Scottish Covid-19 Inquiry

Day 2

July 27, 2023

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(10.00 am)

LORD BRAILSFORD: There are two things that I want to do before we start formally.

The first one is a little formal statement by myself.

Now, I realise that some of you attending or watching yesterday may have expected to see or hear more in the way of acknowledgment of the impact the pandemic had on individuals and families. The reason that didn't happen is that this presentation was not designed as a formal hearing of the Inquiry, but as a session on epidemiology, on science. The focus of this session is to help those interested in the work of the Inquiry understand more about the underlying science which will form the basis of much of our investigation and, of course, deliberation throughout the coming no doubt many months.

On 28 and 29 August, as I told you yesterday, we will have a further hearing, or we will have a hearing, and the focus will then shift from the science to the people affected by the virus and the strategic response to the virus. On those days, we will hold what we consider to be our official preliminary hearing, and that will constitute a formal opening to the Inquiry's

evidence—gathering and hearings to follow, which I think again, as you all know, will commence late in October.

At that stage, in August, there will be an opportunity for core participants to the Inquiry —— including, of course, bereaved families, care home relatives and others affected —— to participate. We also plan to show a film at that hearing to highlight the impact of COVID on people, to signal the beginning of our hearings dealing with impacts on the people of Scotland. I should say that I'm grateful to those families and individuals who have provided photographs which we've used in that film, and I thank them for their contribution and their own going participation in the Inquiry. They are and will continue to be at the forefront of my mind when conducting the work of the Inquiry and when it comes to deliberating and considering the report.

As I said yesterday, core participants will receive a communication from the Inquiry next week, providing more and detailed information about what to expect from the August hearings and how they should participate therein.

So I hope that clarifies matters.

That's the first thing I wanted to say.

The second thing is that Dr Croft has kindly told me

that he's realised, on consideration last night, he made an error of a technical nature, and he would like to correct that. I think that's entirely appropriate.

Can I simply ask you to explain what the error was and then correct it.

DR ASHLEY CROFT (continued)

THE WITNESS: Yes, thank you, my Lord.

If I could ask you, my Lord, to turn to page 463.

LORD BRAILSFORD: 463.

A. In fact, before that, let's turn —— so sorry. We'll come back to 463.

Let's turn to page 659. This is about gargling to prevent COVID—19. We were just using it as an example of Cochrane reviews.

LORD BRAILSFORD: I remember that. 659, did you say?

A. 659.

LORD BRAILSFORD: Oh, yes. The Almanza—Reyes and Gutiérrez—García papers.

A. Of course, yes. That's right. So we started yesterday —— well, we began by talking about Mr Gale's hypothetical acne, and that Mr Gale went to his GP and he said, "Right, doctor, what's the risk of my being cured of my acne, my hypothetical acne, if I take this antibiotic?", and the doctor said, "The risk is —— in other words, the likelihood of you being cured, is 6,

60%, because in the trial, of those who took the antibiotic, 6 got better and 4 didn't get better. So 60% likelihood. What's the risk of your getting better if you don't take the antibiotic?", and if you remember, the risk was 30%. So, therefore, the risk ratio was 2, meaning he was two times more likely to ——

LORD BRAILSFORD: Two times ——

A. Yes.

Right, so applying that same understanding to the gargling, if you remember, Almanza—Reyes and Gutiérrez—García were two randomised controlled trials, both in Mexico, and they both involved healthcare workers. They were quite small numbers, but produced a very powerful effect from gargling.

The combined impact of —— the combined pooled effect measure gave a risk ratio of 0.07 —— could you see that in the third line down? —— with a confidence interval of 0.02 to 0.23.

LORD BRAILSFORD: Yes.

A. In the heat of the moment, I said that means they're 93% less likely to get COVID—19 if they were gargling than if they weren't, and that's incorrect.

These were healthcare workers working in Mexico in intense environments where the transmission of COVID—19 was very intense, so they were obviously quite alarmed

and worried, and they did this gargling. The first group were gargling with a solution of silver nanoparticles, and that was compared with a group who just did conventional gargling. We don't know what with.

The second group, Gutiérrez—García, they were all wearing PPE, which is interesting, both arms of the trial, and the experimental arm were also gargling with this neutral electrolysed water, and they did that three times a day, and the other group did it for three times a day.

The first group were followed for nine weeks, I think, and the second group were followed for two weeks. So they weren't very long trials. Again, the pooled effect measure was 0.07. So what does that mean? What is the risk ratio? It's actually 14. So it's 100 divided by 7.

LORD BRAILSFORD: Okay.

A. So in other words, the people who were gargling were 14 times less likely to acquire COVID—19 than the doctors and nurses who weren't gargling, which is impressive. You don't often get such good effect measures, but this is, I think, indisputable, because they were randomised controlled trials, they were well done, and the heterogeneity was 0, the I—squared was 0%,

so it was reasonable to combine these two trials. And the confidence interval there —— you can see that along that dark, bold line —— ranged from 0.02 to 0.23. So, therefore, we can have 95% confidence that the true measure lay somewhere between 50 times more protected, which is 0.02, they might have been 50 times less likely to acquire COVID—19, or they might have been four times less likely to acquire COVID, which is 0.23. So that, I think, is a correct interpretation.

It might have been helpful if Jefferson had given some interpretation, but they put this forest plot in because they obviously found it interesting, but they were so busy talking about other things, they didn't interpret it.

So that —— just to finish, my Lord —— tells us a couple of things. Firstly, there are very effective measures that one can apply to prevent COVID—19, such as this one; simple, effective, cheap.

Oh, yes, in the Gutiérrez—García, the control group were wearing full PPE, but nevertheless they still had quite a high number of cases of COVID, 10 out of 79. So it shows there may be some protective effect, but the ones who were wearing full PPE and also gargling only had one case out of 84. So much better to be gargling.

It shows you can do randomised controlled trials

even at times of high COVID intensity, as long as they're designed correctly. They don't have to be expensive. The second group said they had no funding. The doctors and nurses just decided to do it amongst themselves. So this pre—empts the possible argument that might be used by the policymakers: well, we couldn't do research at this time because everything was so fraught. But people were doing good trials. We may later on discuss the Danish trial and the Bangladesh trials that were done in this time.

So my last point —— sorry to go on —— is: what made these doctors and nurses in Mexico do this?

LORD BRAILSFORD: Quite.

A. Which seems odd, and the answer must be: they must have read the science. They must have read Jefferson 1, which is the precursor of this, and that was the original systematic review that came out in 2011.

If we look at Jefferson 1, again, there's a surprising —— page 463. That's the third forest plot down called, "Analysis 1.9 ... Case—control studies, Outcome 9 Nose wash". They must have looked at that and thought: that's pretty impressive.

So they are looking at two studies there, Chinese studies, that were done in China during SARS outbreaks, mini—outbreaks.

LORD BRAILSFORD: Yes.

A. And these are case—control studies. So they're not as powerful as randomised controlled trials. Case—controlled studies are what they call quasi—experimental studies, and they level IIb evidence in that hierarchy.

But, essentially, what happened here was that Chen and Liu, Chinese investigators, they compared cases, people who had got SARS and then they recovered —— they were all healthcare workers, we think —— and they matched them with equivalent age—matched and sex—matched healthcare workers who didn't get SARS, and they worked out: what was it that enabled one lot not to get SARS, and the first lot to get SARS, and they found a powerful protective effect from nasal gargling or nasal washing of some sort.

Interestingly, the total —— the diamond there shows an odds ratio which is similar to the risk ratio, which is 0.3. So it's the same kind of order of magnitude as the Mexicans found later on.

So that, I think, explains all of that, and it's of interest, and I think of practical importance as well.

Thank you, my Lord.

LORD BRAILSFORD: No, thank you very much indeed.

Yes, now, Mr Gale, when you're ready.

Questions from COUNSEL TO THE INQUIRY (continued)
MR GALE: Thank you very much, my Lord.
Doctor, we had reached, at the conclusion yesterday afternoon, page 11 of your report and section 2.3, headed, "What are coronaviruses?"
I want to take quite a lot of this short because it's there for everybody to read ——
A. Yes.
Q. —— and those who wish to investigate it further will have the opportunity to do so.
I think you indicated towards the bottom of page 11, last paragraph, that the coronaviruses are further categorised according to a classification scheme developed in the 1970s by the Nobel laureate David Baltimore.
A. Yes.
Q. Then there are four features: the molecular architecture, their genome, their replication strategy and whether, in the case of RNA viruses, they are positive or negative.
A. Yes.
Q. Now, I'm slightly interested in what is said in the second paragraph on page 12, where it is said that:
"RNA viruses [which I think we know COVID is one of those viruses] ... have high error rates, with genomes

diverging by as much as 2% in the course of a year — 1 million times greater than the divergence rate of eukaryotic cell genomes."
Now, can you translate that for me, please?
A. Thank you, Mr Gale. That's a direct, word—for—word transcription from a textbook, and it's in jargon, for which I apologise.
Sorry, I have lost the place on the page. We're on page 12 ——
Q. We are on page 12 of your report, the second full paragraph on page 12. "RNA viruses", it begins.
A. Right.
So RNA viruses are single—stranded nucleic acids, and so they don't have that same, shall we say, property of DNA viruses that has are double—stranded, and DNA viruses, as they replicate, are less likely to have slight changes in their molecular structure. So that's my understanding of this concept.
So because they're more fragile, every time they replicate —— and that's their whole purpose in existence, which is to replicate —— there may be slight changes in the molecular structure, particularly in the ordering of the nitrogenous bases we were talking about yesterday, the guanine, adenine, cytosine and uracil in the case of —— so there's a slight ordering in those

bases. The code for which they exist is different. It's a bit like a computer code. It's similar to a kind of virus that gets into your computer, makes it behave slightly differently.
So the genome is the totality of their genetic information, the total genetic profile, and that can change with viruses, as said here, by 2%. So in the course of one year of continuous replication, you may end up with viruses at the end that are 2% different to the original parent virus. That's how I understand this.
Quite what that means is hard for us to understand, but the reference I have taken that from goes on to say that this is 1 million times greater than the genetic transformations that would be seen in eukaryotic cell genomes. Eukaryotic cells are those that are found in advanced organisms such as humans and mammals, and so therefore our genomes are also undergoing slight mutations all the time, or slight changes, but nothing like the rate we see in viruses.
Q. You go on in that paragraph to say:
"Many of these viral mutations are non—functional ..."
A. Yes.
Q. And then you qualify that:

