drug resistant organism, an MDRO. Extremely rare in our practice. We see very few of these organisms. A few weeks later, we had a second case. A different patient, a different part of the ward, and so that rang alarm bells, because we-- it’s not what we expect.

Q It’s back to that definition of “normal”?

A Yes, exactly. So we didn’t then wait to say, “Well, let’s do whole genome sequencing.” An investigation was started to try and establish what was happening. By the time we did get whole genome sequencing back, to our surprise they were actually completely different. So it’s the circumstantial nature of the evidence that needs to be verified by something that’s more definitive. So I agree I wouldn’t wait, but on the other

hand, we would be making assumptions. Since then, to my knowledge, we've not seen another case. So these things happen. The assumption was the organism had come in with a patient who'd come into the hospital from another institution.

Q Well, that enables me to ask a generalised conceptual question about whole genome sequencing, and I'll perhaps use your experience as an example. I don't think there's anybody who disagrees with the conclusion that if two bloodstream samples are not closely related in terms of their whole genome sequencing, they are not identical. The question then-- I mean, if they are closely related, they come from the same source. That seems to be uncontroversial. You'd accept that, if they had come back to you as within a few snips, I think----

A Yes.

Q -- you would have gone, "That's the same source"?

A Yes.

Q Right. Now, I don't know enough about this particular organism, but if we, for example-- Let's change it slightly and make it an enteric organism that can be in both the gut and the water, it can be colonised on patients, all these are possibilities, and you have two cases in a ward – as you say, in different parts of the ward – and initially you suspect they might be related by just applying basic epidemiological principles. However, you do eventually get whole genome sequencing back, and the two cases are some distance apart in terms of their genetic relationship. What definitive conclusion can you draw about whether there are or are not two different populations of that organism in your water supply?

A Well, firstly, I'm not quite sure why I'm thinking it's coming from the water supply, unless we've grown them from the water supply.

Q No, I use water supply as a-- I'll rephrase that. In your environment.

A Oh, in our environment.

Q So let's imagine that you have this organism which you know lives in the environment. People have said, and it seems clear to me anyway, that if you had a single source that had a single population of micro-organisms who are all genetically very closely related and they get into two separate patients, you will find genetically closely related tests.

A Mm.

Q I've understood that correctly?

A Yes.

Q What happens if, in that environmental source, there are not one population, there's in fact two or three or four? How can the fact that two samples are not closely related exclude the possibility – and I always get these phrases wrong – that the environmental population of that organism in your unit is polyclonal? Is that the right word we're looking for?

A I think so.

Q Yes. How can it exclude the possibility that what you actually have is a polyclonal reservoir?

A So, I really on that point have to defer to Professor Hawkey, as he is an expert in this area and we're drifting into areas of microbiology that really are not my area of expertise, so I think I should not attempt to answer that.

Q Fair enough. We'll do that with him. I think it's on Wednesday of next week. Another topic that's come up is of a similar sort of nature, in that it deals with a conceptual possibility that you want to consider when understanding this data, and that is the concept of antibiotic resistance. Now, tell me whether I'm asking the right question here. Am I right to think that this topic is effectively this: the extent to which the use of certain antibiotics in a unit will influence the population of organisms in that unit such that you get more than a normal amount of them? Is that the topic we're looking at?

A Yes. Broadly speaking, I think you've got it right.

Q Broadly speaking?

A Yes.

Q So at least I'm asking about the right topic.

THE CHAIR: Sorry, just clarifying my thinking: the use of antibiotics in a unit----

A Yes.

THE CHAIR: -- the policy, the practice, will influence the population of micro-organisms. Now, did you say in the unit----

MR MACKINTOSH: I meant that in the broadest sense----

THE CHAIR: -- or in the patients?

MR MACKINTOSH: -- both in the environment, the patients, anywhere.

THE CHAIR: Right. Okay. So it's not simply within the patients?

MR MACKINTOSH: I didn't want to push it in one direction. I want to keep it very vague to check my understanding.

THE CHAIR: All right.

MR MACKINTOSH: So in a

sense, there's a population, we'll not worry about where it is, it is influenced by the use of antibiotics. That's the theory that we're investigating, effectively.

A It's more than a theory. I mean, it's absolutely definitive that the antibiotics used within a Haemato-oncology Unit have a profound influence on the microbiological environment of that service.

Q Indeed, that's why there's considerable policies in these units about the use of antibiotics and what antibiotics they should use----

A Yes.

Q -- and when.

A Absolutely. It's not just because of that, though. I mean, the policies around antibiotics are primarily derived to ensure appropriate treatment of the condition known as febrile neutropenia.

Q Right.

A So that's in general what they're principally addressing, and within those policies there's huge variation, actually, in-- in what is recommended at any given site. That should be driven by the microbiological data that exists for that specific unit. So you cannot take a policy at one unit and apply it to another unit. It has to be tailored for a given site, for the

reasons we've just talked about: because the microbiological environment in any given institution may be very different from another.

Q So can you, in a sense, summarise the position taken by you and Professor Hawkey on this topic as it applies to the data for the BSI? So what did you think might be happening and why?

A I have to answer this in two ways. One way is to-- is to leave Professor Hawkey to address the specific question which he has raised and really written in great detail about in our report relating to the impact of the use a drug known as meropenem on increasing rates specifically of-- potentially of Stenotrophomonas infections-- or Stenotrophomonas maltophilia infections.

Q Yes.

A So that's a very specific issue. In broader terms, for me it's very clear that the antibiotic policies, not specific to- to the practice in the Glasgow hospitals, but something that's very widespread in haemato-oncology units throughout the United Kingdom, is the use of an awful lot of antibiotics in terms of trying to prevent infection-- So in other prophylaxis, and I believe the units in Glasgow

were using a drug called ciprofloxacin----

Q Why do you believe that? How did you find that out?

A It's in-- it's in the bundles. I forget-- I'd have to look at my notes.

Q But did you know that when you wrote the report?

A I knew from work I had done previously of febrile neutropenia policies in the adult service.

Q In Glasgow?

A In Glasgow, yes.

Q You don't refer to this in your report, Dr Agrawal. Why do you not refer to this in the original report, if you knew it at the time?

A It wasn't-- Should I have referred to it? I tried to explain to you----

Q What's worrying me – and I'll jump to the point – is this, is that I get that Professor Hawkey is looking at meropenem----

A Yes.

Q -- and we'll deal with chapter 4 with him on Wednesday, but a critique that can be made of him is that he hasn't considered various pieces of information which don't appear to have been made available to him. I mean, it doesn't say he couldn't have asked, but they don't appear to have been made available to him. The meropenem issue arose in this Inquiry probably most prominently when Professor Leanord gave evidence last year, and he looked at a paper produced by Ms Harvey-Wood and Dr Peters and one of the pharmacists, who was called Dr Gourlay I think, in October 2018, and he looked at a chart and thought, "Well, maybe there's something going on here."

So he gave evidence about that, various people responded, and we now have seen, and we've checked this morning, that in the unit in 2018, they gave consideration to whether there was a problem of antibiotic resistance (inaudible) meropenem, and for reasons I'll put to Professor Hawkey on Wednesday, they concluded that there wasn't.

Now, I suppose the question for him on Wednesday is, "How can you give an opinion on what might have happened without knowing what actually happened?" So in this case of this particular antibiotic, what knowledge do you have about what its use was, and where does it come from?

A So, I'm not sure I understood the question.

Q So you've mentioned a new antibiotic and how it was used.

THE CHAIR: Well----

A Oh, this is in the context of-- So what-- what I'm trying to----

THE CHAIR: Well----

A Sorry, yes.

THE CHAIR: -- can I just see if I'm following?

A Yes, sure, of course.

THE CHAIR: You were asked a question by Mr Mackintosh about-- and it appeared to me that you were making clear that you were answering it at two levels.

A Yes.

THE CHAIR: First of all, you mentioned meropenem, and I took it that you were at that point saying, "Well, that's really a matter for Professor Hawkey," but you wanted to make a broader point----

A Yes.

THE CHAIR: -- and you used the example, again if I was following you, of the use of ciprofloxacin as a prophylactic.

A Yes.

THE CHAIR: Now, I don't want to discourage you from answering Mr Mackintosh's questions, but if you were making a broader point which effectively we have cut off----

MR MACKINTOSH: Fair point, my Lord.

THE CHAIR: -- I would wish you to have the opportunity to make your broader point.

A Thank you.

MR MACKINTOSH: So, what's the point you're making about ciprofloxacin?

THE CHAIR: I just hope I'm following where we are.

MR MACKINTOSH: What's the point you're making about ciprofloxacin?

A No, you are. You are. Thank you. The point I'm trying to make is, in order to understand the issues, as far as I see them from my perspective, of what's happened in terms of infection, be it contaminated water, be it a failure of ventilation system-- Notwithstanding any of that, there's a lack of clinical perspective as far as I can tell from the information that I've seen, and that clinical perspective is the actual management of patients. It may be the Inquiry has addressed all of this. I'm not aware of that. A huge impact comes from the use of antibiotics in general, it's not just meropenem. It's how antibiotics are used. It's when they're prescribed. It's how they're changed, and it's the management of patients in terms of, effectively fever-driven antibiotic use, which is how we practice clinically.

If that is not looked at, if that's not-- if that's not taken into account,

then it's impossible to make any assessment of what that might be doing to the microbiological environment of the unit. I emphasise that because we have spent a lot of time in the work we do at St Bartholomew's Hospital to try and address those issues, and what we've found is that-- We were talking about antimicrobial resistance a second ago. It is possible to reduce antimicrobial resistance by addressing these issues. Simply put, if you can use less antibiotics, you can change the antimicrobial-- sorry, the microbial environment of the hospital or your unit, and that changes what happens in the patients and the infections they get.

Q Yes, I appreciate that, and I don't think anyone's contradicting that. I don't think anyone's said otherwise. The problem I am facing as counsel to the Inquiry is how to deal with this evidentially. So, meropenem made its arrival on our consciousness in the evidence of Professor Leanord. To be fair, we should probably have seen it in the CNR overview report, because it is discussed there. We had evidence about it last year and there's bits and pieces coming out over the next few weeks, and Professor Hawkey will be very interesting about

that topic.

The problem I'm having, and the reason why I slightly jumped down your throat there – for which I apologise – is, if we think about ciprofloxacin, if I pronounced it correctly, it's not mentioned as a possibility in your HAD Report, it's not mentioned in your rebuttal document, it's not mentioned anywhere. I mean, all I've done is a keyword search in the two bundles, so I may be wrong and no doubt some will correct me over lunchtime, but if that's the case, how do I deal with that? Because is it your position that we should be looking out to see whether there was an excess use of ciprofloxacin and therefore how did the ciprofloxacin resistant – would it be the right thing – organisms behave? Or what would we see in the signal if this was a problem?

A I don't think I'm actually saying that. I mean, I think that the points you make are all relevant, but what I am saying of things that are in our document is that an aspect of-- of what's going on – Is it the water, is it ventilation, is it line care – is also the clinical behaviour, and that's not just the clinical behaviour of teams, it's also the clinical behaviour of policies----

Q But that's not

controversial. My question is this, is that, why do you think that's not already been considered?

A Because I'm not aware of that information. I'm not-- I did say earlier that this may be something the Inquiry has already addressed, but----

Q No, no, the reason I raise this is because----

A -- I'm not aware of that.

Q The reason I'm raising it and I'm pushing back on you is this, is that, your report raises issues, and we have spent some time attempting to understand those issues, put them to the Case Notes Review authors, put them to our own experts, the core participants, which include the Health Board, the National Services Agency, two groups of families, and a group of doctors. I've proposed questions to you and we've had a sort of exchange about this, and to an extent I'm therefore ready to have a conversation with Professor Hawkey about meropenem. Whether I'll do it well is an entirely debatable point, but we've done some work. But I'm not sure it helps to float possibilities other than just to say, "Have you checked for this?" when you actually don't know what happened at the time, because you haven't read any of the contemporary investigations other than the Case Notes Review. So how are you helping us by bringing this in at this point? A slightly grumpy comment from me. But do you see my concern that I'm trying-- Now I'm going to have to investigate this as an issue, which wasn't previously on my radar. You could have mentioned it in the report.

A I accept that. I mean, the simple answer to that is the report does mention the importance of, when looking at preventing infection, that it's a very-- And I know the Inquiry has heard a lot about infection prevention. It's a very broad area, including clinical management, clinical behaviour. It's not just infection control policies; it's not just making sure the water is clean and the air is being handled as it should. I think that's the point I'm trying to make.

Q Okay. Well, what I'd like to do is to put a document to you which has confused me, and I might get your help. So it's in 44, volume 1. It's document 6 and 7. I think we're going to 7 first. That's page 248.

Now, this appears to be a paper which which we acquired in a particular way, and I'll tell you how we acquired it, but it bears to be written by Seán MacBride-Stewart, dated from the end of January of last year. It's titled "Public Inquiry Analysis of

Antimicrobial Prescribing." We didn't instruct it. It appeared in the document, the data that was supplied to us by the Central Legal Office on behalf of GGC when you transferred, as it were, under our wing in March, and colloquially we refer to this whole thing as the data dump. It contains a lot of spreadsheets and, for example, all your imaging, when you did the Aspergillus project, all in there.

A Oh, I see. Okay.

Q But this is sitting in there along with document 6, which is at page 246, which is an email. We'll take that off the screen. Now, the point is that that document isn't mentioned in the HAD Report. We've not been given it by the Central Legal Office. Had you seen it?

A No.

Q No.

A No.

Q Okay. Well, that makes it much easier. We can just put it to one side.

A No, I've not seen that.

Q So, what I want to just check, and I think you've already answered this question, which is that whilst you are raising these possibilities, from your perspective you're not raising them because you have gone and looked at what actually happened other than to say, "When you look at this data, remember antimicrobial resistance as a factor and consider it." Is that effectively your position?

A Yes, just one very slight caveat.

Q Please.

A It's not antimicrobial resistance. It's antimicrobial use.

Q Thank you.

A Resistance may follow, but----

Q It's the use policies.

A Yes.

Q Right. It's the cause and effect.

A Yes.

Q Okay. What I'm proposing to do now is to look at what might be termed as the water chapter, but not so much what's in the water chapter, because Professor Hawkey wrote that, but more the bloodstream infection works in chapter 7 and what we were testing, and in fact I can't do that because I need to let you think about Ward 4C in order to do that. I think it's tied up in that issue, so we'll pass over that for the moment, and what we'll do is we'll talk about clusters.

So if we go to the HAD Report, 44, volume 1, which is section 7.2.2,

which I think starts on page 67, there's a discussion of the clustering exercise and the logic behind it. And then if you go on to the next page, page 68 and table 3, there is the criteria listed, and I'm showing you that just as a sort of aide-mémoire. Did you have any involvement in this clustering exercise?

A No.

Q No?

A No.

Q Well, that makes life a lot simpler.

A Yes.

Q So that would be Professor Hawkey I talk to about that.

A This would be Professor Hawkey and Dr Drumright.

Q Yes. Her position is that she did the maths on this but the idea is Professor Hawkey, and her second attempt is all her, and I discussed that with her.

A Yes.

Q Right. What I want to just do is to deal with the broad-brush of the criticism of the whole of the CNR from your perspective, and so if we go to the introduction to the report, which starts on page 16-- And I'm assuming you've read this and you agree with the contents.

A Yes.

Q Yes. So if we can go to the end-- Well, actually, let's get to the bottom of page 16. You say:

> "A comparative exercise is essential, as before it can be said that there is an increased (i.e. abnormal) risk of infection it must be first understood what a normal level of risk is. If an increase of infection rates is observed, this does not necessarily mean that it is attributable to the built environment, as the causes of infection are multifactorial. Further investigation would be required to establish if the built environment was linked to any detected infections."

What would be the nature of that further investigation?

A Sorry, excuse me.

Q Of course. So that's the second line of page 17:

> "Further investigation would be required to establish if the built environment was linked to any detected infections."