"... but some will allow the virus to evade host immune responses and medical therapies."
A. Yes.
Q. Is this effectively a mutation?
A. Yes. Yes, that is a mutation, yes. Every change in the genetic structure will be a mutation. But in general, mutations —— and we undergo mutations ourselves, particularly those, for example, on the skin that are exposed to ultraviolet light. They may mutate. Some of those mutations in ourselves may cause the cells to become cancerous, to lose their normal properties.
So most mutations don't result in a functional advantage for the cell or the organism, but some of them might. Some of them might make the virus more pathogenic, more easily transmissible and, therefore, if they're more easily transmissible, they may get into cells more easily, they may evade the immune response more readily, and, therefore, they become more pathogenic.
Q. Perhaps, to put it simply, they become a moving target so far as human ——
A. Yes, correct.
Q. —— immune responses and medical therapies are concerned.
A. Of course. We see the same phenomenon with influenza viruses, which again are RNA viruses. They are around

us all the time. And when conditions are particularly good for their transmission, which means the winter, they will cause disease, and all the time influenza viruses are undergoing slight mutations. It's called antigenic drift. So there's a tendency for them to always be a bit different.

Occasionally, influenza viruses will undergo a major mutation, and that's termed antigenic shift, and that gives rise to a new subtype of influenza virus, which often can be very dangerous, because the new subtype may be one that people have never been exposed to because it's so different.

The same phenomenon, as I understand it —— I'm an epidemiologist, not a virologist —— is seen in coronaviruses where, as you say, Mr Gale, they are a moving target.

Q. Could you just read the final paragraph, so we have it in the notes, that begins "In summary".

A. Yes:

"In summary, coronaviruses are enveloped, single—stranded, positive—sense ribonucleic acid viruses that infect mammals and birds and that typically (but not exclusively) target the respiratory tract of their hosts."

Q. We move on now to what diseases are caused by

coronaviruses. I think some of this we've already looked at in the context of your appendices on SARS and MERS.

A. Yes.

Q. I think we can take that as read.

I think yesterday you indicated that there were seven known human coronaviruses, and that is set out in the table on page 13. I think the first four of those coronaviruses are the ones that you didn't specifically refer to, but I think they're there just simply for the record.

A. Yes.

Q. The ones with which you were concerned are the last three: MERS, SARS and SARS—CoV—2.

A. Yes, those are the novel coronaviruses that emerged —— we don't know with certainty how, but more likely than not as a result of some kind of animal reservoir spilling over into humans.

Q. Can we move over now to 2.5 at page 14. There we'll see, again, this is some material that we've already looked at, and, as you say, the term is "architecture" ——

A. Yes.

Q. —— and that is shown in the figure.

Again, I think one of the things that probably most

of us do remember from general presentations during the COVID pandemic was the significance of the spike, hence the name "corona".

A. Yes.

Q. Can you just explain what the spike is and what it does?

A. Yes. The spike of SARS—CoV—2 —— this picture shows SARS—CoV—2, which is very similar to the original SARS virus, the coronavirus that caused SARS. First of all, it's quite a large virus, so it's quite complex, so potentially there's the possibility of it changing in unexpected directions and causing problems with an immune response. But it's surrounded by these spike proteins which project from the envelope. The spike proteins are the means by which —— the surface proteins by which the virus achieves entry into the target cells of the host, which may be an animal or it may be human, and how the virus does this is through identifying and fusing with a receptor, which is a protein, on the surface of the cell called the ACE2 protein. So the spike protein attaches to that receptor, undergoes a slight transformation, and then the virus can then enter the cell through that entrance.

So I consider it's a little bit like a Yale key, which will fit into a lock, it won't into other locks, even though there are other locks, and it will open the

lock and then you can go in.

Just to go back to the previous table, the receptor that SARS—2 had —— so the table on the previous page, page 13. That's the table of the seven human coronaviruses.

LORD BRAILSFORD: Yes.

A. That's right.

So if you look at the right—hand column, my Lord, you can see that SARS—CoV—1, which was discovered in 2003 and caused the epidemic of SARS, has the same receptor, the ACE2 receptor, angiotensin—converting enzyme 2 receptor, as the SARS that we're preoccupied with now, and that was quite helpful because a lot of research had been done on the ACE2 receptor, exactly how SARS—CoV—1 interacted with that. So quite a lot was already understood about, if you like, the dynamics of infection with regard to this novel coronavirus.

Q. I think we can also see in that table that the virus identified in 2004 had the same entry receptor.

A. Yes, that's right, yes.

Q. Right. Can we go back to page 15, please.

A. Yes.

Q. I'm interested —— again, just taking various passages from what you say —— in what you say in the second paragraph on page 15. What you say there is:

"The mutations that SARS−CoV−2 accumulates facilitate the phenomenon of immune escape − meaning, that re−infection with the virus can occur after natural infection, and also after vaccination."
A. Yes.
Q. So is that essentially saying that we can expect to be infected with COVID more than once, even if we are vaccinated?
A. Yes, just as we can expect to get flu more than once. But there may be some degree of −− continuing with flu, with every flu season, there will be a slightly different flu variant, flu virus variant; in fact, there are a number of influenza virus variants circulating all the time. It will be slightly different to last year's because the virus is mutating all the time.
Q. Yes.
A. But often we will have a degree of cross−protection from the time that we last encountered that flu virus, so it won't affect us as badly.
The same we would expect to find with SARS−CoV. If we've acquired it, especially if we've acquired it naturally, we may well acquire it again after a period of perhaps a year, but we are likely to not have such significant illness the second time round.
Q. I think that's what you say in the following paragraph.

A. Of course, here I'm quoting the indisputable scientific literature. That's my reading of what is set out as indisputable facts.
Q. Yes.
We move on to 2.6, and we are now looking at the spread of the virus. You concentrate, first of all, on airborne spread versus droplet spread of respiratory pathogens −−
A. Yes.
Q. −− and the distinction you then set out in the following paragraph. I think you say that the distinction is based on the size of the infecting particles.
A. Yes.
Q. And in airborne spread, very small particles −− and you've given the dimensions −− and then you move on to droplet spread.
In very simple terms, what is the difference between airborne spread and droplet spread?
A. Well, I'm not convinced there is a great difference, but enormous arguments rage about the difference and what it means. But, basically, they're transmitted through the air, but some particles are heavy −− I guess they're surrounded by mucous −− and they fall on the ground around you, and others are very small and light and they waft through the air for some distance.

I guess, in practical terms, if most of the transmission is occurring through the droplets that are falling around you, then you want to focus on the immediate environment, whereas if most is occurring at a distance, the immediate environment may not be as important.
But with most respiratory viruses, it does seem to be the case that it's a combination of the two, the airborne and the droplet spread, that results in transmission.
Q. I think in relation to airborne spread, you do say that:
"Being very small, the infective particles can remain suspended in the air for long periods of time and travel long distances and be inhaled into the air passages of potential new hosts."
A. Yes.
Q. Droplets, as you say, I think, to put it crudely, tend to be perhaps more localised.
You make reference in your discussion on droplet spread to droplets contaminating environmental surfaces −−
A. Yes.
Q. −− or inanimate objects, that is fomites. Now, I think you do define −− we don't need to look at it −− what "fomite" means.

I think, again, we have probably all heard during the pandemic of the need to wash surfaces and handrails, etc. Is that what you're talking about?
A. Yes. Yes, that would seem to be quite a logical thing to do and one that doesn't involve a great deal of inconvenience or expense. It's all part and parcel of basic hygiene.
Q. The problem obviously is that if somebody touches somewhere where there is an infected droplet, and then puts their hand to their mouth −−
A. Yes, indeed.
Q. −− that is a potential transmission.
A. Indeed, and your mask might become a fomite if either you have put infectious droplets onto it by breathing out or you've breathed in infectious droplets. It potentially could be a fomite. So masks have to be worn correctly and disposed of correctly.
Q. I think then, in page 16, going on, you helpfully set out:
"... the range of infective particle size (and hence the predominant mode of spread) will be affected by factors such as ..."
And you list them: the volume, the character of the secretions, the extent to which droplets are converted to aerosol particles by evaporation.

That's something perhaps you might explain: how is a droplet evaporated and converted into an aerosol particle?
A. Well, if droplets are mainly fluid and they are landing on a surface, especially if they're exposed to the sun, they will evaporate in the way that fluids do.
Q. Simple as that.
A. They end up as minute airborne particles.
Q. Then you refer to the duration of airborne suspension ——
A. Yes.
Q. —— which is influenced by environmental factors, and we will come on to those environmental factors in a minute.
A. Yes.
Q. But that includes temperature, humidity and prevailing air currents.
A. Yes.
Q. And then, finally, the distance travelled, which again is obviously influenced by environmental factors.
A. Yes.
Q. If you just read from the bottom of page 16 to the end of that section, doctor, so we just have, effectively, your summary.
A. Thank you. So really this is the summary of the, I think, undisputed knowledge about transmission:
"In reality, both the size of respiratory particles

produced by an infected person and the distance they can travel are likely to fall within a spectrum; for any given respiratory pathogen, therefore, disease acquisition may occur through both the airborne and the droplet mechanisms of spread."
Q. And, obviously, the same transmission can be occurring at the same time.
A. Indeed, yes.
Q. You then go on to talk about what you say are the aerodynamic factors.
A. Yes.
Q. I think you begin by prefacing this with the word "Perplexingly".
A. Yes.
Q. You say:
"... and although COVID—19 is generally thought of as an acute respiratory illness, there are very low levels of SARS—CoV—2 in the respiratory tract during the early phase of disease ..."
A. Yes.
Q. "... this is the case with all coronavirus infections."
Is there an explanation for that?
A. Well, it does seem to be the case that the virus particles are particularly trapped in the nasopharynx, which is the next paragraph, and they don't go straight

into the lungs, which is the mode of transmission with some airborne diseases, for example TB or asbestosis. You have to be a particle that wafts straight into the lungs and causes the problem there.
So the nasopharynx is acting, it would seem, as a kind of filter and first line of defence by collecting all these virus particles, and then, from there, they disseminate to the rest of the body.
That ties in with what we were just talking about, which is nasal washing and nasopharyngeal gargling.
Q. If you go on, on that page, to the penultimate paragraph:
"SARS—CoV—2 is primarily transmitted from person to person following close ([less than] 6 feet, ~2 metres) exposure to respiratory fluids carrying infectious viruses."
Then you give examples; very simple examples of somebody breathing, singing, talking, coughing or sneezing, that:
"... release large infective particles (droplet nuclei) into the air; these particles may land on the exposed mucous membranes of a ... host, causing infection."
A. Yes.
Q. Then you go on to infection from touching contaminated

surfaces, which you say is also possible, and then perhaps you would just read what you say in the final paragraph of this section at the top of page 18.
A. So the paragraph starting, "Finally"?
Q. "Finally ..."
A. So:
"Finally, SARS—CoV—2 exposure can occur when very small infective particles (aerosol particles), suspended in the air, are inhaled directly. Aerosol—generating procedures commonly take place in hospitals, and in dental surgeries; hospital procedures in this category include (but are not limited to) tracheal intubation, manual ventilation, non—invasive ventilation and the use of certain high—flow oxygen treatments."
Q. Thank you.
You then go on to talk about something you prefaced in the previous section, the environmental factors, and I think we can read what you say there.
I think, on a practical level, it's quite interesting what you say, that in indoor settings, transmission is thought to be much less common.
A. I thought I said the opposite.
Q. I'm sorry.
A. Yes, the opposite. Yes, it's more common in ——
Q. Yes, in outdoor settings, it's much less common.

A. In outdoor settings, much less common, yes. It is. And this would be because the viruses are naturally fragile and they are degraded by ultraviolet light coming from the sun, and fresh air disperses them even more.

Of course, it immediately calls into question: what do you do about people outdoors? And certainly in England, the -- well, by way of comparison, in Scotland during the lockdown, the golf courses were open; in England, they were all closed all the time during every lockdown. To me, that doesn't sound logical, because on a golf course you're in the fresh air, you're physically distanced from other people, and the risk of transmission must be very low.

Q. I think there was talk in the very early days of the pandemic about the -- to use a buzzword -- super-spreader event, or potential super-spreader event, of the Cheltenham race meeting, which I think occurred very early in the pandemic.

A. Yes.

Q. Now, obviously that was --

A. Out of doors, yes.

Q. -- outdoor, but it was presumably a lot of people in close proximity to each other.

A. Yes. Yes. Sure, yes. Indeed. That foreseeably could have been a super-spreader event, yes.

Q. Thank you.

You make reference at the bottom of page 18 to outbreaks on buses and trains.

A. Yes.

Q. You then make reference to what are termed "attack rates". Again, I think this is a term you define.

A. Mm-hm.

Q. But it's the proportion of people exposed who go on to develop infection, and you say that that has been as high as 36%. That's in relation to buses. I think you give the citation for that in your footnote. Then, similarly, you do that with trains.

Then on the top of page 19 you refer to transmission during airline travel, and I think you say it can be as high as 60% in subsections of an aircraft. It probably is fairly obvious as to why that would be.

A. That section was probably packed. But, equally, it's interesting that aircraft flights might result in a 0% transmission rate.

Q. Yes.

A. So it has to be interpreted cautiously, that piece of information.

Q. I think you then mention Wuhan --

A. Yes.

Q. -- and that the experience in Wuhan shows that

transmission can be massive in a short space of time, with thousands of new patients diagnosed daily.

A. Potentially, yes.

Q. Then you have a section on patient-specific factors. In this section, you introduce a qualification on, I suppose, the degree of infection, and you say that:

"Individuals with mild to moderate COVID-19 may shed infectious virus and respiratory secretions for up to 10 days following the onset of symptoms ..."

Just so that we understand, can you put in simple terms what you mean when you qualify the infection as being mild to moderate?

A. I can't, because it's a subjective term, but what I presume is meant here is -- generally, severe illness is generally categorised as one where the person has to seek medical advice from a GP or from a hospital. So mild to moderate, I imagine, would be the person being at home, not necessarily seeking medical advice, and self-medicating, or maybe not even self-medicating, just -- but, on the other hand, not entirely well.

Q. I think you say that that fact was known from early on in the pandemic.

A. Yes, it was, and it was a factor that was also a feature of SARS, so it was logically a feature also of SARS-CoV-2.

Q. I think you then go on to say:

"Immunocompromised people with severe disease may shed the virus for longer (potentially, for up to [10] days)."

A. I think actually that's 20 days.

Q. Oh, I'm sorry.

A. I believe there's a transcription error which I corrected. But it's longer because they're taking longer to clear the virus.

Q. Yes. So that's the same figure as the one you've given before.

A. Oh, right.

Q. So it must be different. It must be 20 days.

A. Yes, of course. Yes, I beg your pardon.

Q. Then, again, something I think we probably can all remember from the general presentations during the pandemic. You say that:

"... the concentrations of SARS-CoV-2 RNA are highest one day before symptoms appear, leading to extensive spread of the virus by asymptomatic people not yet showing any signs of illness."

A. Yes.

Q. Is that effectively one of the dangers of the situation?

A. It is, yes. Yes.

Q. I think in the next paragraph you do say that some

people may remain asymptomatic, albeit having acquired the virus and acquired the infection.
A. Yes, especially if they're very young and healthy.
Q. Yes. I think you make the point that, in the case of infected children, at least one-third are likely to remain asymptomatic during infection.
A. Yes.
Q. You then give the example of the Diamond Princess cruise ship --
A. Mm.
Q. -- which was quarantined -- I think we all remember the footage on the television of this -- off the coast of Japan.
A. Yes.
Q. What subsequently emerged from the research was that 52% of the 634 people who were laboratory-confirmed cases were initially asymptomatic --
A. Yes.
Q. -- and most began to show symptoms, but an estimated almost 18% of infected individuals never showed any symptoms of infection.
A. Indeed, even though, one imagines, they were middle-aged and elderly passengers, but they were still asymptomatic.
Q. Right. Thank you, doctor.

The next section of your report, 2.10, deals with the origins of COVID-19.
Now, this is, I think, an area that probably everybody is now -- or at least informed readers will be -- relatively familiar with, and I'm not going to take you through it in any detail.
There are two things I would like to, however, ask you about.
A. Mm-hm.
Q. At the top of page 21, you say that:
"It is now believed by many that the novel Wuhan virus was transmitted to humans via horseshoe bats ... and potentially other intermediate hosts, to whom individuals may have been exposed at wild food markets in the centre of Wuhan ..."
I think that was what was initially thought.
You also say:
"... an alternative theory is that the virus resulted from a 'lab leak'."
A. Yes.
Q. I think there is a lab in Wuhan which may have been seen as being the source of the infection.
I take it you're not expressing any view on which you consider; you're just putting those forward as the two alternatives?

A. Indeed, those are two theories. I was a bit sceptical about the lab leak theory, but it does seem that the SARS -- if you remember, SARS 2002-2003, there were several subsequent mini-outbreaks, and three of those -- we touched this yesterday -- seemed to come from lab leaks of some sort, so clearly it's possible.
Q. I think, helpfully, we can see the progression over about 20 days of the infection of persons with the virus shown in the maps that you've shown on that page, and we can all look at that.
A. Yes.
Q. The other point that I would like to take from this section on the history of the virus is what is said at page 22 at the top, where you refer to the basic reproductive rate, which I think we probably all remember being referred to as the R number.
A. Yes.
Q. I think you indicate that the original wild-type strain of SARS-CoV-2 was estimated as having an R number of 2.8.
A. Yes.
Q. And, as you say:
"The R [number] denotes the number of persons directly infected by an infectious case during his or her entire infectious period, on entering a totally

susceptible population."
Then I think you've indicated, by comparison, the R of seasonal influenza is typically between 1 and 2.
A. Yes.
Q. So, in simple terms, the R number represents the number of people who would be infected by one person.
A. Yes.
Q. Sorry, having said there were two things, there are in fact three things I would like to just take from this section, and this further matter follows on from what we've just discussed.
If you go to page 25 at paragraph 2.13, you will see you deal with the emergence of variants, late 2020, and I think you say there that:
"... new variants ... had emerged carrying several amino acid substitutions."
A. Yes.
Q. "The variants mostly had higher R ... numbers than the original wild-type strain and were said to be more transmissible due to mutations in the receptor-binding domain of the spike ... protein."
You've explained the significance of the spike protein.
A. Yes.
Q. And then there are references to two of the variants

that I think we are all, again, familiar with. The Delta variant had an estimated R number of 5.1 ——

A. Mm—hm.

Q. —— and the Omicron variant, which emerged in late 2021, had an estimated R number of 9.5.

A. Yes.

Q. I think that was perhaps one of the alarming aspects as that information came out.

A. Yes. Yes. Yes. Yes.

Q. Yes.

Can we move on now, doctor, to 2.14 at page 26.