What would be the nature of such an investigation?

A Well, if the built environment was suspected, then that would be----

Q It seems to flow on from

"If an increase in infection rates is observed." You see at the top of this page?

A Yes, yes. So that would be looking at all the factors that we've been discussing, so is there any evidence of increased infection in water?

Q In the water samples, sort of thing?

A Yes, water sampling. Yes, of course. We also need to look at the line care. In fact, I would say the whole range of things we've talked about.

Q I recognise this is a hypothetical at this point, if an increase beyond what is defined as normal rate is observed in the data, is your position that you were then required to have further investigations?

A In terms of trying to attribute what's the cause of the increase.

Q Yes. Is this where we get into the conversation that I hope you overheard with Dr Drumright, where we look at scenarios and counterfactuals and we consider them all?

A Yes.

Q Yes. And would you broadly ascribe to her approach that you've got five or six different things to consider, and we discussed what they might be?

A Well, I would suggest it's a reality, yes.

Q And then:

> "However, if there is no increase in infection rates then it can be included there is no increased level of risk from the built environment or other potential factors."

And then the final paragraph.

A I actually don't----

THE CHAIR: Can I just----

A Oh, sorry. Please do.

THE CHAIR: With apologies, does that actually follow?

A Thank you, your Lordship. It was just about comments on that myself, but I'm not sure----

THE CHAIR: Please comment.

A -- having just heard it read back to me that I would actually agree with that last bit. So it may be that there's no detectable increase in infection rates. That would not mean that the environment was not putting people at increased risk.

MR MACKINTOSH: Yes, because one of the problems with-- I mean, I recognise I'm about to talk about water and it's not your thing, but are you familiar with the idea that

water systems are managed in a way that focuses on risk?

A Mm.

Q And you manage the risk by a process of managing the water through testing and controls and -

A Absolutely, yes.

Q And in your hospital and your unit, do you manage the ventilation system in a similar way? You focus on risk, rather than on----

A Yes.

Q So would you, for example, have your ventilation system validated every so often by engineers?

A Yeah, absolutely. The pressure gradients, etc., changes.

Q Since you've been at Barts, has Barts had any new ventilation systems fitted, so new bits of the ward or retrofits or anything like that?

A Absolutely. Yes, we have.

Q Would you countenance opening a ward without validating the ventilation system first?

A No.

Q Why?

A Because you would like to know that the new hospital is working to the level that's expected from the specifications, and the reason for that, of course, is because we

believe that's helping protect patients from harm.

Q And so how does that desire to have a validation exercise connect to the risk that the patients are exposed to? I mean, can I go as far as saying that an unvalidated ventilation system creates a risk, or is that linking two concepts that shouldn't be linked?

A I think the dilemma here for me is that we have a new hospital, or a change, a ventilation system that's been commissioned, been designed, and it should meet its design specifications. If there was a question of, "Well, can we open-- can we put patients in this space?" then without validating it, that would seem demonstrably unsafe. However, you would need to know something about the performance of the ventilation system, but that would not necessarily mean you were putting patients at harm.

Q Yes, because you've used three words that I think even at six minutes to one I should probably explore with you. So you've used the word "risk," you've used the word "unsafe" and "putting patients at harm," which I recognise is not a word, but it's a concept. How do you see those three concepts relating to each other?

A All three are theoretical

because, as has been discussed at some length within our various reports, firstly there's very little evidence that the ventilation systems currently use-- There's a specification in SHTM-0301, the Scottish----

Q Which is very similar to HTM-0301.

A Yeah, absolutely. I think they're virtually identical, but the English version is there's virtually no evidence this has any clinical impact on----

Q Because there were some studies done 30 years ago.

A Yes, and in fact clinical practice has moved on so far that the same patients we've thought needed that sort of protection before in some cases are being managed outside of the hospital entirely or partially. So the concept of the ventilation sounds perfectly reasonable, but the data doesn't support that it actually makes a difference, and hence perhaps why so many guidelines do not specify that these ventilation systems are essential for managing patients----

Q So how does that connect to what I think you just said, that putting patients into a unit that had been unvalidated would be unsafe?

A I guess from what my-- It doesn't feel comfortable as a clinician not knowing that the hospital or the ward we're about to use has not had some checks done on it. You would want to know its cleanliness levels. You would want to know that the air handling unit is filtering to the level it should be filtering, because one of the problems with these systems is not just, "Are they performing to specification?" but if there's a problem, are they actually leading to infection? For example----

Q Because they might be blowing the wrong way.

A Exactly. For example, yes. So that's why I say it would be unsafe to attempt to open a new ventilation system unvalidated. But then when you step back from that, why would you do that? That doesn't make any sense. When you have your fully validated ventilation system and it's working – perhaps this is a more constructive way of looking at it – does that mean the individual is not going to get infection from airborne pathogens? No, it doesn't.

Q So, one of the----

THE CHAIR: Maybe we want to explore this further after lunch, but----

MR MACKINTOSH: Well, there's a couple of bits I just want to pick up before we-- You talked about harm as well. It's the third concept. I

think we need to unpick what you just said, and I need to think about it quietly over a cup of tea. But in terms of harm, if it's your position that there's no research base for the idea that a non-compliant ventilation system causes infections, which I think that's what you just said, how is it causing----

THE CHAIR: Right, when you----

MR MACKINTOSH: I think I need to think about this more, but----

THE CHAIR: I mean, do you mean causes infection or reduces the available protection against infection?

MR MACKINTOSH: Yes. Let's not do that before lunch. Let's just do one final thought. I'm going to give you a thought and I'll come back to this after lunch. So we had evidence in the Edinburgh part of this inquiry from a Professor Humphreys, and he was dealing with this quandary of the lack of research information. In addition to observing you can't actually do this research because it probably wouldn't be ethical, I think he used an analogy about an old car, the sort of cars that we had in the '70s or '80s, in that as cars acquire safety features, or indeed you take them away, there isn't an obvious point when they become terribly dangerous. But as you take safety features off, you must increase the risk.

Now, I always worry I misquote Professor Humphreys because that's a terrible summary, but to what extent are we dealing with an issue like that, which once you've got a process where you now have modern cars with anti-lock brakes and seatbelts and all these things, and airbags, taking them away feels dangerous? Now, I recognise there is an evidential basis for all those features, but how would you react to the idea that having a unit that doesn't have the features it could have had is either suboptimal or gives rise to risk or is unsafe? Where would you stand on those three words being applied to that scenario? Not building a unit with the standard fit-out.

A I think I come back to what I was trying to say earlier. I obviously didn't manage it very well. So there is a regulatory-- Is that even the right word? There is exactly the sense that you said earlier, that it feels like you're going backwards. It feels like you're going in the wrong direction, and if you're going in the wrong direction, then you must be potentially putting patients at risk of what you were trying to prevent them from being at risk of. However, for all the reasons listed in the report, that approach fails even when you have a fully compliant system based on our current

regulations, and it fails for many different reasons. So your car with all its gizmos still can have an accident. You're not going to eliminate risk, and the nature of the risk, and also the nature of the ventilation systems we have, means that you are not achieving what you think you're achieving.

I think it may have been alluded to yesterday by Dr Drumright, I'm not sure, that even what we think we know about ventilation systems actually is not supported. There's some, you know, very limited amounts of, are they performing even to the levels that we think they are with the specifications required? And hence clinical practice is actually sort of moving on from the need for that, but it's still there. So we all have-- I say "we all have"; I think all bone marrow transplant units in the UK performing allogeneic stem cell transplants-- Just a little caveat that there are different forms of transplant which absolutely do not need that environment, but standard practice still is in the UK that those sort of transplants in general are performed in a compliant unit, despite the fact the evidence isn't there that it prevents infection.

Q I think that's probably a good place to break, unless anything occurs to my Lord at this precise point.

THE CHAIR: Can I just take the opportunity of making a general observation for your comment. Now, I think you agree with me that the sentence we looked at beginning with "however" actually does not follow. In other words, simply because there is no increase in infection does not mean there's absence of increase in risks. Would it be fair to say that if one looks at your report, the approach is to look not strictly speaking at risk, but what I'm going to call eventuation of risk. In other words, the occurrence of the event which one is trying to avoid.

A I think that's exactly what our report is looking at, yes.

THE CHAIR: Right.

A Yes.

THE CHAIR: So even if we find the word "risk," we've got to understand that the approach of the report is to say, well, has there been an outcome which is supposed to be avoided?

A Yes.

THE CHAIR: Right. Well, I suspect we may come back to some of the things we've touched on this morning at two o'clock.

THE WITNESS: Thank you.

THE CHAIR: I hope you have the opportunity for lunch, Dr Agrawal.

THE WITNESS: Thank you.

(Adjourned for a short time)

THE WITNESS: Good afternoon.

THE CHAIR: Good afternoon, Dr Agrawal.

THE WITNESS: I just brought some notes in that Mr Mackintosh asked me to look at.

MR MACKINTOSH: Yes. So I wanted to check about this 4B/4C issue.

THE CHAIR: Mm-hmm.

MR MACKINTOSH: Now, we'll put the notes to one side for a moment because you looked at them but we want to just see-- are these your own notes prepared in preparation for this?

A So these are copies of the documents you mentioned earlier.

Q Right, fantastic. Well, so----

A There's one sheet which has my notes relating to that.

Q Right. So let's just start back at where we left off, which was which wall is which in Adult land.

A Yes.

Q So just to nail things down we have 4C and 4B.

A B.

Q And your report from 21 was about 4C?

A Indeed it was.

Q So what was the patient population you're correlating for C?

A Thank you, Mr Mackintosh. Yes, it's a little annoying when you don't recall the things you've written yourself, so my apologies for that, but 4C which is the ward I was writing about and issues of ventilation is a haemato-oncology ward and a renal ward, or it certainly was at that time, looking after all adults with various haematological diseases having treatment except patients undergoing bone marrow transplant procedures and they were on-- still are, I believe, on 4B.

Q So would that be, in effect, a different approach from your unit where they're all on the same unit?

A No, effectively, it's identical and we have two wards, 5A and 5B. I mean, in our setup, they're physically next to each other. We call 5B the bone marrow transplant unit but, in reality, it's slightly interchangeable.

Q So, well, let's actually talk about ventilation and your knowledge of that.

A Yes.

Q Just going to take a little note. Yes, the screens haven't come on yet so I wonder if the feed is working. I wonder if that can just be checked. Normally we see us. Yes, that's helpful. So thank you, Ms Crawford. So let's just orientate ourselves.

A Excuse me.

Q Approximately which year were you instructed to start work on the 4C report?

A Approximately 2020, possibly 2021, since the report data was 2021, yes.

Q That's helpful. So at that point, what did you understand to be the ventilation standards that were fitted to Ward 4C?

A Well, the requirements were for an SHTM compliant system, so I suppose----

Q So what would that have been?

A So that's basically positive pressure, 10 kilopascals, and 10 air changes.

Q And would that not include the HEPA filtration?

A Sorry, and HEPA filtration, yes.

Q What did you understand was actually in place in that ward?

A It was HEPA filtered system, air changes, did not meet the 10 requirements. I think they were----

Q Let's look at your report because I'm not convinced-- I'm worried you might be, as it were, misremembering. So your report is in bundle 44, volume 5, starting from page 102-- 104 we'll go to. Just looking at it, looking at the executive summary, that doesn't mention what the ventilation standards are-- in page 105, it doesn't mention what the ventilation standards are in the ward at the time.

THE CHAIR: Where you used the word "standards," you mean specification?

MR MACKINTOSH: Specification. If we go to page 106 and over the page at 207, can you help us about whether this ward had HEPA filtration as far as you understood it?

A Yes, actually, I think I may be wrong there. I haven't mentioned HEPA filtration here, yes.

Q Because could it be that the ward, in fact, had no HEPA filtration, 2½ to 3 air changes per hour----

A Yes.

Q -- and no pressure differential as per the rest of the hospital?

A Yes.

Q And possibly a small differential between the rooms and the corridor?

A Yes.

Q Right, and so your report is in respect of that ward?

A Yes.

Q And the patient cohort you just described?

A That's correct.

Q Right, let's go and look at 4B. So the point you were instructed, did you know what the ventilation requirement states as 4B was when you were instructed to do 4C?

A Not that I recall, no.

Q Do you know----

A Specifically related to 4C----

THE CHAIR: Again, you've used the word "requirements." Is that the word you mean to use?

MR MACKINTOSH: My Lord, you're right to pull me up. In 4B, at the point you were instructed, did you know what the ventilation was actually providing in 4B?

A I was not instructed to look at 4B.

Q I appreciate that, but I'm just checking whether you knew.

A Oh, I see. I don't recall that being something I knew about since it wasn't part of my instructions.

Q Understandable.

A I don't think so. I don't think so.

Q Right. So let's just step back from this, and from the point of view of writing this report, the HAD Report, you're aware that we've looked at text which describes how the patients in 4C arrived in the hospital with everyone else in the summer of 2015 and then stayed, that cohort. So do you know whether the ventilation system was unchanged between 2015 and when you started writing your report on 4C or whether it changed?

A Remedial work had happened, if I recall.

Q But it was still 2½ air changes, no HEPA filtration----

A But they hadn't managed to get it up to specification.

Q Right.

A That was explicitly stated.

Q Yes, so it's consistent, is what I think we're looking at.

A Yes.

Q Right.

A Yes.

Q So we look at 4B, the Adult BMT Unit. Now, your report describes how those patients were in the hospital for five/six weeks in the

summer of 2015. At that point-- The BMT patients. Do you know the ventilation that was actually fitted in Ward 4B at that point?

A I don't think I do, no. I can't recall that information.

Q Right. When they return from the Beatson, the North in June 2018, do you know what the ventilation system that was in place that met them, effectively?

A Yes.

Q What was it?

A It was a HEPA filtered system with approximately six changes per hour.

Q Right.

A Sorry, six changes per minute and a pressure gradient that was still not meeting 10 kilopascals.

Q And there was an airlock door at the entrance to the ward?

A Yes.

Q Yes, right. Do you know anything about the ventilation that was in place in the ward they'd been in at the Beatson, the BMT patients?

A I was not provided with any information about that. I only know from documentation elsewhere that the Beatson air handling unit performed to a higher standard. That's what I've read but I've not seen any documentation around it, yes.

Q Right, and so when we think about the design of this study, you look at North, South and Adult BMT in three categories. So let's look at the ventilation for all three. What knowledge do you have about the ventilation that the North haematology patients were accommodated in?

A I don't.

Q You don't.

A I don't.

Q Are you effectively making the assumption that it is in compliance with the standard that Ward 4C was not in compliance with?

A No, I'm making the assumption it was normal ventilation in a hospital ward, but I don't know that.

Q All right. What do you mean by normal ventilation in the hospital ward?

A Well, assuming that, since I don't know the structure of the wards in the North Sector – or the South Sector for that matter – and they were not handling bone marrow transplant patients, so my assumption was they were not-- they would not be put in HEPA-filtered rooms. There wouldn't-- there wouldn't be a requirement for that, I think. So I assume that wasn't the case, and I would also assume the case they were not in positive pressure rooms.

Q Why would there not be a requirement for the North Sector equivalent? If by a requirement you mean SHTM 03-01?

A Yes, yes.

Q Why would there not be a requirement on the North Sector patients to be in, as it were, the standard that 4C didn't meet?

A Well, as I was referring to earlier, many patients are not managed in an SHTM 03-01 compliant, so----

Q No, I understand that.

A My assumption had been they weren't.

Q So, if I understand correctly, the point you asked to address in this report, the 2921 report, was, does it matter that the patients in 4C are in a non-SHTM 03-01 compliant environment?

A Yes.

Q Is that broadly fair----

A Yes, yes.

Q Right. Did you make an assumption that the patients in North Sector were in an SHTM 03-01 compliant environment?

A Okay. Sorry, Mr Mackintosh, I just need to clarify, because we're talking about two different reports: the one submitted to this Inquiry and----

Q Well, you're right to do that----

A -- and the one from 2021.

Q Well, I'll break it down then so it's easier----

A Fine----

Q When you did the 2021 report, the question you were asked, in essence, was, "Is there an issue arising from the fact that Ward 4C is not in compliance with SHTM 03-01?" That's correct?

A Yes.

Q Putting that to one side and now dealing suddenly with a new report----

A Right, yes.

Q -- what was the purpose of looking at the Aspergillus or bloodstream infection rates in these three cohorts of patients? What were you trying to explore?

A We were exploring what were the incidence rates of bloodstream infections and infections attributable to Aspergillus in the different sectors covered by the Greater Glasgow Health Board.

Q I know you didn't find this, but let's imagine that in the case of the Aspergillus work you had found an above normal incidence in either Ward 4C or in Ward 4B when it was at the Queen Elizabeth. What I'm putting

to you is, would you have needed to know what the other parts of the study were exposed to in order to work out whether that was related to the ventilation?

A Well, I would-- If I may just phrase your question----

Q Please.

A -- slightly differently, because it comes back to the design of what we thought-- how we wanted to try and answer the question.

Q Right.

A So we wanted to answer the question by looking at observed rates.

Q Yes.

A If we observed rates that indicated there was an increased rate at Queen Elizabeth University Hospital versus the other sectors, then we would have surmised that there was potentially an issue as compared to the other sectors.

Q Why?

A Because the other sectors-- And this is contemporaneous, so the other sectors were treating patients with haematological malignancy, and they were-- It's not just a bone marrow transplant individual that's at high risk, it's patients with acute-- in particular, patients with acute leukaemia undergoing treatments for acute leukaemia. They're at high risk----

Q Where would you have expected them to be? In which ward?

A Well, in the Queen Elizabeth University Hospital, they would be in 4C or 6A, if-- my understanding.

Q Would the 6A ones have been children?

A Well, I think the-- the children moved temporarily to the adult wards, didn't they? So----

Q Right. So could it be that 6A accommodated, before the children moved, to care of the elderly ward and has never accommodated haematology patients? Is that possible?

A I don't know. I'm not aware.

Q Right. So the point I'm putting to you about the design of the study in respect to ventilation is that, the patients in the Bone Marrow Treatment Ward were first in the Beatson, then they were briefly in a non-HEPA-filtered ward in the Queen Elizabeth, and they were back in the Beatson, and then they were in a HEPA-filtered ward in the Queen Elizabeth, albeit with not right air change rate. Why would you expect to see any particular signal in that data if

they had HEPA filtration effectively for the whole period apart from the five weeks in summer of 2015? Why would you see anything? Why did you look?

A Well, I think if I've understood your question correctly, then there's an assumption in there that if you have broadly similar construction, broadly similar specifications – in fact inferior in the Queen Elizabeth's University Hospital – then you would not necessarily see any differences in infection rates, which is a logical assumption. But that's exactly what we were testing.

Q So yours was just an open exploration of whether we would see anything?

A Absolutely, yes.

Q So the fact that you have effectively tested a group of patients who, when in the Queen Elizabeth, apart from a very brief period, had HEPA filtration, doesn't prevent your broad exploration being (w)?

A No.

Q No? Okay. Let's just touch on the water for the moment, because we're doing geography, so we might as well do it all in one place. In the BSI study, which of the patients, the adult patients, that you looked at at the Queen Elizabeth, were exposed to

unfiltered water in the Queen Elizabeth?

A I don't know the answer to that. We-- we did not attempt to answer that question.

Q It's just that, if we understand the evidence correctly, apart from the five weeks in the summer of 2015, none of the Adult BMT patients were ever exposed in their own ward – maybe when they went elsewhere in the hospital it was different, but in their own ward – to unfiltered water. How can you draw conclusions from the absence of a signal in the Adult BMT patients about the water if you don't know they weren't exposed to the water?

A Can I go back to the letter of instruction----

Q Yes, of course.

A -- for a second, please?

Q So that's bundle 44, volume 1----

A I haven't brought that in with me, so I----

Q No, no, it's fine. We've got it on the screen.

A Thank you.

Q It's on page 234. Would you like the appendix?

A No, I-- No, because it-- Was it-- If we go to the next page, please.

Q Yes, of course. Next page, 235.

A Part of the information we were given-- Sorry, do you mind going back a page?

Q Yes, 234?

A I-- Yes, I think it's actually in the bottom-- The very last sentence on that page, it-- it-- We're told here that:

> "It should be noted that the QEUH and RHC have a shared water system, but that individual wards have differing ventilation provision."

Hence we were certainly under the impression that it was the same water, or more or less the same water, being provided to different parts of the hospital.

Q Because, if we go through to page 237, had you read the document in the footnote, the HPS summary----

A Yes.

Q -- you would have learned that at points during 2018, various interventions were taken, including fitting points of use filters, and whilst there is definitely a debate about whether they were fully effective, that's an intervention which changes the nature of the water that patients

are in contact with. We also know – which isn't in that report, to be fair – that later on, certainly by 2020, the Health Board become confident and increasingly confident that they've managed whatever's in the water. Whether contamination is the right word or microbial proliferation, whatever the word is, they've managed it. So there's a temporal change in the water experience, but you weren't aware of that.

A Can we look at the next page, please----

Q Yes, of course.

A -- for a second?

Q Over the page to 238.

A And the next page? Because my-- I think we're somewhere else now, aren't we?

Q We're on Professor Hawkey's letter.

A My recollection of it is the letter did-- did say that interventions had happened, but I cannot see it.

Q I think it does, actually. It might well be on 235.

A (After a pause) Let me just look at this.

Q So you'll see, at the top of the page – and this is about ventilation, but for completeness – there's a reference at the end of that first paragraph to remedial works to

Ward 4B.

A Yes, and in the next paragraph----

Q Then there's mitigation measures at the bottom half of the second paragraph.

A Yes, we're told that the point of use filters were used and subsequently installation of a chlorine dioxide system to dose the water with dates there.

Q Yes, so----

A So that information we did have.

Q Yes. So whilst you don't have the data of the point of use filters in there, should I be in any way concerned that you're looking to see if there is an increased incidence of infections in, in the case of the adult BMT patients, patients who aren't exposed to unfiltered water apart from five weeks in 2015?

A Just to clarify, is your point, Mr Mackintosh, that the BMT Unit pateients-- the Adult BMT Unit patients, were in an environment without filters?

Q No.

A Is that----

THE CHAIR: It may be quite the opposite.

MR MACKINTOSH: It's the exact opposite. I think what I should do is take you to the chart which I discussed with Dr Drumright yesterday. It's the same bundle, it's page 93. We'll just look at the top half of the page. So this is, "Incidence of environmentally relevant bacteraemia within BMT patients only over time" and the blue line is at the Queen Elizabeth, and the red line is at North. Now, ignoring for a moment the five weeks in 2015, which I'm not sure makes it into this data table, the position as we understand it is that, for the whole period in the blue line, those patients had all their water sources in their ward filtered at point of use, and that once you get past late '18, as described in that letter, there's now a chlorine dioxide dosing system. So what I'm concerned about is that in the text that relates to this chart, you say on the previous page at 7.2.6:

> "We were interested in examining single patient populations as services transitioned from another hospital ... to determine if there was an increase in number of cases of environmentally relevant bacteraemias, which could be indicative of environmental sources of infection."

Now, why would transfer of

patients into a ward which had filtered water and a system which was about to become-- have chlorine dioxide, have any prospect of having an increase in environmentally relevant bacteria? What's the mechanism that would be involved in that?

A I go back to the original approach we took. So you're asking me, "Why did we not-- Why weren't we aware of these things?" which I think we've been over several times now.

Q Yes, we have.

A And our approach was, "Well, we will look at the data and see if that says anything," and then the question of, "Well, if it shows something, what does it mean?" was something then we'd-- we'd come back to.

Q Because at the end of this paragraph on page 92, you make an observation that the data – that's the chart we looked at – suggests:

> "... the incidence of bacteraemia attributable to environmentally relevant organisms may be occurring at lower rates at QEUH than the norm for the [Greater Glasgow] area, although these findings should be interpreted in light of other factors that could influence incidence over time."

The other factor being that they're now in a ward with a filtration system and, if I understand other witnesses' evidence correctly, the largest amount of water testing anywhere in the United Kingdom going on. But you didn't know that?

A No.

Q No? Okay.

A No.

Q What I want to do then is to return, if I might, to your report from 2021, because whilst I appreciate it was produced in a different context for a different purpose-- So that's in volume 5, and if we go to page 105, please. The questions I have relate to the sources of infection you describe on page 109. 109, please. There we are. So, 4.1.3, "Sources of infectious pathogens," you list them. There are three here: patients themselves, airborne pathogens, transmission of pathogens from visitors. I just want to understand here whether that's a complete list, whether there would be other forms of ventilation-related sources of infection, particularly waterborne material that is in the air because there's been spray or shower or something like that or fluid-generating procedures.

A Yes, so that was raised in various comments to our-- to our report, in fact.

Q Yes.

A Obviously I'm not sure this-- In fact, this was seen by some of the other----

Q It was, yes.

A -- expert witnesses to the Inquiry, and the point was made that, well, you can-- There's generation of aerosols, so in other words airborne particles, from taps, sinks, showers, and hence if you have a ventilation system-- or you don't have a ventilation system, but if you have a ventilation system that's not providing the air changes that it should be providing based on specification, then that would lead to a material increase in-- Well, I-- that's how I read the criticisms-- a material increased risk of infection of those non-normally airborne pathogens.

Q Yes, the concept of dilution seemed to be talked about.

A Yes. So in the comments made, there was no description. I seem to recall it was Drs Mumford and Mookerjee's comment----

Q Yes, it's their commentary.

A Yes, so they didn't specify which non-airborne pathogens they felt were most involved by this, because it was unclear to me which pathogens they meant, and they were referring to a previous report by, I believe-- I hope I've got this right, Dr Dempster.

Q Well, it's a joint report between Dr Mumford and Ms Dempster----

A Oh, sorry.

Q -- who was, I think, lead HI and Infection Control Nurse for NHS England.

A Thank you. But I did go and look at what was said in that report, and the implication of that report was all environmental organisms would be regarded as being subject to this problem of not being diluted, as the concern is, by not having enough air changes. Then-- So in order to try and consider the point being made, I looked at some of the references provided and also references provided in our documents around infection control issues relating to waterborne, let's say, outbreaks. I couldn't find any evidence that any of those outbreaks had been resolved by addressing ventilation.

So I was-- And these weren't referenced, these statements, so I was slightly struggling to see what the basis of this was, and there's actually

a statement in-- in those criticisms that they were less concerned about airborne pathogens and more concerned about non-airborne pathogens becoming an-- an increased risk to individuals because of the rate change not being as desired.

Q So that it doesn't dilute the air?

A Yes.

Q Right.

A And I wasn't able to really see any basis for that, and I certainly didn't find references to support such statements, which I think would be required in order to be able to comment on it at all.

THE CHAIR: Can I maybe just take a step backwards? Looking at page 109, which I appreciate is your 2021 report as opposed to the more recent report, you're making entirely general points at 4.1.3.

A Yes. Yes.

THE CHAIR: And airborne pathogens include viral, so that's viruses, fungal agents, fungal spores, most notably Aspergillus. Now, my question is do airborne pathogens, just speaking generally, include, first step, bacteria? Airborne?

A Well, certainly tuberculosis is the most obvious agent that comes to mind in terms of being of airborne concern, so yes.

THE CHAIR: Right. So as a matter of generality, the air is a vector for at least some bacterial infections.

A Yes. Yes.

THE CHAIR: Now, thinking about water which is in aerosol form-- My apologies for not knowing what the verb is. But water in aerosol form, is that a potential vector for bacterial infection?

A Yes, I would agree it's a potential, which is slightly different from saying, "But what is that potential?" I would have difficulty quantifying that.

THE CHAIR: Right. Now, would you go the distance of saying ventilation has no-- Specialised ventilation, increased air change, pressure having the effect of excluding one area from another and filtration preventing penetration of the air into the patient's bedroom. Do you say that has no role in reducing such risk as is presented by airborne aerosols?

A Of organisms that would otherwise not be airborne, sorry, just to clarify?

THE CHAIR: Mm-hmm.

A Yes. Yes, I do say that. I can see no evidence that that is the case from the references provided.

Even those references referring to outbreaks relating to such aerosols being generated from taps, for example, and shower drains, they were resolved without any recourse to ventilation.

THE CHAIR: Well, leave aside at the moment the particular article references. If, as you accept, potentially pathogenic bacteria may be carried within water in aerosol form and therefore in the air, is there any reason in principle why ventilation might not reduce the risk presented by such airborne microorganisms?

A No, I cannot say-- No, in principle it could reduce the risk. There are a variety of reasons why I think that probably isn't the case.

THE CHAIR: Do you want to expand a little on that?

A Yes. So going back to comments I made pre-lunch, what we know about ventilation systems currently with the specifications required is that they do not perform as we think they are. There are areas within a ventilated room-- There are blind spots. There are areas of current that are not delivering what we hope they're delivering, and so we already know that what we have doesn't perform as we would like to think it is, and the other reason being, I haven't seen any evidence that it would make a material difference. Theoretically, yes, but clinically and practically, I haven't seen that.

THE CHAIR: Thank you.

MR MACKINTOSH: I did notice there were no references there, but I went back and looked at Dr Mumford's report with Ms Dempster, which is the one I think you looked at.

A Yes.

Q Yes. And so, nervously checking to make sure I've got the right document open on my computer before we go to it, if we go to bundle 21, volume 1, I will jump straight into the report by Dr Mumford and Ms Dempster at page 136, if I might. And you're correct that at paragraph 9.28 there isn't a reference, but what I wondered was whether the question of whether there is a risk from aerosol liquids remaining in the air is tied up entirely with the size of the droplet. We have some evidence from Mr Bennett about that, which you may not have looked at.

A I have, actually.

Q Oh, right.

A By chance.

Q So did you see how he discussed Stokes' law and the idea that smaller particles remain in the air longer than larger particles?

A In the long list of documents that I received on Tuesday, I did my best to look at as much as I could reasonably look at and digest, of which that was one, and, yes, I did see that.

Q Right. How do you feel about that idea that that's the best way of thinking about the process, that you think about the size of the droplet and what it could contain and the small----

A Well, I think it's a bit like the whole challenge we have with ventilation and what is good and what isn't good, and that is there is in fact a huge degree of complexity in items that may seem ostensibly very simple. You have an aerosol generated by use of a tap or a shower; there are pathogens there-- there are microorganisms there, so they become aerosolised, and so hence there's a potential risk. But then what is that risk and how does that translate into clinical practice is not so simple to establish.

Q The way I was thinking about it, and again, I'm probably wrong here, sometimes you look into a room on a bright sunny day and you see dust particles in the air, and, well, that will be human skin, lots of lovely things in there, almost inevitably. That's the nature of human society. But in a high

air change environment, they will be extracted out and hopefully replaced by air that doesn't contain these sort of things. You'd accept that.

A Yes.

Q So whilst it may be that I have to press Dr Mumford on a basis for this when I see her next week, would you accept that there is a point that low air change rates will to some extent fail to extract particles – they may be small ones or light ones – when a high air change plant would extract those particles before they fell on a patient?

A Again, can I answer this in a slightly different way from the question if you don't mind? It rather feels to me we're missing the point here.

Q Right.

A It rather feels like that. So the suggestion is that environmental bacteria can become aerosolised and hence, if there's a problem with the ventilation system, that that risk is real, when the real concern around environmental bacteria is not there. It's endogenous in patients.

The question I think we have to ask ourselves is even if they're aerosolised and even if they're not being cleared, how do they then get to

cause infection in the patient? And that would be a question of transmission, of how does the pathogen get in its droplet form to the patient. And the idea that they're being breathed in, because we're talking about airborne routes of transmission, seems very unlikely. What's much more likely is the water droplets falling on surfaces. Those surfaces then have the organism on there and then transmitted almost certainly by contact.