You deal at the bottom of that page with, "How quickly does COVID—19 develop?" I think you say that those exposed to an infected person typically develop symptoms between four to five days post—exposure, although obviously you've indicated that people can remain asymptomatic during that period.

A. Of course. Yes.

Q. You then say that:

"The median incubation period of COVID—19 (i.e. the time interval between the individual becoming infected ... and him or her then developing overt symptoms ...) is 4 days (with an interquartile range of 2—7 days). The incubation period can be as long as 14 days."

A. Yes.

Q. Now you deal with those who are at high risk for COVID infection. Perhaps I can hand over to you just to read through what you have said there, please. So 2.14 and following.

A. 2.15, "Who is at high risk" ——

Q. I'm sorry, 2.15.

A. "Who is at high risk for severe COVID—19 infection?

"Individuals who are older, male, from deprived areas, or from black, ethnic or minority groups are at higher risk of severe disease and death from COVID—19."

If I could just qualify that, my Lord. Really the key figure there is older. People who are older than 85 years are at very high risk. The others are at increased risk, but those categories are not equivalent in terms of risk. But they are all at higher risk.

"Substance use (e.g. alcohol, opioid or cocaine use disorder), and current or former smoking both increase the risk."

Q. It is something that the Inquiry is going to look at in due course: is there a particular reason why black, ethnic or minority groups are at higher risk, do you know?

A. Their genetic make—up is the same as ours, but it may be that their home circumstances are different. They tend to be multi—generational, more so than we are. We tend

to live in nuclear families, compared to many ethnic minority groups. So there could be societal reasons as opposed to physiological explanations for it. But it's an area that does really warrant further research.

Q. Yes, because I think it's an area that has been commented on ——

A. Yes, indeed.

Q. —— very considerably ——

A. Yes.

Q. —— and data tends to suggest that there's substance for that.

A. Yes. Some of the ethnic groups do have a higher prevalence of obesity, and that in itself is a risk factor. So that could be another variable that explains this.

Q. You go on to say:

"The risk of severe COVID ..."

And, again, you've got a qualification word there, "severe", and I take it that you've indicated earlier what "severe" is. It's somewhat subjective.

A. It is somewhat subjective, yes.

Q. But it's the sort of level of infection that would tend to suggest that it's time to see your doctor?

A. I guess so, yes. Yes.

Q. And you say that there are further increasing risk

factors, and you list them there. I think they are probably fairly obvious: obesity, diabetes, hypertension, cardiac disease, frailty and impaired immunity, and you go on to say reduced ability to cough and clear bronchial secretions.

We have come across and will continue to come across the concept of impaired immunity or immunosuppressed. Can you just explain what that is?

A. Yes. The immune system is the body's way of combating pathogens, combating infections. It's very complicated, but there are two sides to the immune system.

There's the innate immune system that we're all born with, and that's kind of a general kind of surveillance system that surveys all the potential pathogens that might have gone into the body. It's also called the cellular immune system, confusingly, because there are individual cells within that aspect of the immune system that have particular functions, and that will go out and seek and destroy pathogens. We're born with that, and that tends to decline with age.

The other side of the immune system is what's called the humoral immune system, which is based on antibodies. That tends to be disease—specific. So we develop antibodies for specific diseases when alerted by the innate immune system, and that tends to get better as

you get older; you encounter more diseases, so your stock of antibodies builds up. Furthermore, sometimes antibodies may be cross-reactive. They may be effective against one particular virus, and they may also be partly effective against a different virus.

LORD BRAILSFORD: May I interrupt at this stage.

You indicated, doctor -- I think, in fact, Mr Gale indicated on your behalf -- that the list you give is pretty obvious, and also it's fairly objective. But there's one word you use which I think is a little subjective: "frailty".

A. Yes.

LORD BRAILSFORD: "Frailty", I think many of us or we would all have different interpretations. Perhaps we automatically think of an old person.

A. Yes.

LORD BRAILSFORD: But what do you mean by "frailty" there? Are you able to be a little bit more objective?

A. Yes, it is subjective, and there are frailty indices that will be used in care homes. My wife is a nurse, was for a long time staff nurse in charge of nursing homes, and actually there's an important distinction between nursing homes and residential homes there. So when new residents come into the nursing home or the residential home, they will be assessed for frailty.

There are different measures, but they combine various factors, mainly based on mobility.

LORD BRAILSFORD: Okay.

A. Beyond that, I'm a bit out of my depth, actually. But clearly people have found that there are people who are old but very robust and there are people who are old but very frail, and it is the ones who are old and frail who are particularly at risk of COVID-19. Presumably this is because their innate immune system has gradually diminished with the passage of time, whereas their adaptive or humoral immune system is still all right. They've still got antibodies to a range the diseases they've encountered during their life.

LORD BRAILSFORD: Thank you.

Sorry, Mr Gale.

A. Just -- we were talking yesterday about the swine flu epidemic. That was striking because older people seem not to acquire swine flu, if you remember --

LORD BRAILSFORD: Yes, you said that.

A. -- because they had encountered the same influenza virus in the 1950s, but the younger people were getting it because they hadn't.

MR GALE: I think, just to complete the list, you say that people with chronic liver disease, especially cirrhosis, are at a high risk of severe COVID.

A. Yes.

Q. I think as was apparent from your papers that you referred to --

A. Yes.

Q. -- this is an area with which you have a particular interest.

A. Yes.

Q. Could you just read the final paragraph and perhaps expand on it a little, page 28. It's the final paragraph of 2.15, please.

A. "In the early stages of the pandemic the crude (i.e. all-age) [average] case-fatality rate ... from COVID-19 was reported [in the medical literature and the press] as ranging from 1% to 13%-14%; this very wide variation in a key measure of pathogenicity was explained at the time as being possibly due to different case definitions used and, to some extent, the intensive care capacity of hospitals."

Q. Right. Can you explain -- because obviously an explanation is being given as to that wide range, and at the moment I'm, I have to say, slightly struggling to understand that.

A. Yes. So when the pandemic came along, everyone was taken by surprise, and some centres were reporting, "13 or 14% of our patients that we've admitted are dying

of COVID-19", others were saying, "Well, we were only finding 1% are dying", and it's puzzling that a measure that isn't objective, which is terminal, like death, should have accrued such a very wide range of reported observations.

There are a number of explanations, and one is that there are different definitions of COVID cases in different countries, so different case definitions were used, and some of the people who died may not have died of COVID; they may have died with COVID. So that could explain some of the variation. And some countries may have had better intensive care capacity than others.

Q. And even within countries, the intensive care capacity can vary.

A. Indeed, and some thinking nowadays is that possibly some of the early treatment protocols were actually harmful. They were well-intentioned, of course, but they might have actually been causing more harm than good, because current treatment protocols are very conservative. The current approach in general to COVID is one of medical supportive measures, which Mr Gale suggested is equivalent to palliative treatment, which is right. That seems to be the best way of managing patients with COVID, with medical supportive measures, and very importantly nursing them so they're lying on their

front, so that the secretions drain out of their mouth, not when they're lying on their back.
Q. Now you deal with a section on, again, a subject that we will be looking at in some further detail, and that's COVID-19 and pregnancy --
A. Yes.
Q. -- and the general context of women's issues.
A. Yes.
Q. You've indicated that there was something of a difference: the risk was higher in the earlier stages of the pandemic, and that there was a different level in the Omicron era of the pandemic.
Now, I think you indicate in the first paragraph that the risk was higher if -- and you give a number of factors, which I think are factors that are associated with risk in pregnancy --
A. Yes.
Q. -- outwith the complication of the pandemic.
A. Yes. Yes.
Q. And then the data that is available for the Omicron era indicates that:
"... pregnant women were substantially less likely to have a preterm birth or maternal critical care; fewer stillbirths and no maternal deaths were observed in the UK in this period."

Is there any reason you can --
A. Yes.
Q. -- postulate for that?
A. Yes. The Omicron era began in November 2021, as I mentioned earlier, so it began late on in the pandemic. So the explanation may be that the treatment protocols had been adapted by then and were more on the lines of supportive care, which was giving better outcomes, and perhaps were less interventionist, using aggressive therapies such as intravenous fluid replacement, whereas oral fluids might have been preferred.
So this no doubt is being discussed even as we speak, and the answer to that question should become apparent with the passage of time, but I can just surmise as to what the reasons might have been.
Q. And also the vaccine programme?
A. Yes, indeed. So many of those women would have been vaccinated and, as we will discuss later, the vaccine does seem to confer, or does confer, based on the Cochrane analysis, less severe disease.
Q. Move on now to 2.17, "Who is at low risk" --
A. Yes.
Q. -- "for severe COVID-19 infection?"
Within this passage there is, perhaps, a somewhat

concerning sentence, and one that perhaps needs to be considered in a little more detail. I will read it out:
"In general, COVID-19 has a milder disease course in children and young adults than it does in older adults. The majority of children recover completely after acute SARS-CoV-2 infection and any persistent symptoms will improve with time."
Now, I think you're presenting that --
A. Yes.
Q. -- from the point of view of a public health physician.
A. Yes.
Q. And obviously you are looking at, if I can put it this way, the larger, bigger picture.
A. Yes.
Q. And, of course, within that larger, bigger picture, there will be and will have been exceptions to the generality that you are stating there.
A. Of course, yes.
Q. And I think yesterday we talked about it: that it's perhaps of little comfort to those who have lost someone --
A. Yes.
Q. -- whether that person be in the area that is perhaps at most risk, such as the elderly, but also those who have lost someone in the apparently less risk category.