Q Yes, because there's-- You would accept there is evidence that flushing toilets will put faecal matter into the air unless you put a lid on them or you extract it.

A Yes. Yes.

Q Right. So I was just looking through Dr Mumford's chapter. I really will ask her about this next week, but I do remember her giving evidence about splashing, and I do know that Mr Hoffman gave evidence about how larger particles will fall out of the air quite fast, and I suppose in the case that if a large particle or a splash droplet falls onto a surface, contaminates that surface, if it's equipment, or is then touched and transmits because of the actions that then follow from that to the patient, that to some degree the absence of the dilution that would have cleared that particle before it fell, is that not part of the sort of mix of all these different solutions we might apply?

A If I was going to-- In a sense, I'm the wrong person to ask. I'm not an infection prevention and control expert. However, in trying to answer that question, if I was going to address that problem, my approach would not be to say, "We need a ventilation system with high levels of air changes." My approach would be, "We need to cut the route of transmission," which basically in this context would be hand washing and in particular line care, because the most likely route of transmission to the patient then would be-- Well, the microorganism still has to get inside the patient, and probably, in this context, it would be access into the bloodstream via use of a central venous catheter of some nature, venous access. So that would be the direction I would go.

Q A sort of primary protection.

A Yes.

Q Right. Okay. Well, let's move on. Let's go back to Ward 4C.

THE CHAIR: Just before we move on, thinking of prevention of access of pathogen to patient, you

were having a discussion with Mr Mackintosh in relation to dilution by air change, right?

A Yes.

THE CHAIR: But – and if this is over simplistic, please tell me – a system which conforms either to HTM-0301 or SHTM-0301 in respect of a neutropenic ward will include, by reason of HEPA filtration and positive pressure from the patient's bedroom, a means – not necessarily a perfect means, but a means – of reducing the amount of airborne material that makes physical contact either with the patient or with the patient's immediate environment.

A Yes, yes.

THE CHAIR: Thank you.

MR MACKINTOSH: I could spend hours going through your report, but I've read it, and we'll keep reading it, and I'm not going to go back to JACIE because we've read it and we've had some evidence of people. We have to balance it out. But I want to go to methods of management of infection risk. It's paragraph 5.3 on page 115 of bundle 44, volume 5. Now, you list a series of measures that could be taken, and you describe in (a), "...ideally in single rooms with en suite facilities," which I think to be fair 4C does have, whereas at Barts you have twin-bedded bays with a shared bathroom.

A Yes.

Q I felt it was probably a good idea to ask you to discuss your ward at Barts a little bit. So we've worked out that 4C had no HEPA filtration, two and a half to three air changes an hour, which is 40 litres a second into each room, a small pressure differential to the corridor at no HEPA filtration and no pressure to the rest of the hospital. Now, if we think about the part of your unit at Barts that accommodates the non-bone marrow treatment, at it's known, patients, what do they have?

A So, the fifth floor of the King George V block at St Bartholomew's Hospital is the cancer floor. Wards 5C and 5D are the haemato-oncology wards. That's not to say patients with haematological malignancies are not managed on other wards on the floor because of bed issues. That's a frequent occurrence, in fact, a constant occurrence. And on 5C, which is the equivalent of 4C, as it happens, in this context, we have HEPA-filtered rooms with an airlock positive pressure----

Q An airlock on the room?

A Yes. So the positive pressure is from the room into a SAS,

effectively.

Q What's a SAS?

A So, an antechamber before coming to the rest of the ward, so the corridor. The corridor is not HEPA-filtered.

Q Right.

A So the HEPA filtration stops at the level of the patient's room. The positive pressure is pushing air out of the room into the antechamber and the pressure in the corridor is pushing air into the antechamber, which is then extracted.

Q So the room pushes the air to the antechamber and the antechamber pushes the air outside.

A No, no. The antechamber is-- The air's pushing out of the antechamber.

Q So it comes in from the room into the antechamber and it goes out.

A Yes, because we don't want the air getting into the rest of the ward because we may need to house patients there who have transmissible airborne infections, such as viruses principally. But that is not the setup for the whole ward. So some beds on the ward are not HEPA-filtered, do not have positive pressure. It's the normal ventilation of the hospital.

Q What air change rate would they have?

A I don't know. I mean, the air change rates in our HEPA-filtered rooms is the 12 per minute, but for the other rooms----

Q You mean 12 per hour, because that would be quite exciting, 12 minutes.

A Yes, per hour. That would be slightly windy in there, yes. But the non-HEPA-filtered part of the ward is a standard hospital ward of beds, and they are also two-bedded with shared bathroom facilities.

Q And would you put neutropenic patients in the bays that aren't HEPA-filtered?

A We would, and that frequently happens, but we try and avoid it, but it's----

Q Why do you try to avoid it?

A Because the patient is neutropenic and we're trying to offer them greater protection, but it's not practically possible. So let me rephrase that slightly. We do not have a policy to say a neutropenic patient cannot be on a bed in 5C that's not HEPA-filtered. We don't have anything----

Q If we think about HTM-0301, the appendix, which I'm going to hope you remember because I'm not

sure I can find it at short notice, it does describe, I think, the concept of a neutropenic ward.

A Right.

Q I know the SHTM-0301 does. I just wonder what you thought those words would mean. I mean, only in the context of haematology and adults, which is your field.

A Yes. So let me tell you about 5D, because we've only spoken about 5C.

Q Which is the 4B equivalent.

A Yes. So 5D is a 20-bedded ward which is entirely HEPA-filtered. So to get into the ward, you have to go through an airlock, and once you're in the ward, every----

THE CHAIR: Sorry, you have to go through a----

A An airlock.

THE CHAIR: Yes.

A So two double doors, of which there's air pressure in between. Once you're in the ward, then the whole ward is HEPA-filtered, then all the rooms that have positive pressure, there's no antechamber----

Q Yes.

A -- because the whole ward is HEPA-filtered, and there's one room on the ward that has negative pressure in order to accommodate

patients who may----

Q Be infectious?

A Yes, exactly, yes.

Q And so, effectively, what you seem to be telling me is that the differences between the Barts arrangement and the Glasgow arrangement are, in no particular order, in the 4B equivalent, you have a HEPA filtered corridor?

A Yes.

Q And you presumably have a higher air change rate than 4B if it's at six?

A Yes.

Q But otherwise it sounds as if you're looking, in your eyes, at the same sort of thing.

A Yes.

Q But in 4C or indeed 5C, the difference is that there are isolation rooms of a sort. They are positive pressure isolation rooms, would they be called, or----

A Yes, yes.

Q Yes. The positive pressure isolation rooms for what proportion of the patients?

A There are 14 such rooms.

Q And how many beds in the bays?

A So we have 14 isolation rooms and the bays have six beds.

Q So the----

A In total. So two-- three two-bedded bays.

Q Yes. So the majority of the patients in the ward are put in HEPA-filtration space?

A Yes.

Q So this is my question. Let's imagine you went into work on Monday and they said, "Terribly sorry, Dr Agrawal, there's a massive electrical fault in Ward 5C. We're going to move the entirety of the fifth floor to another ward where there are bays, no HEPA filtration, three air changes an hour, no pressure differential. Are you happy with that?" What would be your response?

A Well, the first response would be, "Why do we need to move the patients," if that's where they're moving to?

Q Let's imagine they're going to be drilling really big holes in all the ceilings.

A Oh, I see. Things are happening, okay. We need to move the patients.

Q Yes.

A There quite obviously has to be a practical response to this.

Q Yes, of course.

A So patients have to be put in a place of safety somewhere,

would I be worried about the infection risk, which is I think where we're coming from?

Q Absolutely, yes.

A And the answer that is no, because we already manage patients on the sixth floor because of space issues where there is no HEPA filtration and there is no positive pressure in the way that's being described. It's routine to manage patients undergoing autologous, so it's not the same as allogeneic----

Q Yes.

A -- but autologous haematopoietic stem cell transplants or bone marrow transplants in a ward environment without----

Q And so what's the primary mechanism by which you manage infection risks on the patients on the sixth floor and, indeed, on the bays as well? What's your primary protection mechanism?

A It's hand hygiene.

Q Yes.

A It's wearing masks and it's clinical-- it's routine clinical care, both from the nursing staff and the medical staff, at least once a day.

Q Do you have any feel as a role for prophylactic antimicrobials as part of the protection?

A Absolutely, and we have

extensive policies on prophylaxis which, basically, in the context of this Inquiry, is we do not use antibiotic prophylaxis in our inpatient population at all. So there is no category of patient now we will give ciprofloxacin or a similar drug for prophylaxis.

Q So they receive no prophylaxis at all?

A Antibiotic prophylaxis----

Q Right, but they receive antifungal prophylaxis?

A They may receive antifungal prophylaxis, they may receive antiviral prophylaxis, but it depends on the nature of the underlying disease and the nature of their treatment, but we do not use antibiotic prophylaxis in any inpatient except for exceptional circumstances.

THE CHAIR: Now, I'm guessing that that ties in with what you told us this morning about your particular interest and concern about the growth of antibiotic resistance by reason of antibiotic use.

A Yes. Can I just-- That's correct, Lord Brodie. Can I elaborate on that point?

THE CHAIR: Yes.

A It's not just my concern.

THE CHAIR: No, I wasn't intending to suggest it was in any way idiosyncratic.

A The point I wanted to make that, as a haemato-oncologist, I have a particular interest in this area, whereas I would suggest the majority of my microbiology infectious disease colleagues already think it may not be a good idea to give prophylaxis but it is still given to many, many-- as standard in many units around the country, and around the world for that matter.

THE CHAIR: Just to check a medical fact, as it were, am I right in thinking that some individuals are less tolerant of particular antibiotics than others?

A Yes.

THE CHAIR: And am I right in thinking that there are adverse side effects associated with certain antibiotics?

A I think it'd be fair to say that adverse side effects are associated with all medicines, including all antibiotics----

THE CHAIR: Well, it occurred to me as I was asking the question that's probably true, but that's true of antibiotics.

A Absolutely, yes. In fact, the concern goes beyond side effects of the antibiotics. The concern extends to masking infection, and I would actually say that's a principal concern, is the inability to diagnose

infection that's present because we've used medicines to try and prevent that infection that have not worked.

MR MACKINTOSH: Now-- Carry on.

A So the reason is very simple. If you're going to use good antibiotics such as – the class is called fluoroquinolones – so generally ciprofloxacin, levofloxacin. You can use other drugs to prevent a problem, then you're very unlikely to be able to detect any infection that's present in blood cultures. So blood culture positivity rate decreases dramatically in those circumstances, does not mean the patient doesn't have an infection, but it means you cannot detect it. Why do we think the patient has an infection? Because they've developed a fever when they're neutropenic, which is a daily occurrence in every single unit around the world. So febrile neutropenia is a normal occurrence. How you manage that has a profound impact, not just on patient outcomes, but also the microbiology of the unit, which is one of the issues for the Inquiry.

Q I think I'm going to make a fool of myself, but I'm going to have a go. If I understand what your evident position is, is that most units, potentially including 4C, would have made a practice of widely prescribing prophylactic antimicrobials.

A Yes.

Q And that'd be in the general practice and it may still be practiced by a lot of the country. Have I got that right?

A Yes.

Q Your unit, in part because of your work dealing with antibiotic resistance, has developed-- Oh, by the way, there is a transcriber, so when you nod sagely and I think you agree with me, you have to say yes, otherwise the transcriber doesn't get it.

A Oh, sorry. Okay.

Q But your unit, partly in response of your desire to reduce antibiotic resistance, has a more sophisticated policy where you choose what to do based on the risk. Would that be a fair way of putting it?

A It would be a fair way of putting it, and I would also say it's based on an awful lot of evidence – that's to say published evidence – and it's based on multiple international guidelines recommending that approach.

Q So when you have a patient for whom the risk justifies the prescription of antimicrobials of any sort----

A Yes, yes.

Q -- you would prescribe them?

A Yes, and we do.

Q Right, and you do.

A Yes.

Q Am I right in thinking that if you had a patient for whom the risk would justify prescribing such a medication, but they can't tolerate it, you would have the option of moving them into one of those isolation rooms or even up to 5D. Is that a possibility?

A No, I think we're----

Q Am I talking apples and oranges here?

A Yes, yes.

Q Right. Well, can you help me break this down? Because obviously, I understand as a non-medical person that a blanket policy has the advantage of covering everybody.

A Yes.

Q And I had planned to ask you a terribly clever question about what would happen if you have a blanket policy and you can't prescribe somebody.

A Oh, I see, yes.

Q And I might still ask that. But if you're a more sophisticated policy of risk management, is there ever a scenario in your ward where the risk indicates the appropriateness of prescribing some antimicrobial of some sort and, yet, for other reasons to do with the patient's conditions, you can't actually prescribe it?

A Yes.

Q Well, what do you do then to manage any risks that was prompting the original decision?

A So just to be clear about this, we're talking about prevention as opposed to treatment?

Q Yes, absolutely. Prevention, yes.

A Yes. So probably the best example, given that we do not use antibiotic prophylaxis, so that's not a good example now.

Q Right, even if you think, "This person's at risk, I'm going to specifically use it for them," you wouldn't do that?

A But the whole patient population is at risk.

Q Right.

A And the patients particularly in the focus of this Inquiry, are at high risk. They are all at high risk. But the management is-- Let me try and describe to you the scenario of what happens with the policy at 4C, which is, having seen some of the febrile neutropenic policies, is to give ciprofloxacin prophylaxis to this patient

population.

Q Yes.

A The consequence of that is these patients will all develop fever. So the problem is it's actually got nothing to do with microbes. It's to do with clinical management. The patient develops fever because we've given them drugs to render them neutropenic, damage the gut so they develop mucositis, they have central lines inserted and they develop terrible mouths which were inflamed. So there is leakage of microbes into the bloodstream from the gastrointestinal tract. So that's all well-documented. These individuals become febrile. They've just had chemotherapy. They are neutropenic or they're about to become neutropenic. So we do what is well-established to protect people from life-threatening infection and give antibiotics.

Q So that's the current when you wrote the report, 4C policy?

A Yes, but the problem with that approach, and there is a problem, is that you've already given the individual a very good antibiotic and it hasn't done what it was supposed to do.

Q So your approach is to wait until the problem emerges and then treat it targetively.

A Yes, yes.

Q Right, I misunderstood. So now I understand.

A Yes.

Q So your approach in your unit is, to some extent, reactive but it's done in the knowledge that the reaction will be more effective?

A Yes.

Q Right.

A Absolutely.

Q So we go back to the Glasgow policy.

A Yes.

Q When you wrote this report, they were operating a policy of prophylactic antimicrobials.

A I believe so, yes.

Q Would that system work for the patients who can't receive that prophylactic antimicrobial? In a sense, what I'm saying is either you have to be in a complete Glasgow system where everyone gets it, or in a very sophisticated system like yours, you can't mix the two.

A Yes. Yes. I should just comment, it's very nice of you, Mr Mackintosh, to call our the system very sophisticated. I'm not sure----

THE CHAIR: You should always be careful about counsel.

A That's very kind of you. I'll take the compliment. I'm not sure

how sophisticated it is. If you're a patient in a situation where the rest of the patients are all getting a prophylactic drug and you're not because of intolerance or basically allergy, most likely, then what should happen? Well, you can use an alternative drug if you want to give prophylaxis, and there are other options. The drugs we've mentioned are not the only ones. I assume, I can't remember this, that colleagues in Glasgow will have something written in their policy to say what should happen to a patient who is allergic to a prophylactic drug.

MR MACKINTOSH: So the final question on this topic is, is this a realistic scenario, which I'm about to put to you? You have a patient or patients who will not tolerate the particular antimicrobial that's involved and they don't end up with any, perhaps because of their intolerance, and they end up in a non-HEPA-filtered space. Should we be in any way concerned that, in a sense, neither policy, that is HEPA filtration or antimicrobials, has been made available to them and they have fallen between those two stools? Is that something we should be concerned about?