A. Yes.
Q. You then go on to talk about the case-fatality rate in the third full paragraph on that page, and then you have set out a table.
Can you tell me where that table comes from, and perhaps just take us through it, please.
A. Yes. That table is taken from what's called the Green Book, which is a Department of Health manual that is used by anybody who is in any way involved with the vaccination process, whether epidemiologists or GPs or nurses who administer vaccines. It used to be a green book, but nowadays it's all online, so this table was taken from the online version, published earlier this year. I've got a copy there.
The table shows deaths in 2020 in England, but I think the table can be generalised to Scotland as well, and it breaks the deaths that one can attribute to COVID down into males and females, and it stratifies them very helpfully by age groups.
So the first line is the deaths that occurred in the under 18s. So looking really at the right-hand column, there were 32 deaths in England in 2020 in the under 18s that are attributed to COVID-19, so there's a very small rate of death in the under 18s in that year. The following year might have been even

smaller because, as I was saying, the treatment protocols were better.

2020, the height of the pandemic, the rate was, 100,000 children, 0.26. So that's equivalent to two per million; two out of a million children would have died of COVID that year.

As we move up the age categories, that number gets higher and higher, until we get into those who are 85 and older, and you can see there were 26,954 deaths in total attributed to COVID, so a massive number, in the 85s and over, and the rate there for every 100,000 people who are 85 and over, it's actually 19,160.

LORD BRAILSFORD: 1,900, I think.

A. Well, that's —— sorry, for every million.

LORD BRAILSFORD: Every million.

A. So let's take 100,000. Thank you, my Lord. So rate per 100,000 is 1,916.51.

I did a little calculation on that, and if you take the over 65s, that comes to 59,628 deaths, those over 65, and that is 89% of all the deaths.

LORD BRAILSFORD: 89?

A. 89% of all the deaths were in those over 65, and in fact one in four of them was in those over 85.

LORD BRAILSFORD: Yes.

A. So very much a disease related to age and extremes of

age, based on the England figures, but I would confidently suggest that Scottish figures would be comparable.

MR GALE: Yes. You don't see any reason why they would be different?

A. I don't see any reason.

Q. If you go over the page to page 30, you make a reference there to:

"A very small majority of children infected with SARS—CoV—2 (approximately 1 in 3,000) developed a multi—system inflammatory syndrome with Kawasaki disease—like features; this is known as mucocutaneous lymph node syndrome and as paediatric multisystem inflammatory syndrome temporarily associated with SARS—CoV—2 ..."

You said a small minority of children. What would be the symptoms of that and the effect of that?

A. Well, again, it would depend on whatever system was being involved. So because COVID can infect any system, it's a multisystem disease as we agreed yesterday, I guess any system, from the heart, the kidneys, the brain, the lungs, or it may be many systems at the same time.

Q. Anything that was the subject of the attack?

A. Yes. Indeed, yes.

I had meant to put in there a further point, but perhaps it was obvious, but one of the standard textbooks I've been citing did go on to say that usually they get better. Usually they get better. We have to take that on trust.

Q. I am reminded, can you just perhaps indicate what —— we probably know the word "Kawasaki" from a different context, but could you tell us what Kawasaki disease is?

A. I don't know what that is.

Q. You don't know?

A. It's clearly a very rare disease that paediatricians are familiar with and they were surprised to see this in children.

Q. Okay. Right.

You then go on to the pathological processes that occur in COVID—19.

A. Mm.

Q. I think probably we can simply read that section at 2.18 for ourselves and perhaps move on to 2.19, which are the clinical features of COVID—19.

You refer first of all to early features. Perhaps you could just go through those, please.

A. Yes:

"In patients with symptomatic COVID—19 infection, the initial symptoms are non—specific and appear after

an incubation period of approximately 2 to 7 days; typically [these initial] symptoms will include:

"— fever;

"— headache;

"— myalgia (i.e. muscle pain) and

"— malaise (i.e. general unwellness).

"At the same time [or around the same time], the patiently may experience anosmia (i.e. loss of smell), and dysgeunia (i.e. distortion of taste)."

Q. And I think we probably are all familiar with those being publicised at the time of the pandemic, and I think any of us who have had COVID will probably recognise all of those symptoms.

A. Yes.

Q. You then go on to the later features, and again it would be useful perhaps if you just read through that section, please.

A. Yes. So the majority of individuals with COVID will recover spontaneously, and in China, up to 80% of those who had been infected had only mild symptoms not requiring hospitalisation. But in those other patients with severe symptoms, over a matter of days or weeks, COVID—19 may progress to one or more severe syndromes. A syndrome is a symptom cluster, so it could be respiratory syndrome, with dry cough, sore throat, nasal

congestion, shortness of breath, and low oxygen saturation, or it could be some kind of cardiac syndrome, or coagulopathy, meaning a clotting disorder syndrome, or some other immune system that results in treatment difficulties.

Q. Would you just continue on reading?

A. Yes. So:

"Other symptoms [beside these syndromes], such as profound fatigue and skin rashes may also be present.

"50% of patients with confirmed COVID-19 will report ... gastrointestinal symptoms ... [mainly] diarrhoea (in 38% of those who are sick) and vomiting (in 13%)."

And sometimes the patient will have gastrointestinal symptoms and nothing else.

"Some patients with severe COVID-19 may deteriorate rapidly and develop life-threatening complications, including:

"– thromboembolic events [clotting events];

"– cardiac disease;

"– acute kidney injury;

"– sepsis;

"– septic shock; and

"– multi-organ failure."

Which is the herald of death.

Q. Yes. Just continue on, please.

A. So:

"Natural immunity ..."

When you've acquired SARS-CoV-2 naturally -- naturally means it lasts up to one year before beginning to wane, and that sort of fits in with the natural immunity we normally experience with influenza:

"... although the new strains and variants, such as Omicron, appear to exhibit greater immune escape [so they manage to evade the immune system] making reinfection more common."

Q. You now deal, doctor, with, "How does COVID-19 present in the elderly?", and I think we look at this bearing in mind the material that we've looked at --

A. Yes.

Q. -- in relation to death rates.

A. Yes.

Q. So perhaps you could -- again, it's perhaps a section that is useful for you to read through, albeit that I appreciate you have a table there. If you could just read the text and just take us through the table.

A. Yes. So, in the elderly, the presentation is different and atypical. The elderly may experience delirium and reduced mobility -- also immunocompromised individuals as well -- and often they don't have a fever.

So there's a table summarising how COVID-19 presents

in the elderly. So the non-specific signs and symptoms are different. So often they don't have a fever. They may not be breathless. They may, however, become delirious, and even severely delirious. They often don't have lung problems, particularly, but they can have clotting problems, thromboses, or gastrointestinal upset, diarrhoea and vomiting, like we've just talked about. So it can be difficult to pick up COVID-19 in the elderly if you're caring for them, or a doctor or nurse.

The outcomes are different. Often they carry COVID-19 virus asymptomatically for extended periods. The elderly have high morbidity, that means they are more likely to have severe disease, and mortality, they're more likely to have a fatal outcome.

The elderly who survive COVID-19 will often experience functional decline, meaning their natural ability to function in their environment will get worse, and they may need rehabilitation, which is likely to be extensive and expensive.

Here is the word "frailty" again. Those old people who are frail, over and above being old, have particularly poor outcomes, but even people who are very frail, it seems, can acquire COVID-19 and survive.

And then it says here a frailty assessment is a good

tool. I'm not quite sure how it's done, but there are numerical measures you can use to categorise somebody as being frail or not, and they can help people, for example, who manage care homes to decide as to how to manage a particular individual and what risk they may or may not be at. But they shouldn't be the only consideration; other factors should be involved as well.

Q. Obviously all of those signs and symptoms in an elderly person would be distressing --

A. Yes. Oh, yes.

Q. -- for that person and for those who are their relatives, carers, loved ones.

A. Yes.

Q. But particularly, I suppose, delirium.

A. Particularly delirium, when the person in front of you is just not the person that you knew two weeks ago, but someone quite different, yes.

Q. You go on now to a subject that I think we are hearing more and more about, and that is long COVID.

A. Yes.

Q. The audience will be aware that the Inquiry has published an opinion on long COVID, to the extent that it intends to investigate long COVID.

So, with that in mind, could you just take us through that relatively short passage that you have at

2.22, at page 33 to 34, on long COVID.
A. Yes. It's relatively short because there isn't much about it in the textbooks as yet. It is a recent phenomenon. But here we are:
"As is the case also with other viral infections, such as infectious mononucleosis (i.e. ... 'glandular fever') ..."
Glandular fever can often result in very long-term debilitating symptoms:
"... COVID-19 may give rise to prolonged symptoms that persist for more than 4 weeks; this is known as long COVID. In the UK, 4.5% of COVID-19 cases report long-term symptoms 12-16 weeks after initial infection."
So about 1 in 20 of cases:
"Other terms for long COVID include post-COVID syndrome, and post-acute sequelae of COVID-19 (PASC).
"Reported symptoms of long COVID are varied, involving most organ symptoms and affecting both physical and mental health. Commonly-reported long COVID symptoms are:
"- shortness of breath;
"- fatigue;
"- headache; and
"- difficulty thinking or concentrating.
"In addition to [those four common symptoms], there

is growing evidence of long-term cardiovascular sequelae of COVID-19, including cerebrovascular disorders [and I guess that means minor strokes, transient ischaemic attacks], cardiac dysrhythmias [where the heart rhythm isn't normal], heart failure [where the heart pump is no longer efficient], ischaemic and non-ischaemic heart disease [same thing, really], myocarditis [inflammation of the myocardium, the muscle of the heart], pericarditis [inflammation of the fibrous sac surrounding the heart] and thromboembolic disease [which means long-term clotting disease]."
So all of those would fit under the category of long COVID.
Q. Yes.
In this Inquiry we have a group representing -- it's called Long Covid Kids.
A. Yes.
Q. So obviously children suffering long COVID.
A. Yes.
Q. Is that something of which you are aware of any research having been done?
A. I would hope there's extensive research being done into long COVID, but I'm not familiar with the details of what research is being done in various centres.
I believe it was first recognised as an entity in

the UK, and initially other countries weren't aware of this phenomenon. But they now have become more aware, so it may be research is being done in other countries as well.
Q. Yes, thank you.
A. The belief --
Q. I'm sorry.
A. I'm so sorry. I understand that the belief is that for most people with long COVID, they do gradually get better; for most, obviously not for all. By analogy with glandular fever-type symptoms, where people can be very exhausted for months and months, but gradually they get better. But in the short term they will be often very disabled indeed.
Q. You move on to a section on diagnostic tests.
A. Yes.
Q. I'm, with respect, going to take that as read. I think a lot of the tests are quite well known to us from our own experiences, as are the -- and I'm not going to take you through the laboratory findings or the radiological abnormalities.
But what I would like to take you on to is 2.26 at page 36, please, which is, "How is COVID-19 treated?" and "Medical supportive care". I think this is something, again, we touched on yesterday --

A. Yes.
Q. -- under reference to, I think, one of the Cochrane reviews.
Perhaps you could just read through what you have said there in section 2.26.
A. Yes:
"In the early stages of the pandemic, COVID-19 patients with severe respiratory distress were often treated aggressively with intravenous fluids and mechanical ventilation; it became apparent however that intravenous fluids could exacerbate fluid in the lungs and further reduce oxygenation.
"Another early clinical observation was that lying in a prone position (i.e. on the stomach, [is better than lying in the] ... supine position on the back [which is how you lie in intensive care, you're on your back]) led to improved oxygenation in patients who were receiving supplemental oxygen therapy through a face mask or nasal tubes. This [simple nursing measure] resulted in fewer intubations [where a patient is first of all paralysed through drugs and then a breathing tube inserted into them] ... which themselves were a[n additional] cause of morbidity and mortality."
All of that is taken from standard textbooks, not my opinion.

Then:

"The current mainstay of COVID-19 management is medical supportive care.

"COVID-19 patients who are coughing should initially be managed using simple non-drug measures (e.g. honey)."

This is the British National Formulary advice to doctors, give them non-drug measures initially.

If a patient has a distressing cough, you could use a cough suppressant, a simple cough linctus, in the short term, for example codeine phosphate.

If a COVID-19 patient has a fever, the pharmaceutical advice is tell them:

"... to drink fluids regularly to avoid dehydration, and to take [simple] antipyretics [drugs to take down the fever] (e.g. paracetamol or ibuprofen) ..."

Q. The second aspect of COVID-19 treatment is pharmacological therapy.

A. Yes.

Q. You say that most drugs tested have shown marginal or disappointing efficacy against SARS, and you give the reference at footnote 186, which I think is to Louten, Essential Human Virology --

A. Yes.

Q. -- and it's either a paper or a book from 2023, so it's post the height of the pandemic.

A. Yes.

Q. I suppose to most of us, who immediately think that there's a pharmacological remedy for anything that we're suffering --

A. Yes.

Q. -- that may sound quite a surprising finding.

A. Yes.

Q. Did you find it surprising?

A. Well, first of all I might qualify that by saying that the steroids don't come into the category. Steroids seem to be effective, dexamethasone. But I think what she's talking about there are antiviral drugs, so viral-specific drugs. Steroids come into the category of sort of standard, non-complicated measures.

But a large number of antiviral drugs at various times were put forward as the wonderful remedy, and I think one came into the timeline with the words saying "Great news, this antiviral drug has now been approved for COVID", and it was a little bit surprising to see it stated that antiviral drugs don't seem to have much efficacy.

I got a bit worried about this, because in the UK Inquiry they were told the opposite; they were told that drug therapy had had a remarkable effect in improving COVID outcomes. Again, I think what was really meant

there was probably dexamethasone. So I was a bit concerned.

Last week I thought: well, I'll see if there's a Cochrane review about -- I just chose one drug at random -- remdesvir, to see what the Cochrane people say about it. I found a Cochrane review which was published earlier this year, so it's publicly available. I'll just read one line from it.

Cochrane review, published in January 2023, and is called, "Remdesvir for the treatment of COVID-19". They found a number of studies. They used the standard Cochrane methodology. They found nine randomised controlled trials of remdesvir to treat COVID-19, 11,218 participants, and they were randomised to receive remdesvir or not receive remdesvir.

I won't go through the details of the studies, they're available, but the authors' conclusions:

"Based on the available evidence up to 31 May 2022 [which is their cut-off point for the trial], remdesivir probably has little or no effect on all-cause mortality or in-hospital mortality of individuals with moderate to severe COVID-19."

So that was certainly the position as of last year.

So --

LORD BRAILSFORD: You say you found that; was that produced

in the bundle of documents?

A. It wasn't. We could have had Cochrane reviews for every test, every --

LORD BRAILSFORD: True, but could we get a copy of that, Mr Gale, please?

MR GALE: Sure.

LORD BRAILSFORD: Thank you.

A. Yes. Indeed, there will be -- a number of other antiviral drugs are mentioned there, my Lord, and there will certainly be Cochrane reviews, either published or in progress, for all of those.

Of course, bearing in mind that Cochrane reviews are dynamic, and as they say, in a year's time they might change their view, but it's unlikely they would change it.

MR GALE: There is only one other sentence I would like to take you to. It's page 39.

A. Yes.

Q. It is in relation to -- it's after the various drugs that are mentioned, including remdesvir.

A. Yes.

Q. There's a paragraph which says:

"The safety of COVID-19 antiviral treatment during pregnancy has not been established."

I think the reference you give to that is from the

British National Formulary.
A. Yes. It's to -- yes. Yes, thank you, yes. Yes.
Yes, the British National Formulary have become rather conservative, and they don't want doctors to start giving antiviral treatments, perhaps because the patient is demanding it, because they feel that the safety margin is still under exploration.
MR GALE: Right.
My Lord, that's perhaps a useful point at which to break.
LORD BRAILSFORD: Of course, yes. Thank you.
We'll take 15 or so minutes now. Thank you very much indeed.
Thank you, doctor.
(11.28 am)
(A short break)
(11.53 am)
LORD BRAILSFORD: Thank you. We're just about to start again.
However, there's something I would like to say. It has been brought to my attention, I should say, by Mr Gale's junior counsel, who must have been talking to people during the coffee break, that some of the solicitors that are present here are busy and assiduously taking notes.

Can I save them some work, to be perfectly honest -- and I apologise, perhaps I should have thought about this yesterday -- but this is all being recorded. The availability of a transcript will be there, and you will be able to get a transcript of this. I cannot say -- I'll try and say, perhaps, at the beginning of the afternoon session -- when you can expect it, but my understanding is it should be available in very short order, probably next week sometime.
So if that assists you and eases your hand muscles, I hope I'm doing some good.
Right, Mr Gale, when you're ready.
MR GALE: Thank you, my Lord.
Just another piece of housekeeping. Dr Croft referred to the Cochrane review of remdesvir, of which he handed a copy, I think, to you. There is now a copy available for your Lordship.
LORD BRAILSFORD: Thank you.
MR GALE: And we have copies if anybody wants a copy. It will be available, perhaps at lunchtime, to be picked up. Fortunately, it's not quite of the length of the other Cochrane reviews that we have; it's only two pages, so it's manageable.
Right, Dr Croft, can we go back to your statement at 2.28, please, because you begin here with

a consideration of probably what is a general but discrete area, and is in the context of, "How is COVID-19 prevented?", and there are various aspects of that.
We will look at this in some more detail in section 3 of your report, which we will be going to next, and in particular the physical measures taken against COVID, but perhaps you can just take us through, first of all, the general public health measures that you identify.
A. Yes. Shall I read on --
Q. Yes, please read on.
A. So general public health measures to prevent COVID-19:
"COVID-19 may be prevented through standard infection control measures, along with the public health management of infected cases.
"The most basic public health measure against COVID-19, which was implemented in all countries during the ... pandemic, was promoting frequent handwashing. Large-scale frequent surface decontamination efforts were deployed in public spaces, but the effect of these cleanings on reducing transmission was and remains uncertain."
Q. I think that's something that we will be touching on in due course.

A. We will, yes.
Q. We move on now to PPE.
A. So the second category of prevention is PPE:
"All countries advised the use of personal protective equipment (PPE) by frontline healthcare staff during the COVID-19 pandemic. Challenges included the rapid depletion of PPE, the lag between the spread of infection and the acquisition of evidence required to inform precautions to control its spread, and frequent changes in PPE guidance in response to its availability."
Q. I think, just pausing there, one of the other challenges may also have been the varying quality of some PPE.
A. Yes, indeed. And most countries recommended, and in some cases enforced, the use of face coverings by all adults, not simply healthcare staff, in places where close contact was likely.
Q. Yes.
We move from, as it were, PPE and general health measures to lockdowns now.
I think you commented yesterday, under reference to the approach taken by the government of China in relation to lockdowns --
A. Yes.
Q. -- and I think the terminology came from that time.