A It's something we should

be mindful of. The word "concerned," I'm not sure I would use because, clinically, I would still manage the individual in the same way, accepting that they can't have the prophylactic drug that we would have liked to have given them because they are allergic to it, for example.

Q Right.

A The HEPA filtration issue is really a specific issue, in my opinion, relating to airborne pathogens and, in that context for this patient population, specifically mould infections of which by far and away the most important is Aspergillus. It's----

Q If you have patients who can't receive antifungals and they're placed in non-HEPA filtered spaces----

A Yes.

Q -- and again, back to risk. Is that a risk that needs to be addressed? How would you address it?

A Well, in fact, in both reports, my previous report and this one, the mitigation of that risk is extensively discussed and the mitigation of that risk includes, so you can't use an antifungal drug because the patient's allergic to it. I'd have to come back to the fact that there are several alternatives----

Q I appreciate that.

A -- introduced, so there may be alternatives. It's come back to the importance of clinical care. I mean, you really can't underestimate that because that is how we alert ourselves to there being a problem or there isn't. So that includes observations done by the nursing staff, it's the information the patient tells us, and it's the daily ward-round examining the patient----

Q Because that gives the ability to react?

A Yes, yes.

Q Right.

A In the context of fungal infection, we have blood tests that allow for rapid assessment of, could the patient have a fungal infection? Which is a bit different from bacterial infections where we have blood culture, but that's always reactive.

Q Because it takes a bit of time.

A Yes, you can use the test for fungal infection as screening if you wish to.

Q I'm going to propose to move on now. This is the same document we have on the screen, but now page 116. You referred in your report from 2021 to various data about infection risks, infection results, and it's paragraph of 5.5, and you make a

comparison between data you've given for Ward 4C in 2016-2020 and a slightly longer period-- sorry, a shorter period, at Barts.

A Yes.

Q What I was particularly intrigued about this was the extent to which you-- why did you refer to Pseudomonas?

A I think this was-- I'm, again, doing this from memory now. I think these were all results from respiratory samples so this was Pseudomonas in sputum samples.

Q Sorry, what I'm intrigued by is why would Pseudomonas, which is a water-- sorry, potentially environmentally relevant microorganism be relevant to ventilation given that you've just given evidence that it's not relevant?

A I don't think it is relevant to ventilation at all.

Q So why is it in your report?

A It's in the report because there were sputum with Pseudomonas positivity.

Q So how would the patient have acquired-- That's a stupid question because you don't know, but what are the potential paths by which a patient would acquire Pseudomonas in their sputum?

A If you don't mind, Mr Mackintosh, I just suggest you save that one for Professor Hawkey, because what I don't want to do is tell the Inquiry something which may be wrong.

Q I know but the problem that I-- the reason I'm pressing you is because in this report, and I recognise it's in a different context, you draw a distinction between the infections that are recorded in 4C and the infections that are recorded in your unit and, whilst I appreciate that Aspergillus might well be relevant, and I suppose norovirus might be relevant to ventilation as well, do you see the number of gram-negative infections and Pseudomonas infections relevant to the quality of the ventilation in a ward? Because if you do, aren't you not agreeing with Dr Mumford?

A No, I don't.

Q Right, so they probably shouldn't be here is what you're saying?

A I do say in the second sentence, "in an analysis limited to bloodstream infections", so-- because that was the data available. So, if the comparison is more to do with-- The point I was trying to get across was, the documented infections on 4C, at least numerically in absolute numbers, seem to be very low.

Q Right. Based on the information you had at the time?

A Yes.

Q Right, okay. I think the final topic to mention with you discusses the issue of thermal wheels in ventilation. We've discussed air changes, I think, to the sufficiency. You seem to recognise the role of HEPA filtration preventing particles arriving. So, what view do you take of the use of thermal wheels in a ventilation system in a ward like 4C?

A I'm afraid I don't have the-- the knowledge to comment on that question. I simply can't answer that.

Q Well, I'm going----

THE CHAIR: Do you know what a thermal wheel is? I mean, you would be excused if you didn't.

A I only know it from-- from reading material in relation----

MR MACKINTOSH: Well, if you don't, that's absolutely fine.

A -- to this Inquiry.

Q I have one document I need to show to you, which is an SBAR from, I think, 2018. Bundle 4-- It's not on the list, so bundle 4, page 156. So this is an SBAR from July 2019 prepared by the then lead infection control doctor. You've not

seen this, I think.

A No, I don't think so.

Q No. That was really the question. You wrote a report about Ward 4C----

A Yes.

Q -- and you hadn't been shown the SBAR----

A I don't recall seeing this.

Q Is an SBAR a term of art in England as well as Scotland?

A I actually don't know the answer to that. I've only come across SBARs in the context of this Inquiry.

Q In this Inquiry? Right. But it seems to be, it states, a "Situation of background assessments and recommendations," a single-page document, and you'll see that ultimately, for reasons she's given evidence about, Dr Inkster recommends an upgrade in the ventilation to Ward 4C. It's in the final box.

A Yes.

Q Would you accept that there's a slight similarity? That's not even a fair point, but you accept she's suggesting there should be some upgrades, but you didn't see this.

A I hadn't seen that, no.

Q No? Okay. Just again – I know you haven't seen it because we asked you about it in one of the

questionnaires – you hadn't seen the Innovated Design Solutions report into the ventilation in Ward 2A and 2B either?

A No.

Q We've done, I think quite successfully-- at least, we've asked lots of questions and got lots of answers, about you not knowing the facts on the ground at the time, so I'm not going to revisit that. What I want to do now is to look briefly at one aspect of the BSI for results in chapter 7, simply because we've already looked at the other one, which was the adult BMT results which we briefly looked at. So we're in volume 1 and we're on age 73. So I went through this with Dr Drumright, and I can't now remember whether it was in the morning or the afternoon. This is the section entitled, "Bacteraemia in adult patients [7.2.3] in GGC January 2013-June 2023 with Potential Environmental Relevance."

Then the bottom of the page, there's a discussion of what we're to see on Figure 5, noting some spikes. The bottom of the page, an adjustment for unrealistically small denominators, and then over the page, having carried out that adjustment, there's a discussion of what's then seen in Figure 6. Now, I wanted to just check: I've had evidence from Dr Drumright

about the merits and demerits of adjusting for unreasonably small denominators. Is that something you'd want to add your words to, or are you happy to leave it to her?

A No, I think I'd best leave that to Dr Drumright.

Q Yes. Now, this page at the top, however, if we-- The sentence I'm going to look at is actually looking at Figure 6, so we'll look at Figure 6, which is on page 75, and I think there are two spikes: one in blue, which is for the Queen Elizabeth, and one in red, which is at, by that point, entirely the North. If we go back to page 74, the middle of the second line:

> "...with a single possible true spike in incidence at [Queen Elizabeth] in early 2018 followed by significantly lower incidence for the remainder of the time period."

Now, what I want to put to you is some information that we have about the events that are happening at that time. So early 2018 is the point when the water incident happens, awareness suddenly grows, and we have minutes of Water Technical Group meetings discussing the discovery of contamination that they describe as widespread. Then we have the interventions we've already discussed. I appreciate you've not looked to all the context, in epidemiological terms, and you're not an epidemiologist.

A No.

Q So really what I'm asking is, if you look at page 75 and that blue spike in the middle and the lower period afterwards that's described, should we be looking into this amongst the adult haematology patients? Should we be asking ourselves what's going on here, or is it just a spike, but you describe it as a true spike? Should we be looking into it?

A I actually don't feel I can answer that objectively. I would really need to consult with both Professor Hawkey and Dr Drumright as to what that figure is telling us. So the term "true spike" means that there appears to be a real increase at that time point. If this time point is a year-- I can't tell if it's a month or a year, actually. I'm not-- I haven't quite----

Q Well, you described it as early 2018, but I'm again slightly suspicious that it might be late 2017, based on the table, but the text says early 2018.

A So in that context, it would be-- the first thing to do would be to look at which organisms are we

referring to, is there a pattern? And I can't remember----

Q So let's just think of the steps, because I think it's important that we just think of the steps rather than reaching a conclusion. We've had Dr Drumright talk about scenarios and counterfactuals, and she's given us some thoughts on that subject. Would we talk to Professor Hawkey about organisms and that sort of stuff?

A Oh----

Q And that would be where we would go?

A I think so, depending on the question----

Q But you don't feel able to add to that topic of conversation?

A I don't think so, no.

Q Okay. Thank you. I might just take a moment just to check that we're about to get to a topic change. (After a pause) I think what I'll do now is I'll move on to the topic of Aspergillus and chapter 8, and then I'll come back to the final charts that were produced by the three of you after that. So if we go to chapter 8, which is on page 123. Now, why did you carry out an examination of rates of infections for Aspergillus in this hospital?

A Yes, I think that's partly addressed in chapter 6, if I recall----

Q Yes.

A -- and when considering the impact of ventilation, I would still insist that the-- the most relevant outputs of that would be airborne pathogens.

Q Aspergillus would be the most common?

A So, as we've just discussed, I mean, potentially-- Well, particularly mould-- mould infections, the spores, so fungal spores, and principally, epidemiologically, numerically, that's Aspergillus in this patient population. There are other mould infections that occur, but numerically it's Aspergillus.

Q Is the fact that it's numerically the biggest of this group----

A Yes.

Q -- but in a small field, the reason you pick it? Because then there might be a chance of doing something with the numbers?

A Yeah, yes.

Q So to take, for example, Cryptococcus neoformans, you couldn't do it for that. Could you?

A No, I mean it would just be too difficult. I mean, I don't see-- It's such an infrequent organism to find in this patient population.

Q Yes.

A It's not that it doesn't

happen, but it's really very rare. Aspergillus, as I know the Inquiry has already heard, is an environmental, ubiquitous organism. We're all breathing it in here at this point in time. It is quite literally everywhere, hence the risk of this to this patient population in particular.

Q So having decided to carry out this exercise, you weren't of course supplied with a nice spreadsheet of results, were you?

A No. Once again, the question was, "How are we going to attempt to answer the question of, are there-- were there increased rates of infection in the Queen Elizabeth University Hospitals, including Royal Hospital for Children?" and so we again took the same approach, which was to ask the Glasgow Health Board to provide us with as much data as they could, including retrospectively going back to before the hospital was built – in other words, other sites – in order to try and establish an answer to the question. So it's, in principle, the same approach.

Q But it actually involved you making some actual clinical assessment?

A Yes.

Q Yes. So that's different from what effectively was going on

with the bloodstream infections?

A Absolutely, because the bloodstream infections, if you have a positive blood culture, then you have a positive. With Aspergillus infections, and in fact mould infections in general, it's really quite complicated. Just-- So say a patient had invasive Aspergillosis, it's unfortunately not simple.

Q Because it's not just a blood test?

A Well, principally because, to define that the patient has had invasive Aspergillosis, you need to biopsy a lesion which contains the hyphae of Aspergillus growing into tissue, or you have to culture the organism from sterile material. So for example, that might be from cerebrospinal fluid in individual who (inaudible)----

Q So----

A -- involved----

Q -- you would have a series of different tests and you'd have data for those tests?

A Yes.

Q Did you also use imaging data?

A So the challenge with imaging, and there are many challenges with imaging-- So one approach would-- So let me just go

backwards. It is in the report, but perhaps it would be useful for the Inquiry if I just quickly summarise it. There are really three arms to accepted international criteria for classifying invasive fungal infections, in this case, Aspergillosis. The first one is, is the patient at risk? And by definition, all of these-- this patient population is at risk. So we tick that one, that's easy. The second is microbiological data. Have we-- Do we have any data to say this could be Aspergillus? And now that data can come from histology, it can come from culture----

Q Histology, for the uninitiated, is?

A A biopsy of a tissue somewhere.

Q Right.

A Since these lesions are airborne, since the pathogen is airborne, is that-- airways and specifically the lung, that are the main sites of infection, taking a biopsy from a neutropenic, unwell, thrombocytopenic individual is usually not an option, so it's-- it's not available to us. Culture of sterile material is rare, so that happens rarely. In fact, in my experience, the only time you're going to culture this organism is from a brain abscess, otherwise it's not going

to happen.

So that leaves us with other microbiological data, which is sputum, what's called bronchoalveolar lavage fluid. So for patients well enough to have their lungs examined and washed, then that's a very useful material.

The third thing is blood tests. So that's where fungal infection is slightly different from bacterial. We do have a number of blood tests that can guide us, and so we asked the Glasgow Health Board to provide us all the data that they had----

Q For all those things?

A Yes, for those things. Yes. Imaging, the third branch, is complex, and the reason it's complex is because these individuals have imaging done very frequently for many different reasons. So, the prospect of trying to go through all of their imaging would have been a not insignificant task.

Q So did you select, in some way?

A So the only-- Yes, I did select, but what I selected was those cases that didn't meet criteria, but if they had suggested imaging, I would have preferred to count them as cases rather than not count them as cases.

Q So in a sense, you've got

the ones from the various tests----

A Yes.

Q -- and you add to that----

A Yes.

Q -- some that don't have the tests, but where the imaging is suggestive.

A Yes. So let me just be really specific about this. The criteria I used would not be accepted by the international criteria.

Q So it's not comparable with the international criteria?

A No. So I was using a much less stringent criteria, and by doing that I would almost certainly inflate the number of cases. That was very deliberate, because I did not want to underestimate. I'd rather err on the side of having more-- a few more cases, perhaps, rather than a few less, given it's so difficult in the first place to decide if a case is a positive or a negative.

Q So how do you-- I mean, obviously this must take a significant amount of time.

A It took some time, yes.

Q Can you give us a feel for when you started doing it?

A When did I start to-- Without looking at my records, I'm-- I'm really struggling.

Q Are we talking months or weeks or days or hours?

A It probably took me a few-- Well, I mean, the initial tidying up of the data in order to get it into a format that I could then use was-- was a few weeks, and then I spent a fair bit of time joining up all the dots, so to speak.

Q Yes.

A So it's a rather manual exercise.

Q The reason I mention that is because, whilst I don't wish to minimise Dr Drumright, to some extent, she takes a big spreadsheet, she puts it in a software package, and it spits out some numbers. I mean, it's a lot more complicated than that, but it's a relatively quick process in comparison to just checking that.

A I don't know if Dr Drumright would agree with that----

Q No, I'm not saying she would----

A -- but I would agree with that.

Q You would agree with that? Excellent. Now, what I want to do is ask you to comment on the NSS response to your document. Now, the reason that-- They did two responses, and it's the second one that I want to put to you. Unless I've gotten confused, it's in bundle 44, volume 3,

and it's on page 222. Because we can read your response to the first report----

A Right.

Q -- in your HAD response, and we read that. This one is a response to Ms Cairns seeing your actual data sets.

A Yes.

Q Because the timing didn't quite match. Have you read this document?

A I have.

Q You have? Right. If we pass delicately over the effective summary and we go to the review of data at page 225, the first issue that she raises is on paragraph 6 at page 226 about de-duplication. Now, before we go any further, I think I have to put something to you that Dr Drumright and I might have inadvertently said yesterday. I don't think NSS is suggesting a 14-day de-duplication exercise, which I think is what I ended up putting to Dr Drumright yesterday.

A Oh, I see. Okay, yes.

Q But what they are I think suggesting is a de-duplication exercise.

A Yes, yes.

Q So, given the length of the incubation period, which I suppose varies quite a lot, what's the appropriate way to deduplicate, and why do you not specify a time parameter in your process?

A Yes. I'm just struggling a little bit with the best way to phrase this, because it's actually-- it's a slightly nonsensical question in the context of mould infection.

Q Right.

A It's not how mould infection works, it's not how the treatment of mould infection works. So the analogy with bloodstream infections where a bacterium gets into the bloodstream, leads to infection, detected in the blood culture, treat with antibiotics and it clears and the patient gets better, principally due to antibiotic treatment and with the help of neutrophils, if the patient's neutrophil count recovers. If there's a second episode at a defined period after that, then that's regarded as a new infection.

Q Right, because the first infection would have been cleared.

A Exactly, yes. Now, so if you go backwards here-- So the first thing, as I've just told you, it's incredibly challenging to actually establish the case as a case, and then once you've decided it is a case and the patient's treated for it and hopefully they get better, then they will get better

in the context of their treatment and recovery of their neutrophils.

Q But they'll still have it in their body.

A Yes, exactly. That does not mean there is no infection. So it's very, very different from bacterial infection, and that manifests itself by-- Typically this patient population is having cyclical courses of chemotherapy for example, so the next cycle of chemotherapy, patient suffers all the toxicity of the treatments, becomes neutropenic, and hence there is a risk of that pre-existing infection causing a problem.

Q Because it comes back, because----

A Yes, because it never completely left. So then trying to say there should be a 14-day, 28-day window becomes slightly nonsensical. So the only way I can-- This is for mould infection. It's different for candida bloodstream infections. So, the only way I can sort of rationalise this is to say in an individual who had an infection, was treated, they got better, their immune system recovered and they were well for a period of time that's quite hard to define. So there is no accepted duration of treatment for invasive aspergillosis. The typical statements made are from six to

twelve weeks of treatment, but that isn't actually based on any data.

Q So can you deduplicate at all?

A No, I don't think you can. I think you'd have to have-- So there were one or two individuals in the dataset who had multiple positives over a series of months, and some individuals who had multiple positives over a period of approximately one month. So the key-- It's very difficult to say that any of those represent new infections.

Q So, effectively, did you exclude the ones in the one month and keep the long ones or just include everybody?

A No. I looked at evidence around hospital admissions at-- If an individual had not left the hospital, then this was all the same episode, and unfortunately, it may well be possible that the individual died. I didn't have that data, so I don't know, and so what we're looking at are the consequences of unresolved infection.

Q But you took that as one incident?

A Yes. These are not repeatedly new infections, no. No.

Q But the definition there is a bit soft.

A My definition or this

definition?

Q Your definition, the one you used. Or is it with simply----

A What, deduplication? No. That's the point I'm trying to make. This is a nonsensical concept for invasive aspergillosis.

Q But your deduplication method that you ultimately chose was the one admission, one infection method.

A Yes.

Q So they came back and they got aspergillosis again, that was two infections.

A I'm afraid it's not that simple.

Q Because sometimes you had them-- you took that as the same infection.

A Yes, yes. It depends on what was happening in between.

Q We just have to accept to some degree that these numbers contain this judgment by you.

A Yes.

Q Right. And you think that's because of the inherent nature of aspergillosis.

A Absolutely, yes.

Q Right. Now, if you look on page 227, I wondered if you have an opportunity to consider this challenge about the PCR test you used. Do you understand the criticism----

A Yes, yes, yes, I do.

Q So what's your response to it?

A So firstly, my apologies to the Inquiry and to Dr Cairns, in that I didn't put every single methodological detail in the original report, for which I apologise. I thought I'd been very detailed about classification of cases, and I had been quite detailed. Then when I got into the actual nitty-gritty of looking at the data, that spreadsheet, the Excel data file, was not provided to the Inquiry when we submitted our report. That was provided subsequently when questions were returned.

Q It's the one that Ms Cairns has responded to.

A Yes, but it's all in there.

Q Right.

A So in there I've annotated each apparent outlier, each case where there is a positive, as in the case Ms Cairns is highlighting, but it wasn't considered, and that's to do with the fact you can have a PCR positive result, apparently, but at a CT level, so CT represents the number of cycles to get to positivity. So if the test becomes positive at a low number of cycles, that's telling you you have a

strong positive, and if it takes a lot of work to get to the positive, it tells you have a very low level of detection.

Q And so you had a threshold in that.

A Yes, and at that level of detection it's not always easy to know, is the test picking up water or is it picking up the pathogen?

Q And so the effect of this is a bit like the use of imaging, in that you will end up with slightly more cases than you meant to.

A Yes. Yes.

Q Right.

A Yes, so what I didn't accept were cases that really had no evidence that there could have Aspergillus infection. So I thought it was reasonable to say I can't start accepting everything that has no evidence.

Q But when she first saw this, she wouldn't have been able to see this?

A Not initially, but on the----

Q Yes. I mean, that's just a general point. You didn't include in your report an appendix, data tables and----

A No. Yes, so that was----

Q Any particular reason why?

A Absolutely none. I think it was just an omission, I'm afraid.

Q Right. You wouldn't count it as misleading us?

A I'm not sure why I'd wish to do that, and I wasn't.

Q If we go on to section 9, I think this is the clinical review of imaging issue.

A Oh, yes, yes.

Q Which we've already discussed. To what extent is this use of imaging an essential part of effectively doing what you did?

A So again, it may well not be easy to understand. I genuinely mean it's a slightly complex area. We have lots of different tests and, you know, what do they mean? So this refers specifically to those individuals whose only positive test was something called a Beta-D-glucan.

Q Yes.

A And the Beta-D-glucan is an indication of a fungal infection. However, it's not an indication of Aspergillus infection, and as per all international guidance, that by itself cannot be regarded as an invasive aspergillosis. In fact, there's no evidence a patient has Aspergillus. Much more likely to be candida, if anything.

Q Yes.

A However, if those

individuals had imaging suggesting the fungal infection, then I would have had two elements to indicate, even though I can't say it's aspergillus. If there's a lesion on the chest that looks fungal and the Beta-D-glucan is positive, I would have accepted it as a case.

Q So you effectively put it in?

A Yes, but there were no positive imaging in relation to these isolated Beta-D-glucans.

Q The only other point is paragraph 10, which seems to amount that you've missed a case out in 2014. Is that roughly what is being said?

A Yes, I looked at that and I couldn't agree with Ms Cairns on that, no.

Q Do you think you haven't missed one out?

A No, I don't think I've missed one out.

Q Any particular reason?

A It would be helpful if I had my notes because I made a note of it in our reply.

Q But it's in the paediatric data set in 2014 at Yorkhill.

A No, I went back to look at that individual case. I could not find the positive result she was referring to.

Q Okay. So what I want to do now is to put aside Ms Cairns' note and actually go back to chapter 8 itself to try to understand some of what is in the chapter and then step forward, look at Dr Drumright's work and then return before we finish to the BSIs and the concept of risk. So if we go to chapter 8, which starts on page 123 of bundle 44, volume 1, and I'll take this relatively quickly because I think I can jump to the conclusions. Page 125, you plotted out the numbers of cases you identified across the three cohorts. So orange, those patients are in the Beatson in North sector the entire time.

A Yes.

Q And then the amount they have on the chart at Figure 23 on page 125.

A Yes.

Q And then blue, they're in ward 24, Southern General. We know that, you don't, but they're in Southern General until the move in '15, and then they're in Ward 4C through to the end. And then green are in Beatson, ward B8 I think. I may be wrong, but certainly the Beatson until the move. They go back to the Beatson and then they come to Ward 4B in 2018, and that's the green, and you've plotted that at the top.

A Yes.

Q You didn't calculate a

rate for this population. Is there any particular reason why you didn't?

A Yes. Firstly, I would have needed Dr Drumright's expertise in doing that. It sounds simple enough, but I didn't feel I wanted to be handling that sort of data set for a huge Excel spreadsheet. That's one thing.

Secondly, looking back at my previous report, I had commented on the very low rates of infection and I'd attempted to make a comparison of population served as opposed to infection rates; and, making the point that there appear to be very low rates of infection on 4C in that report, I should have at least added some form of comparator here, which I didn't, of that nature. The reason I didn't think about calculating rates is because the observed number of cases, despite my attempt to be very permissive in classification, is very low. From my clinical experience, given the size of the population covered, these are very low absolute numbers of infections.

Q And if we look at Adult South, which is blue, from 2017 it's one case a year.

A Yes. I mean, so, I was struck by this data in the manner I've just said.

Q Yes. Well, if we look at the paediatric data, which is a few pages on at page 128, Figure 124, it's much simpler. 128, please. You've plotted again the columns by numbers, and this time you've helpfully provided a translocation diagram of where the wards are at the top. You do seem to indicate there's something different. If you look at the bottom of the page, you're referring to table 19, but it's the same data as on the chart. What is it you noted in terms of the numbers?

A So, looking at this, it's-- I found it-- So again, this is just absolute numbers, not rates.

Q Yes.

A Which is, I realise now, an error. I should have asked for rates at the very start. I think that would have been helpful, but obviously that came a little bit later. I mean, what I'm struck by here is overall, the numbers remain very low. It's the same population being served in the sense of the total population covered by the Glasgow Health Board is still 5.5 million.

Q Well, it isn't. This is a national centre.

A Right, yes.

Q And the other one isn't a national centre. The other is a regional centre. The adults is regional. This is national.

A Oh, I see. Okay. I hadn't picked up on that difference. The point being, I haven't calculated a rate for this. I'm really struggling with my glasses here.

Q I mean, Dr Drumright eventually does.

A Yes. So, I felt that there is not any indication of rates going up and down based on the paediatric population being on Wards 2A and 2B, which seemed to be the key issue.

Q Because the reason that-- When I look at the top of that table, Figure 24, I don't know whether you know the ventilation arrangements in 2A and 2B and 6A and 2A and 2B in that transition of time. Do you know what was going on with ventilation?

A Well, sorry, when you say transitional time----

Q So it's in between 10 June '15 and 26 September '18.

A Yes.

Q Do you know what the ventilation was in 2A and 2B?

A No.

Q And do you know what the ventilation was in 6A between 26 September and 9 March '22?

A No.

Q No. But you do know it's in 4B because-- And you do know what the ventilation is in the new 2A after March '22?

A I believe that it is HEPA-filtered for the bone marrow transplant service and-- No, I don't----

Q The reason I raise this is that we understand that after 9 March '22, 2A and 2B have 10 air changes, HEPA filtration, and 2A has a positive-- has a lobby, and they're highly kitted out, newly fitted ventilation system, in contrast with the period before. 6A is a standard ward, it's the same as 4C, and 2A, before the move in September 2018, outside its BMT rooms, was the same as 4C. And the reason I say that is, in a sense, if quite a lot of these patients were in the same as 4C from '15 to '22, does that make one look at this chart in a different way? Whereas before then they were in Yorkhill, which had HEPA filtration and positive pressures and airlocks and things. Does the knowledge of what's going on change the way you look at the chart?

A No. I'm struggling to see it does. I mean, looking at the data in front of me as it is there, I don't get a sense of there being a significant change over time with location. I mean, yes, we have got two years of seven patients in total, for example, in 2017 and 2019 with individuals in different locations within the Queen

Elizabeth University Hospital and Royal Hospital for Children sites, whereas we had a maximum of five prior to the move. Does that indicate a problem? Does that indicate a true increase? This is where the analyses performed by Dr Drumright perhaps gives us some clues as to something happening.

Q Right. We're going to go and look at that. I think that would be a useful thing to do. So if we go to bundle 44, volume 7, we'll just do it for the paediatrics. So we go to page 42. 44, 7, 42. Sorry, 44, 5, 42. We'll go back there in a bit. So that's bundle 44, volume 5, page 42. Yes. So, I'm going to pass over the monthly counts table; I'm going to pass over a chart on the next page which Dr Drumright confirmed was actually mislabelled. This is BSI results. And if we keep going through this section, we will eventually-- in fact we go a bit further, we go all the way-- We shouldn't have started here at all. We go to page 75, we go to, "Incidence rates of Aspergillus infection in paediatric haematology-oncology," and 5C.1, would that effectively be your chart, but now monthly?

A I think so, yes.

Q Yes.

A Yes.

Q And then we go on to the next page on page 76, 5C.2 is effectively your chart, but now it's got peaks.

A Yes.

Q Yes, right. But the negative binomial regression is on the next page and Dr Drumright has given evidence consistent with your comments.

A Yes.

Q Now, I appreciate it was her analysis.

A Yes.

Q If we think back the original purpose of your instructions, albeit that you don't have detailed knowledge of the ventilation systems that are in place, what do you take from Figure 5C.3?

A Well, I mean what we're looking at here is what appears to be, over time, an increase but it's at very slow rate----

Q Right.

A -- and the slope of the curve, it's not flat. It is going up, but it's-- yeah, it's a very slow progression. So what does that mean clinically? We would speculate, my suspicion would be that if there is a genuine slow increase, that's probably related to factors, one, partly potentially occurring outside of the hospital, that's

to say changes in Aspergillus epidemiology, which are well known to be occurring globally. It could be as well changes in the treatments delivered to children with haematological malignancies.

Q In that they are more vulnerable to Aspergillus?

A Potentially more vulnerable, new drugs, new approaches, ability to treat sicker children, etc. So I suspect what we're seeing is a combination of factors rather than one specific factor. I would struggle to attribute that to a specific problem of the environment of the hospital.

Q Do you feel confident in getting involved in a disagreement between Dr Drumright and Mr Mookerjee about the statistical significance of a change at the move of the hospital, or is that something you'd prefer to leave Dr Drumright?

A I think Dr Drumright would be the best person for that, not myself, yes.

Q So what I want to do is to sort of go back to the conversation we had about risk just before the lunchbreak.

A Yes, yes.

Q So we can take that chart off the screen. I was asking you about risk and safety and you mentioned harm and his Lordship asked you about whether we're looking at eventuated risk. It seems a terrible question to ask you in front of his Lordship, but what do you take to mean the idea of looking for eventuated risk? Risk which actually happened?

A I can just go backwards to-- I think your original context of when you asked me that was I think, Mr Mackintosh, how would I feel about a ventilation system in a new ward, a new hospital not being validated----

Q It was, that's what I asked you.

A -- and patients going into that unit. What would be the risk or how would I feel about that? Well, I'd be incredibly unhappy with that but, the reason being, I know nothing about this ventilated space and I did mention at the time that the key thing I'd want to know is not the number of changes per hour and is not the positive pressure, it would be is the HEPA filtration working? Is it actually keeping out particles? Is there any chance of contamination somewhere in the system such that is actually pushing in pathogens into the space which would be a potential disaster? So that would be my main concern.

Q It's a precautionary response.

A Yes, if there was clinical need, let's say the hospital the patients are coming from has got to close on----

Q On Monday because the electricals have gone, yes.

A Exactly, yes. Then it's a question of risk balance and, as we've discussed before, it's possible to mitigate the risks of the air handling unit not performing to the level that it should be, according to specification. And as I've tried to really emphasise, I wouldn't be worrying about that excessively since I'm aware of the limitations of the ventilation systems we have that are working properly.

Q To what extent would be your-- the fact that you wouldn't worry to that extent, is driven by the fact that the risk is acknowledged, do you know what it is? It's been validated, it may not be what you want, but you then manage it as an acknowledged risk. To what extent is risk management about acknowledging a risk?

A Yes, absolutely. I mean, my problem with the scenario was that I didn't know anything about----

Q Yes.

A -- it's just there and we don't know anything about it.

Q Well, the reason we ask it is because the ventilation system of the hospital wasn't validated and people didn't know that. So that's why we asked the question.

A Okay.

Q So that's the reason we ask it. I appreciate your answer. I'm grateful for it. I wonder if we go back to the executive summary for the HAD Report to just pick up some questions. It's on page 6 of volume 44, volume 1. Now, it was to explore paragraph 3. Now, we've been over a lot of these factors, and it may be we're diving in too deep but we'll try. So:

> "Infection risk is multifactorial and varies depending on: a micro-organisms ability to cause disease (pathogenicity), its natural habitat (source), ability to spread (mode of transmission) susceptibility of the host to infection (opportunity)."

What about the dose of the-- or the quantity of the microorganism that is in the environment? Is that a factor that we should think about in the multifactorial balance?

A I'm going to defer that to Professor Hawkey, if you don't mind.