Would you just read on, please, at 2.30.
A. Yes. Of course, there's a definition of lockdown in the ——
Q. There is, yes.
A. We will take that as read.
"A major strategy for attempting to limit the spread of SARS—CoV—2 was the introduction by some governments, starting with China, of extreme physical distancing measures; these have been termed lockdowns.
"The components and restrictiveness of lockdowns varied, and not all countries employed lockdowns. Where lockdowns against COVID—19 were introduced, they typically included:
"– the closure of schools, workplaces, non—essential shops, sporting and entertainment venues;
"– a move to 'remote' (i.e. computer—based) working where possible;
"– banning mass gatherings;
"– curfews;
"– stay—at—home orders; and
"– other local, national and international travel restrictions.
"In some countries where extreme physical distancing measures were employed early in the COVID—19 pandemic (e.g. New Zealand), they resulted in complete, although

temporary, eradication of virus in the community."
Q. You say there that in relation to New Zealand ——
A. Yes.
Q. —— it was complete, although temporary, eradication of the virus. What happened after that temporary eradication, do you know?
A. I'm assuming what it means there is that the lockdown temporarily eradicated —— temporarily, there were no COVID cases, because they had a policy of zero COVID, but then new cases emerged. But that's something that does need investigation. I believe there may be some evidence taken from New Zealand later on.
The same was seen in China, of course, where even this year, even earlier this year, they were still having intermittent lockdowns focused on particular cities. They only abandoned it because of severe public unrest.
Q. Yes.
Right, the next area of prevention is social distancing. Again, would you read that, please.
A. Yes. Social distancing:
"In countries where extreme physical distancing measures (i.e. lockdowns) were not considered necessary, or else were temporarily relaxed, social distancing strategies were employed instead; these involved keeping

people physically separate (a target of [greater than or equal to] 2 metres was used in the UK).
"Vulnerable adults, including older and immunocompromised people, were advised to curtail all social interactions; this strategy was termed shielding, in the UK."
Q. Finally in this section: test, trace and isolate measures.
A. So test, trace and isolate measures:
"Other measures used to limit, or attempt to limit, the spread of SARS—CoV—2 were high levels of case identification, with widespread testing in order to identify cases and ensure public health follow—up of potential cases, and enforcing quarantine measures for cases, contacts and travellers from high—incidence countries. The combination of such strategies has been termed test, trace and isolate (TTI).
"More novel approaches to limit, or attempt to limit, the spread of SARS—CoV—2 included the use of mobile phone apps, and (depending on jurisdiction and legal constraints) use of CCTV footage and tracking of a contact's digital signature.
"In the UK (including Scotland), and in other countries also, the processes of death certification were streamlined in early 2020, to deal with anticipated

surges in deaths."
Q. What is the significance of that?
A. The significance is that, under certain conditions, a pathologist is required to perform a postmortem, but those conditions were relaxed significantly, and for a time I believe postmortems weren't even being done on COVID—19 cases. So, to some extent, the certainty that could be attributed to whether or not a person died of COVID—19 or with COVID—19 has to be considered a compounding factor in assessing the mortality. It wasn't done to, if you like, obfuscate the situation; it was a practical measure to deal with the anticipated very large number of deaths.
Q. Right.
Now, at 2.33, page 42, we go on to the sixth aspect of prevention, and this is, in terms of what you say, the most detailed, and that's vaccination.
Now, again, vaccination is something we're going to come to in section 4 of your report, so I'm going to take this, with respect, relatively short.
A. Yes.
Q. I think we can see, at the very beginning, the COVID vaccine was developed at record speed ——
A. Yes.
Q. —— and you identify four factors for that: prior

research, the state of vaccine technology, abundant funding and a large group of willing volunteers.

I think probably everybody who, again, remembers the circumstances of the pandemic and the development of the vaccine, and without the level of information that you would have had, probably thought, expressing a personal view here, as indeed did I, that a vaccine, I expected, would have taken a great deal longer than it did.

You have identified these factors. Was there any worry about the fact that it was developed so quickly?

A. Well, yes, people have expressed concern, and people were expressing concern at the time, and ideally a vaccine should be developed over a period of time to allow close understanding of how the vaccine may benefit the population and, equally, how harms may arise that might not be foreseen at first. So the speed of development was of concern.

Then some vaccines are developed quickly, and vaccines for seasonal influenza are often developed quite quickly, because they will be based on the previous season's circulating virus, and typically they can be developed in seven or eight months. The influenza season ends and the companies that make vaccines start to develop the one that they anticipate will be effective in the next season. So there are some

precedents for fast vaccine development.

But, in general, it's not ideal to develop a drug or a vaccine at high speed because it means that potentially key stages in the analyses of either the efficacy or the harms might be missed or skipped over.

Q. One of the things you mention —— and I think you've already mentioned —— at page 43 in the first paragraph, you mention that vaccine development, the phases were often run concurrently, so that had the effect of speeding up the process.

A. Yes.

Q. And I think also it's fairly obvious that this was a situation at which, if one can say it, money was little object.

A. Indeed, yes.

Q. Now, you give a date of 27 July 2020 in relation to the Pfizer–BioNTech vaccine, and of the Moderna vaccine, and I think then you say that globally vaccines against COVID–19 fall into one of three categories.

A. Yes.

Q. It's perhaps useful if you just explain this. I think we probably touched on it a little yesterday in relation to viral vector vaccines.

A. Yes.

Q. Perhaps you could just explain the difference —— well,

what each category of vaccine is and what are the differences.

A. Yes. So a vaccine works by presenting antigens from the pathogen to the body's immune system, and the pathogen is surrounded by a number of molecules that the body's immune system will recognise as foreign, and the trick is to work out which of these molecules are particularly antigenic, which are the ones that especially trigger an immune response, and then put those into your vaccine and, in that way, trigger an immune response before the body is exposed to the pathogen.

So component vaccines, in very crude terms, are conventional vaccines. You could regard them as being mashed up viruses; mash them all up and inject them into the person. It's not quite as simple as that because, in general, there's an attempt to try and extract some components from the whole architecture of the virus and put them into the body, if you like, preferentially. But that's a very rough way of looking at conventional vaccines.

Q. Can you give an example of a conventional vaccine?

A. Yes. For example, a conventional vaccine would be, for instance, the smallpox vaccine, which was efficient in eradicating smallpox in 1978, and that was effective because smallpox is a DNA virus, and DNA viruses are

more stable. Therefore, if you achieve immunity against smallpox, you will never get smallpox. So you can vaccinate against smallpox and it will be very effective for your whole life. So that was a conventional vaccine.

Q. And coronaviruses are RNA viruses?

A. Well, this is the next sort of vaccine. It's possible to have a conventional vaccine against coronaviruses, because you just mash up the virus and inject it into patients, and, in very crude terms, that was what was done with the conventional technology that produced conventional vaccines.

Q. Right.

A. But there was a new approach, which was a genetic approach, to COVID–19. It had been tried out over about the previous decade, really since SARS, and the idea of the genetic approach really was to instruct the body of the host, the human host, to produce antibodies that would stop the virus from entering the cells.

So we mentioned the spike protein earlier on that is the Yale key that attaches to the receptor on the cells, fuses with the receptor and opens the cell membranes so that the virus can get into the cells. So the spike protein, based on earlier research with SARS, the original SARS virus, was judged correctly to be, if you

like, the key to achieve entry into the cells and so, therefore, was the target for the genetic approach to designing vaccines.

Q. Now, you refer also to viral vector vaccines against these ——

A. Yes.

Q. —— and that is, you say, using novel technology.

A. Yes.

Q. Can you explain, please.

A. Yes. So there are two ways of instructing the cell to produce antibodies against —— actually, I jumped ahead a bit. You're instructing the cell —— you're instructing the body to produce spike protein, and then the immune system of the body then recognises that spike protein and produces antibodies. I should have made that clear. That's the way vaccines based on genetic approaches work: they instruct the body to produce spike protein, so you produce spike protein, and then your immune system recognises a spike protein and responds to that by producing antibodies.

So viral vector vaccine, they work by modifying harmless viruses that are not going to produce disease when they're injected into an individual, and the viruses are modified so they're carrying the DNA —— DNA being genetic code —— that instructs the body to

produce —— manufacture spike protein.

The RNA viruses work slightly differently. They're carrying messenger RNA, which goes into the cells, into the nucleus of the cells, and the nucleus —— and then the —— and then —— can I just check this one, my Lord.

Essentially, the end result is that the cells likewise produce spike protein, but through two slightly different approaches.

Q. Right.

You have set out or taken a table which you can find at page 44 ——

A. Yes.

Q. —— of your report, and I think at the bottom of page 43, you say that the distinction between conventional and novel technology vaccines are shown the table overleaf.

A. Yes.

Q. And the first three categories in the table are shown, and these are: live attenuated vaccines ——

A. Yes.

Q. —— inactivated, killed—off whole—cell vaccines, and component vaccines. So the component vaccine was the one that you were referring to in your text.

A. Yes. Yes. Yes.

Q. But thereafter we are looking at novel technology.

Does that have any meaning other than the fact it is

new? So "novel technology". Or does it imply something beyond it being a newly developed technique?

A. It's a way of categorising vaccines that is helpful. The first two categories are really the traditional ways. The component vaccines are kind of relatively modern ways by which they try and identify particular components of the pathogen that are likely to be especially immunogenic, and the components are concentrated in the vaccine, so they are an advance on the earlier two categories. Then the two bottom categories are the genetic approaches to producing vaccines.