Q Because the reason I wanted to ask it relates back to the

conversation we've just had about dilution. If you have a microorganism that's quite infectious, you don't need much of it, then a small dose, if that's the right word, one might think – and I'll check in with Professor Hawkey – might give rise to a risk. So could that be a reason why dilution is actually not an unimportant thing to consider? Even this is an area with lots of limited evidence, or am I just on the edge of reason at this point?

A I think we addressed this earlier----

Q Yes.

A -- but I want to try and be consistent in my response to that, acknowledging that Professor Hawkey is probably a better opinion than me on this. I feel we are asking a bit too much of the ventilation system with respect to-- when it comes to airborne pathogens, I don't think the question-- we accept that ventilation systems, their role is there even though the data is very limited.

Q Right.

A Once we start to go to non-airborne pathogens, in other words, environmental bacteria, then I feel this is stretching it a bit too far but perhaps Professor Hawkey would be a much better opinion.

Q So I want now to return to a topic,we can take that off the screen, that I put to Dr Drumright, and you may have heard me do this. So did you read the case notes review overview report which is referred to in your document?

A I did.

Q And so you'll be familiar with the idea that they conclude that 30 per cent or thereabouts of infections are more likely than not to be environmentally related.

A Yes, yes.

Q That's their conclusion from their exercise and we can't see their workings, which affects the ability of the Inquiry to use their material. But to what extent is their conclusion inconsistent with the view expressed in chapters 1-6 of this report, which is repeatedly stated that micro-organism X or Y is generally not environmental? I mean, is there an overlap, is what I'm trying to say, between your positions as a trio and the CNR as a trio, that it's possible that up to 30 per cent of the infections could be environmentally related of course, 70 per cent aren't. Are we actually arguing slightly across purposes here?

A Yes, I think we probably are. I mean, it's-- the Case Notes Review, when I was reviewing it, I was reviewing it from the perspective of our

work in trying to answer the questions or the tasks given to us by the CLO, and we had an approach which was to establish rates of infection and have a comparator. So that was our approach.

The Case Notes Review clearly looked at individual cases over a particular period of time, which was much shorter than the period of time we looked at in our analysis, but in great detail. So their activity was completely different from our activity.

Q That they describe it as applying normal infection prevention control, root cause analysis, effectively?

A I do not wish to disagree with that at all.

Q Right.

A I'm just making the point that what they did was completely different from the exercise that we did. So perhaps attempt to direct comparisons between their reporting conclusions and ours is a slightly misguided exercise. But the point, at least for me, was that the exercise-- or the report of the Case Notes Review wasn't providing context in terms of, you know, what's happened to that population of patients before and after the period that they identified there was an issue. So I think what we're

seeing in our dataset is the problem they identified, because the reviewed analyses, which I suspect we're going to come on to----

Q Yes.

A -- we can see there is something happening in the paediatric population and there is an increase in bloodstream infections which, presumably, marries in very nicely with the CNR report because they found there were infections. The next step which is-- I think Dr Drumright's phrase was "attributing potential causation," or, you know, what may be causing that increase is where there is an issue.

Q Well, let's go and look at page 17 of bundle 44, volume 1 which is where first comment on the CNR. Last paragraph:

> "A significant reliance appears to have been placed on the Queen Elizabeth University Hospital and Royal Hospital for Children Case Note Review Overview Report (Stevens, Evans and Wilcox, 2021) (CNR) in relation to whether the built environment at the QEUH and the RHC posed an increased risk of infection. However, we do not consider that the CNR is of

assistance in determining this for the reasons set out below."

It's not quite what you just said, is it? You seem to have changed your position slightly?

A Well, I think-- Well, no, I don't-- Have I? I don't think I have actually.

Q Because you've described it as having validity, and as potentially seeing the same feature, but from a different end of the process. They've come from individual cases and they've seen an exceedance or something and you, through your revised report which we're about to come to, have seen something in the data as well. And there seems to be-- you seemed very emollient about two minutes ago about their value and that sort of thing, and this is rather a different criticism.

A I'm not sure I agree with that summary.

Q Well, then please correct me.

A The point I made, I thought, was that their attribution of approximately one-third of the cases that they reviewed as being attributable to the environment of the hospital, I wasn't really sure how they had done that, not least because, as you pointed out, don't have----

Q Their data.

A -- their workings. So it does make it rather difficult to, or impossible in fact, to comment on how they got to that conclusion.

Q Right. So you don't feel that, I mean, presumably because you haven't got access to the data, you can't really criticise their methodology in practical terms because you can't see what they did. Is that what you're saying?

A Well, that's certainly a fairly profound problem, but also, and this is something I think Professor Hawkey will cover in much more detail is the whole lack of confirmation, let's put it like that, maybe I haven't-- I'm not phrasing that appropriately, of the whole genome sequencing data that does exist to show while there are, I think, at least two infections which by whole genome sequencing do show a link between the clinical infection in the patient and the environment. The remaining analyses haven't found that to be the case and, in fact, found that the clinical isolates that have been typed and environmental isolates that have been typed are all different.

Q Yes.

A So that's just one element of a number of arguments to just make me wonder, on what basis

did the Case Notes Review seem to come to what-- to the conclusions that they did?

Q Well, let's actually look at your recalculated data for the paediatrics, and I'm not expecting you to go into the statistical significance of what you see----

A No.

Q -- or indeed the epidemiology and Dr Drumright talked about the possible scenarios and counterfactuals----

A Yes.

Q -- and she gave evidence of how we might think about it. I think she acknowledged she hadn't read the CNR, but she gave us thoughts about (inaudible). If we go to bundle 44, volume 5, page 50. So if we take the top half of the page, this is the environmental BSI.

A Yes, okay. Yes.

Q Just for completeness, so you don't think I'm trying to hide it, if we go to page 51, we go to the non-environmental.

A Non-environmental, yes.

Q Let's go back to 50. I appreciate you're not an epidemiologist. So there's going to be a point, I suspect, when I ask a question in a moment and you say, "That's not my field, I'll defer to Dr Drumright," but we'll try.

A Yes.

Q So one of the first questions I asked her was I asked her to consider that obviously water and ventilation were scenarios that were being posited and a series of counterfactuals, and they included line care, single rooms and nursing, I think to some degree they became one counterfactual, but they were discussed----

A Yes, I think so.

Q -- and to my antibiotic usage and the effect of that. She mentioned concerns that she had, admittedly without looking at evidence, about laboratory work and contamination, and then concerns about what you might term dysfunctional management at a very broad sense. Now, firstly, are there any counterfactuals you'd like to add to that list before we go any further?

A Within use of antimicrobials, so just to emphasise the point, prophylaxis as well as use of antimicrobials outside of prophylaxis is relevant.

Q Yes.

A But also in terms of the workings of the department, specifically with respect to febrile neutropenia and its rapid treatment.

So delays in rapid treatment of febrile neutropenia in the context of true infections can lead to negative outcomes.

Q Well, they would do, but would they lead to more infections? Since that's the measure we're looking at and not the outcome?

A Yeah, I understand that. The reason I mention that is I think, globally speaking, since this is such a relative-- this is such a frequent occurrence in haemato-oncology practice, febrile neutropenia, the management of that is key. So if you have practices that are not as optimal as they could be, and it's not that easy to deliver, because because I've looked at this very carefully in our centre, delayed treatment is not just going to lead to poor outcomes in terms of potential death or morbidity, but probably more antibiotic use. So I'm just getting at the complexity----

Q Are you suggesting that a theoretical mechanism by which there could be more infections is that the management of neutropenia is not as optimal as it could be?

A Yes.

Q Right. I suspect that-- Yes, well, what I'll do is I'll still ask the questions I was planning to. So I'm going to see if you have any views on

two extreme positions. So since we're looking at BSI, we'll look at water. So there are two extreme positions and I'll put them both to you. The first is that all of the environmental infections seen on this chart in that peak above what's the linear component are caused by the water system----

A Mm.

Q The contamination of it. The second is that none of them are. I wondered whether you would be supportive of keeping either of those in contention or would you exclude them as possibilities?

A Well, I'd exclude the second one straight away. I think it's inconceivable that no infections were caused by environmental organisms, some of which came from the water, I think, and also I think we know that from the-- the whole genome sequencing.

Q Well, there's been two.

A There's two cases.

Q To be fair, neither of them are in your data set.

A Quite possibly.

Q Yes.

A But for the first proposition, I would exclude that as well based on-- on what we are looking at in this-- the data, because what-- what I see here is that events-- When

the new hospital opens to the children, there's a downward trend that's already started before the hospital opens – so in other words, it started at Yorkhill – and continues down and then there's a change.

Q Yes. I mean, the----

A And it starts to go up, yeah.

Q Dr Drumright's evidence was that-- I put to her two possible scenarios.

A Yeah.

Q One is there's a real downward trend that starts in early '14----

A Yeah, yes.

Q -- and the other is that what we're actually seeing is the GAM model trying to smooth to fit the lots of zeros that you could see in late '15.

A Right.

Q And I think, to be fair to her, she wouldn't be drawn between the two.

A Okay, right. Yes, well----

Q But if you're, in a sense, excluding those two possibilities----

A Yes.

Q I mean, I assume that one of the reasons you exclude the 100 per cent is because some of these infections are going to be brought in on the patients because of the nature of the bacteria involved.

A Yes.

Q Yes. If we have a look at the non-environmental infections over the page.

A Mm.

Q For the non-environmental infections, what sort of counterfactuals-- Did you have any knowledge, other than what's covered in the letter of instruction, about the work of the CLABSI group in the Schiehallion unit in '16 and '17 and '18 to address line safety issues?

A Not at the time we did that work, although I do now.

Q I mean, it's mentioned in your letter of instruction.

A Yes, but we didn't see that, I'm afraid.

Q Right, okay. So, if we apply a slightly more extreme position, more options to make it more confusing, will any of the non-environmental infections have any prospect of being caused by water? I think I know the answer, but I'm keen to see what-- It sounds an odd question, but-- Can water have any role in this?

A I-- I have to defer to Professor Hawkey, because I-- I would like to say no, but I'm not sure if that's the right answer.

Q What I'll take is, "I'd like to say no, but I'll refer to Professor Hawkey," as your answer.

A Yes, yes.

Q So, if we look at the other candidates, what have you read about the CLABSI work since we instructed you?

A Mm. It's mainly the report by Ms-- I think it is Ms Jennifer Rodgers.

Q Ms Rodgers. You read a statement by her?

A Yes.

Q Or some paragraphs from her statement?

A Yes.

Q I think it's off the report----

A That's what it-- It's in one of the bundles.

Q Yes, yes, and then a presentation with some charts and----

A Quite possibly.

Q But you-- A little bit of information?

A Yes.

Q Do you see it's possible that work on the line safety being detrimental and then being addressed could form parts of the causes of this peak?

A Yes, and, given that this-- this is non-environmental

bloodstream infections, I would have thought it's likely that a-- line care is one factor and is probably an important factor in this, yes.

Q Right. If you go back to the previous page, I put this question to Dr Drumright, and I think it's useful. I'm going to use the word "some" to mean a non-trivial, relatively significant amount.

A Right.

Q But I'm not holding anyone to any more than that. If we think of all the counterfactuals and we think about the environmental paediatric BSI-- Now, of course, this is where you might say, "No, I would refer to Dr Drumright," but if we start with a contaminated laboratory sequence, is there any possibility that what we see here is consistent with some of those infections being caused by a laboratory problem?

A For the environmentals?

Q Yes.

A I-- I think that's unlikely.

Q Why do you say that?

A Well, whereas I can see for the non-environmental contamination, be it in the laboratory or more likely in the time of blood culture, is----

Q At the moment of taking the blood?

A Yes, is very probable-- It certainly could apply to some environmental, it could do, but I-- it just strikes me as less plausible, but it's not zero. So the sum is-- is----

Q So, when we take the issue of management-- I recognise you don't know anything about the management in this hospital. You've not really studied it, I'm assuming?

A No.

Q No. Do you have any experience of hospitals with dysfunctional management?

A I'd be surprised if most departments don't have an element of dysfunction, if I'm frank.

Q Indeed. So I'm wondering, do you think dysfunctional management would contribute to some degree to a peak like this?

A That's an interesting question. I'd say yes.

Q And why would you say that?

A The impact of-- So-- so the question here is that there are increased bloodstream infections, so serious problem in the Paediatric population. Why might that be and why might it be related to dysfunctional relationships? Because that's going to impact on the workings of the whole department. So it's not just the

dysfunctional members, it's going to be, are people able to deliver the things that they say they're going to deliver? So, for example, I just gave the example of rapidity of managing febrile neutropenia. Are they going to be able to do their line care in the way that they would like to do it? You know----

Q Is the microbiologist going to talk to the Infection Control doctor, that sort of thing?

A Yeah, what's the impact on staff morale? I mean, I can see many ways in which the performance of a department is going to go like this, and the impact of that will be seen in different places.

Q So, the reason I ask this is because I know, and I'm afraid that you don't, but I know and we've heard evidence about when the dysfunction started and how it went on and we might form a view when it was worse or when it was better. To what extent would you expect things to get better after a department stopped being dysfunctional? This is the sort of change afterwards----

A Yes,

Q -- the afterwards comparator.

A I think if it's----

Q Or was this on the edge

of----

A I-- I mean, I just feel, I can't say I've got any objective reply to this, it's subjective, is that it would take a little bit of time. I can't see how, if you've got significant dysfunction, which really means interpersonal relationships, and that's corrected, staff change, there's new leadership, it will take time for that to filter through to everyone who-- to then sort of come up again.

Q Now, with the antimicrobial use, I'll talk to Professor Hawkey in more detail, but am I right to think that we need to look at the actual microbes that are affected rather than a big class like environmental, or is it something that would a signal in a big subset like environmental paediatric BSI in your work?

A So antimicrobial use, antibiotic use, with the meropenem question, then-- I assume you already have that. So, there is an increase, or there appears to be a high number of infections attributable to Stenotrophomonas maltophilia. So, if-- if you want, there's-- there's evidence that says, whatever drove it, could it be related to changes in antibiotic policy? So that's a possibility that is addressed by Professor Hawkey.

Q I'll deal with that with him.

A Yes, yeah.

Q So we'd look at the connection between those things?

A Yes.

Q Right.

A The-- Also, just to mention, if you have a change in antibiotic policy-- and I'm not quite sure if the Paediatric department had the change of antibiotic policy around this time, but it may have included introducing more antibiotics, such as prophylaxis, that they didn't do before, and I have to say, I'm not clear on that.

Q So, should I be asking Professor Hawkey to see if there is a temporal connection between these sort of behaviours and the data?

A Yes, because it-- The reason I mention that is, if that was the case, you would see a decrease in bloodstream infections after introduction of something like prophylaxis that wasn't there before.

Q Right. Now, we move to the water, because I'm going to pass over ventilation in the next chapter-

A Yes, yes.

Q Remembering my definition of "some"----

A Yes.

Q -- is what we see here

consistent with some of the environmental paediatric BSI being connected to the water?

A Yeah, potentially yes, absolutely, yeah, I mean----

Q Why do you say that?

A Well, because it-- to me, it's inconceivable that the infections seen in this group of patients isn't a combination of different sources, of which, in my opinion, and I think most of the literature supports this, the vast majority is endogenous. So, in fact, translocation of the patient----

Q The vast majority, like 70 per cent?

A Yes, if not more. I mean, I-- Some, but "some" in the context of "a lot".

Q Yes.

A Yes, and then the question of, "Well, have some of these infections been acquired from the potentially similar or the same organisms, but from the"-- "from the environment of the patient's room or elsewhere?" Then yes, that's going to be the case in some cases, yes.

Q Okay.

A And I should say, as a normal part of the world that we live in, and I-- when I say that, I'm not suggesting it's because of failures, that's just, I think----

Q Well, you wouldn't know whether they're failures is because you've not looked at the----

A Well, exactly. Exactly, yes.

Q Yes.

A There would have to be something unusual to make you investigate for failure.

Q Well, indeed, and I'm not going to ask you about the material you didn't look at, that would be unhelpful. Now, final question-- No, I think you've answered that. My Lord, I think probably I've asked all the questions I need to ask. We didn't do it quite in the order I planned, so whilst I'm asking my colleagues to see if they have any questions, I might take a moment to reflect to see if I've got everything.

Questioned by The Chair

THE CHAIR: Right, yes. If Dr Agrawal will bear with me, can I ask him to look at what is bundle 44, volume 5, page 117? It's your 18 May 2021 report, Dr Agrawal----

A Yeah.

THE CHAIR: -- and that will be put on the for you. Now, can I just go through this to see if I understand it?

A Yes.

THE CHAIR: Now, I think you start with accepting, meaning to put forward as a proposition, that the absence of any special specialised protective ventilation on 4C, which is a neutropenic but not BMT ward, does give rise to a theoretical risk of increased airborne infection. So, that's Step 1. You accept, for the purpose of this section, that there is an increase in risk associated with the absence of protection in contrast with the protection that's provided in Level 5 in Barts.

MR MACKINTOSH: Now, if you're nodding and you meant to say yes, you need to say yes.

A Oh, sorry, yes. I was going to say yes----

THE CHAIR: Mr Mackintosh is more conscious of that than me.

MR MACKINTOSH: I think Dr Agrawal was going to say yes with a caveat.

A With a caveat, so that's----

THE CHAIR: Right, yes with a caveat. Well, do you want to add the caveat at this point?

A Yes. So, the caveat is that the-- the converse is not true; that's to say, with a compliance system, the patient is protected, it's just that they are less likely-- so the risk is less, but the patients in our unit, in these facilities, do get airborne infections, so----

THE CHAIR: Yes, okay. My fault in using the word "protection". If we take the word protection as not just total exclusion, but reduction in volume, quantity, whatever. Now, starting with that, because this is what you are asked to do in terms of your instructions, apply your mind to whether or not that accepted risk is material or not. Now, Step 1, what did you-- It's not your word, it's in your instructions. What did you understand the word "material" to mean?

A I-- I take that as two potential meanings from the way we perform the work, and I-- it would be fair to say that, in terms of the airborne infections, we'd be focused-- I focus on Aspergillus, that was my work. I take it (1) to mean that the risk is increased, so the risk itself, but the other way I've looked at the term "material" in its context is, is that risk manifest? I think I used a different word from your-- from your own earlier. So can we actually see that that actually leads to material----

THE CHAIR: If I was to suggest a definition of "material" as "makes a difference", would you accept that?

A I'd accept "makes a

difference", yes.

THE CHAIR: Right. Now, you go through a series of factors which I think persuade you that the accepted risk does not make a difference, right? Now, why does the fact that a different risk – that's the overall risk of infection – is mitigated in other ways make any difference to the materiality of the risk that you're considering in this section?

A Yes. So, I think the answer to that, your Lordship, is in the word "theoretical". So, again, it's what do we mean by the words we use? So, when I use "theoretical", I'm accepting that having a ventilation system that meets the standards that we are discussing is supposed to, theoretically, deliver a safer environment for the patient from airborne pathogens, but what I-- I haven't explained it here, but I reference the various sections of the report where I discuss why-- the various reasons why it doesn't do that and why it fails to do that.

THE CHAIR: Well, I can see that one might take the view that the provision of protective ventilation, such as you have in Barts and which I imagine has been installed at great expense, makes no difference. I can understand that as a point of view, but that doesn't seem to be the premise

which you have accepted for the purpose of 5.7, and the reason I'm emphasising this----

A Yes.

THE CHAIR: -- is that we may have to consider the impact of your statement that, accepting there is a risk, it's not a material risk.

A Hm.

THE CHAIR: So, going back, why does A make any difference to the risk which we appear to be discussing in 5.7?

A So----

THE CHAIR: It seems to me that----

A Yes.

THE CHAIR: -- what you're saying is, "Well, a different risk is mitigated in a number of ways," which may be true, but I don't see its logical connection with the exercise which we may understand you to be carrying out in 5.7.

A So this, I think, is slightly controversial, and what I mean by that is that we have various guidelines saying that ventilation systems achieve a certain degree of-- protects us, if I use that word for a second, from airborne pathogens. However, as I've said before, there is very little evidence that that-- that is the case in clinical practice or in studies.

THE CHAIR: If I may intervene, I understand you say that----

A Okay, sorry.

THE CHAIR: It's just the exercise that you appear to be going through here, which is, even if there is a risk----

A I see what-- Okay.

THE CHAIR: -- it's not a material risk.

A My apologies, I think I'm just struggling to----

THE CHAIR: Well, no, I'm sure it's my failure to make myself clear, because I don't quite see how factor A reduces a risk which you may have simply accepted for the sake of argument.

A So I think for the purposes of the point you're making, Lord Brodie, I accept that if there is a risk associated with a non-compliant ventilation system, then any mitigation of that risk doesn't reduce the risk associated with that failed or inadequate ventilation system.

THE CHAIR: Yes. Right. And the no clear evidence point which we've talked about may be true but doesn't mitigate the risk if you assume there is a risk.

A So that comes back to where I feel slightly uncomfortable, although I've accepted your position,

and hence my use of the word "theoretical" in my statement but that's fine. So I accept that.

THE CHAIR: Yes. I mean, this may seem very pedantic, but your report will be considered by lawyers, and lawyers tend to attach importance, maybe undue importance, to the words; and similarly, C, how does that impact on a theoretical risk once it's accepted, for the sake of argument, if we are looking at the situation of a patient who is in a neutropenic ward?

A So, the function of the ventilation system is to reduce the risk of particularly airborne pathogens. Clearly if the individual cannot be in that room for any reason, then immediately----

THE CHAIR: The utility is irrelevant.

A It's irrelevant, yes.

THE CHAIR: Right.

A And that is a part of the normal care of individuals in these rooms. So it's another one of the limiting factors of what can be achieved with ventilation.

THE CHAIR: Yes. I see it is a limiting factor, but again, it doesn't seem to bear on the materiality of the risk that we appear to be looking at in 5.7.

A So, I accept your point

because I've accepted it already at point A, but the impression I get listening to the argumentation is that the risk is going up all the time because we keep on listing points that doesn't affect the risk, but I'm not sure that's the case.

THE CHAIR: Right. And rather than unnecessarily take up time, can I put it to you that, accepting all the points that you've made, if for the sake of argument one attributes some value to protective ventilation, therefore the absence of protective ventilation can be described as introducing a risk, in fact, all the subparagraphs may be accepted as factually correct? None of them bear on the materiality of that risk.

A Okay. I accept that. Yes.

THE CHAIR: Thank you. Now, Mr Mackintosh, were you proposing to take a break to allow----

MR MACKINTOSH: I have one question which I realise I didn't ask.

THE CHAIR: Right.

MR MACKINTOSH: So, this was in the context, actually, of a couple of pages back, page 109. Remember we got into a discussion about dilution in relation to paragraph 4.1.3 in airborne pathogens?

A Mm.

Q What I didn't ask you was another question. You listed three sources of infections: patients themselves, airborne pathogens, transmission to (inaudible) visitors. What about the hospital environment itself? So one might include in that building works or infestation of animals, or-- Particularly building works seem to be a theme. Would that get chopped up and recompartmentalised into the other ones, or is this an additional possible source of infectious pathogens for this ward?

A Oh, absolutely. No, I mean, the hospital environment itself is undoubtedly a potential source for these pathogens.

Q Yes. I'm trying now to remember where in London your hospital is.

A It's right in the middle.

Q I appreciate that, but I was thinking more precise than that-

A It's next to the Old Bailey.

Q Thank you. Well, I know exactly where that is. So there's lots of traffic.

A Yes, absolutely.

Q Wildlife, people.

A Building works.

Q Building works. So does

that influence where a hospital is, the importance or otherwise of HEPA filtration, air changes, these sort of things?

A Building works definitely, so that's very well documented in the literature. Major building works, usually in the precincts of the hospital concerned, is a risk factor for fungal mould infections.

Q Do you do anything when building work starts near your hospital? Do you stop using the bays or anything like that?

A No, we're using the bays---

Q All the time.

A Yes, the unit is constantly full, so it's not a choice. So if there was a major building works going on, let's say, on the floor below, for example, or on the same floor somewhere, then that risk would have to be mitigated by our colleagues in infection prevention and control to address the predictable impact of increased disturbance to the environment by the building works, principally high levels of spores, supra levels of spores in the environment, and that could include something that we don't do routinely; the use of appropriate antifungal prophylaxis, for example. So that would be one approach to that.

Q One thing I wanted just to clarify, because I somehow got this in my head and I don't know if it's true and I want to be sure, your fifth floor, does it have any overarching ventilation treatment that distinguishes it from the rest of the hospital? Is it pressure differentiated from the rest of the hospital, the whole floor?

A Not the whole floor.

Q No, not the whole floor. I must have misunderstood. My Lord, that concludes the questions I have, but it may be that colleagues in the room have questions. We might have a 10-minute break to allow for-

THE CHAIR: Dr Agrawal, our procedure is to check if there's any questions which legal representatives wish asked, so if I can invite you to return to the witness room, I would hope it's no more than 10 minutes.

(<u>Short break</u>)

MR MACKINTOSH: My Lord, two questions.

THE CHAIR: Two questions, Mr Mackintosh.

A Good afternoon again.

MR MACKINTOSH: Two questions, I understand, Dr Agrawal.

A Okay.

Q So the first question, I think, is merely factual. Since we've taken a lot of evidence from you about the nature of the ventilation system on your fifth floor at Barts---

A Yes.

Q -- is that fifth floor 100 per cent mechanical ventilation or do you open the windows?

A There are no opening windows in the hospital.

Q In the whole hospital?

A Not that I'm aware of.

Q Not that you're aware of, okay.

A No.

Q The other question is this. So in the context of either BMT or haematology wards, so either 4B or 4C----

A Yes.

Q -- or in your world, 5C and 5D, in this hypothetical situation, if a ventilation system on such a ward has not been validated against HTM or SHTM guidance, is that something that ought to form part of the patient consent process before procedures take place?

THE CHAIR: When you talk about patient consent process, what do you mean?

MR MACKINTOSH: Well, what I mean-- to help you, what I think I mean, so you can tell me if I'm wrong, Dr Agrawal.

A Yes.

Q Is that when-- I understand it, when a patient has a procedure----

A Yes.

Q -- they will be taken through a consent process, which will inform them of the risks, the benefits and such things and, depending what it is, it may be a long or short process. That's what I mean, but if you want to use a different phrase, that's what I'm trying to get to. The idea where you obtain informed consent from a patient.

A Yes, yes. So it's a hypothetical scenario----

Q Yes.

A -- and I think the answer to that would be it depends on what the consent is for. So if the consent is for the highest risk procedures one can imagine, so let's say myeloablative stem cell transplant. So intensive treatment prior to transplant. I think that individual should be referred to another hospital that has functioning systems.

Q Well, I'm not saying the systems aren't functioning. I'm just saying they're not validated.

A Oh, they're not validated.

But that comes back to the point we had before. So I would need to know something.

Q Well, okay, I'll put the scenario to you. So, given that the hospital ventilation systems were not validated----

A Validated, yes.

Q -- one can make the inference that at some point after it opened, adult BMT and haematology patients might well have been in an unvalidated ventilation space.

A Right.

Q At that point, should they have been informed of the lack of validation, or is it an implication that it's always validated?

A I'm actually just hesitating because I think it's a very interesting question indeed, and I'm just struggling to collect my thoughts on it.

Q Before you finish, I'll add an extra fact which I think you need to know. So at the time we're talking about, which I think in this case will be the five weeks in the summer of 2015 in Ward 4B, and for some time, and I now can't remember how many years, but some time after the opening of Ward 4C, I don't think the treating clinicians knew the system was not validated----

A Okay, right.

Q -- albeit that neither did they check.

A Right.

Q So, in that context, can you assist us in whether they should have actually consented their patients?

A So, firstly, I wouldn't expect the clinicians to address the question, "Has this been validated?" I mean, that would be an assumption.

Q Yes.

A You know, that isn't the way most clinicians would be thinking, I don't think. You'd have to firstly make a decision, "Are we going to use the facility as is unvalidated," so you need to know, and once you know then you would need to do a risk assessment, and if a risk assessment is rather like points where I was just discussing with Lord Brodie then, having attempted to mitigate risk, yes, that patients should be consented on the basis that the room that they're in, the environment that they're in, is not validated.

The challenge I have with this is that we live in a world where that is generally regarded as the right thing to do. That's to inform an individual of the risks of any procedure. There is also-- I mean, I also struggled to see how that necessarily helps the

individual as there's nothing they can do about it.

If they need a bone marrow transplant, they need a bone marrow transplant, so are we not just engendering increased anxiety, in fact, a severe degree of increased anxiety, plus with my position that there are effective ways of mitigating that risk, so the only proviso I have around this is knowing that the system is not making things worse, but we don't know that. So, that would be my one big caveat: if I don't know the system is not making things worse, then I would have-- I wouldn't proceed with high-risk procedures in that environment. So, I come back twice: I would need to know something to convince me that there wasn't-- there weren't Aspergillus spores being pushed into the space. I would need some reassurance, but if I had that reassurance, then I think consent would be -

Q So, would you need-- if there was information about----

A Spore counts would----

Q -- spore counts----

A Yes, for example.

Q -- that would inform that process?

A Yes.

Q Do I get the impression from what you're saying that if you discovered that there were spore counts in an unvalidated system, your reaction is not to consent the patient but to send them somewhere else?

A Yes, if----

Q In the most vulnerable cases.

A -- then I'd be concerned with actually causing harm, yes.

Q I think I----

THE CHAIR: Could I just explore what might be a consequence of that? I understand or at least I think I understand what you said about the practicalities about consent, but you started that section by recognising the ethical obligation of any clinician to obtain consent for certainly for a procedure but possibly for placing the patient in a particular location. You said – and it seems to me entirely reasonably – that a clinician moving into a new facility would assume that the facility is as you would expect the facility to be and that had been confirmed by, in the case of ventilation, validation----

A Yes.

THE CHAIR: -- and you couldn't blame a clinician who didn't assume otherwise. Now, does that have the consequence that there is an obligation on those managing the

facility to ensure that there has been validation in order to, as it were, support the clinician who assumes that the facility has been validated?

A Yes, I need to just caveat one thing I just said and that is it would not be-- the clinical team would not be without any information because the whole JC process – we talked about JC earlier – very much addresses these issues as well and so there would be a director of the facility who would be the JC representative in that facility and it would be their role to know this information.

MR MACKINTOSH: Whether they meet the validation?

A Yes, yes.

Q Has that been JC policy for a long time or is that an innovation in recent years?

A Well, JC doesn't mandate that you need such a facility in the first place, so we come back to-- --

Q Well, that's what I mean because, because I know that, how does a JC director----

A But the point is----

Q -- acquire a duty to find out something they're not required to have is validated?

A So, we have this apparent dichotomy that the regulatory

standards don't mandate this level of protection and yet clinically it's what all bone marrow transplant centres have in the UK, as far as I'm aware. So, if you have such a facility, then it should meet the standards that the specifications require, that the regulations require, and hence I would expect the director to be aware of these things before it was commissioned and opened, yes.

Q Thank you. (To the chair) Thank you, my Lord.

THE CHAIR: Thank you, Dr Agrawal. You have given evidence over what's been quite a long day and thank you for that, and thank you also for the great deal of preparatory work that you have put in in order to prepare for giving that evidence. So, you're free to go, but you go with my thanks. Thank you very much.

THE WITNESS: That's very kind of you. Thank you, Lord Brodie, and thank you, Mr Mackintosh.

(<u>The witness withdrew</u>)

MR MACKINTOSH: Before we stand, Tuesday morning, my Lord, ten o'clock, Mr Mookerjee.

THE CHAIR: Right. Well, thank you to the legal representatives, and we will see each other again on

Tuesday morning at ten. Have a good weekend until then. Thanks.

(Session ends)

17:05