Q. So those two categories are the viral vector vaccines ——

A. That's right, yes.

Q. —— and the nucleic acid vaccines.

A. Exactly, yes. Yes.

Q. And I think we can see that COVID—19 falls within both the viral vector vaccines and the nucleic acid vaccines.

A. Yes.

Q. But also within the component vaccines, which I think you've explained as the —— you put it as the mashed—up vaccine.

A. Yes. That's right. So just to re —— the viral vector vaccines carry DNA that codes for spike protein. They go into the nucleus and the nucleus produces

messenger RNA, which goes into the ribosomes, which produce the spike protein. The bottom category have RNA which sort of bypasses the first process: the RNA goes straight to the ribosomes and produces spike protein.

We can perhaps elaborate on that a little bit later. It comes a bit later in my report.

Q. Yes.

At page 45, at the top, you say that in the States, two mRNA vaccines were approved —— well, received emergency use authorisation in December 2020 by the FDA.

A. Mm.

Q. These were the Moderna vaccine for use in individuals 18 and over, and the Pfizer BioNTech vaccine, again for use in a similar —— not quite similar, 16 or over age group.

So you pass on, then, to look at the initial COVID vaccines procured in the UK, and I think —— again, taking this short —— you've listed four vaccines there: the AstraZeneca, the Janssen, the Moderna and the Pfizer—BioNTech.

A. Yes.

Q. I think at the bottom of page 45, you notice that the UK Government did announce in April 2021 that 20 million doses of the Janssen vaccine had been ordered from the manufacturer, but you go on to say that this vaccine has

never been used in the UK.
A. Yes. That's my understanding. Later on, I think we may have time to consider the MHRA report, and they have nothing to say about the Janssen vaccine because it hasn't actually been used on the public.
Q. Am I right in thinking that it was used in Ireland?
A. I believe it was used in Ireland, yes. Yes, it was.
Q. But we don't at present -- and you are not able to assist us in knowing what happened post the ordering of 20 million doses?
A. Where the order went to, we don't know. We don't know.
Q. The following page, at 46, you deal with later COVID-19 vaccines procured in the UK. Again, you've provided these, and I think this takes us into booster use.
A. Yes.
Q. I think you've set that out in the second paragraph.
Then, at 2.36 of your report, you talk about the current vaccination programme against COVID-19 in the UK, and you summarise that. I think you say, to begin with, that the three fairly obvious aims were:
"- to provide protection for individuals who are considered at highest risk of severe illness or death from COVID ...
"- to reduce hospitalisations; and
"- to protect frontline health and social care staff

from exposure."
A. Yes.
Q. And I think that you then go on to indicate that all UK adults and children aged five and a half or over are currently eligible for a primary vaccination course.
Just explain what a primary vaccination course is. It probably is obvious, but just tell us.
A. I think I might have the wrong citation, I do beg your pardon. That citation should be to the British National Formulary.
Q. Oh, right. Well, with that correction, can you just tell us what a primary vaccination course is.
A. Primary vaccination means the initial exposure of the recipient to the vaccine. So some vaccines are just a one-off exposure, just one vaccine will give you protection for life. Some require two or three sequential vaccines separated by a defined time interval in order to achieve immunity to the pathogen being immunised against.
Q. Yes.
A. So, for example, the hepatitis B vaccination, which I have had, which all doctors have to have, it's normally three courses of hepatitis B vaccine that you have over a period of six months, and that generally gives lifelong immunity to hepatitis B, which is a DNA

virus, so you would expect that.
Q. Yes.
I think at page 47, the first full paragraph, you note that the manufacturers' initial advice during 2021 was that COVID-19 vaccines should be administered as a two-dose schedule with three to four weeks between each dose.
A. Yes. Some manufacturers had a slightly different interval, but the idea was there should be a first dose, then an interval of some weeks and then a second dose.
Q. And again --
A. That was the idea.
Q. -- I think with other things that we will look at, that is obviously dependent on the willingness and the ability of the person to attend for the second dose.
A. Yes. Yes.
Q. So there could be some failures in that programme.
A. Yes.
Q. Yes.
One point you raise -- it's a single paragraph in the middle of page 47 -- is that where possible the same COVID-19 vaccine should be used for the entire primary course.
A. Yes.
Q. Has that always been possible, do you know?

A. It's a standard principle of immunisation technique -- strategy that when you have a vaccine that requires two or three doses, you will try and use the same vaccine for each of those doses, because vaccines tend to work slightly differently, and you may not achieve proper immunity by chopping and changing as to what vaccine you use.
Q. Right. Now, in the next two sections, doctor, you look at the way in which novel COVID-19 vaccines work.
A. Yes.
Q. You look first at vector vaccines and then mRNA vaccines --
A. Yes.
Q. -- going over on to page 48. Again, I will, with respect, take that as read from you without going into the detail of it. But I would like to look, please, at 2.39 at page 49, which are booster doses.
A. Yes.
Q. And I think you note at the beginning that in September 2021, Israel became the first country to demonstrate waning protection from Pfizer vaccine, showing a decline in protection even against severe disease at around six months. This was perhaps the -- at least the booster, for boosters, I suppose, if I can put it that way.

A. Yes.
Q. This was something that set us on the trail of booster doses.
A. Indeed, I think it had been hoped that boosters wouldn't be necessary. Clearly the hope was that two doses would give extensive protection for a long period of time, but it became apparent this wasn't the case and the idea of boosters then came in.
Q. I think you say —— perhaps it would be useful to note —— in the penultimate paragraph on page 49 that:
"Protection against hospitalisation after an mRNA 'booster' reaches over 90% in the 2 weeks after vaccination and then declines towards a stable plateau of around 60% by 6 months."
Again, is that something that was expected?
A. With hindsight, and given the —— as you described it, Mr Gale —— fact that we were dealing with a moving target, it should have been expected, but I don't think that was conveyed to the general public as a likely outcome. Certainly in England the expectation was we went into lockdown, but the vaccines were coming and they would release us from lockdown and everything would return to normal pretty much straight away.
Q. Yes. So you set out at the bottom of page 49 the current UK practice in relation to the offering of

a first booster. Perhaps you would just take us through that, please.
A. Shall I read it out?
Q. Yes, please.
A. So:
"Current UK practice ..."
And this is drawn from the latest text in the Green Book which I've got here, which is April 2023, I believe:
"... is to offer a first 'booster' ... at least 3 months after completion of primary immunisation to the following groups ..."
And there are three groups: firstly, all individuals aged 16 and over; secondly, children aged 12 to 15 years in clinical at—risk groups or who are household contacts of immunosuppressed individuals; and thirdly, children aged 5 to 11 years with severe immunosuppression.
Q. Perhaps just carry on ——
A. Yes.
Q. —— to the bottom of that section, doctor.
A. And then as well as boosters of the primary courses, also seasonal boosters that are programmed for spring and autumn every year. And the current UK practice is a seasonal booster, in addition to any booster you might already have had for your primary course, to be offered,

provided there has been an interval of at least three months from the previous dose, to the following groups, and there are six groups here.
Firstly, residents of and staff working in care homes for older adults. Secondly, frontline health and social care workers. Thirdly, all individuals aged 50 years and over. Fourthly, individuals aged 5 years and above in a clinical at—risk group. Fifthly, individuals aged 5 years and over who are household contacts of immunosuppressed individuals. And finally, individuals aged 16 years and over who are carers.
Q. Yes. You go on now to discuss specific patient groups. The first group you look at are pregnant females, and I think we've already touched on this matter.
A. Yes.
Q. But you say that current UK advice is that pregnant females should be offered immunisation against COVID—19 as pregnancy is a risk factor for severe COVID infection.
A. Yes.
Q. So it's looking at it from that perspective, from the risk of infection?
A. Yes.
Q. I think also we have individuals with severe immunosuppression. Again, we've touched on this.

Perhaps it would be useful at this stage if you gave an indication of what is understood in the public health field as severe immunosuppression.
A. Yes. So immunosuppressed people are those whose immune system doesn't work —— either doesn't work at all or doesn't work efficiently to protect them against pathogens.
Q. Yes.
A. And that could be because they have disease, either a chronic general disease such as diabetes or cardiovascular disease or kidney disease, or because they've got a disease specific to their immune systems like leukaemia, so they have no white cells or very few white cells; or alternatively they may —— or HIV disease, which is a disease of a subset of the white cells; or alternatively they may be taking immunosuppressant drugs such as steroids or some of the current anticancer drugs, for example, methotrexate or monoclonal antibodies, and they work by deliberately suppressing the immune system so as to dampen down the inflammatory reaction of the body.
Q. I think what you say at the bottom of page 50 is effectively a summary of what is contained in the previous two paragraphs, particularly about children.
A. Yes.

Q. You also go on to say at page 51 at the top that:
"Current UK advice is that people with HIV infection, regardless of their CD4 count, should likewise be offered a third dose of [COVID] as part of the primary course, along with subsequent 'booster' doses."
A. Yes.
Q. You now look briefly at an area that you entitle "How safe are COVID-19 vaccines?", and you begin with thromboembolic clotting events. Perhaps you would just read through that, please.
A. Yes:
"Thromboembolic (clotting) events.
"In early 2021 there were multiple reports of vaccine-induced immune thrombocytopaenia [that means low platelets] and thrombosis [that means clotting] (VITT) with the adenovirus vector vaccines Vaxzevria [which is the AstraZeneca vaccine, the Oxford vaccine] and Nuvaxovid [which is a Janssen vaccine]. VITT is a severe but rare blood clotting condition; it develops within 5 to 30 days of receiving vaccination."
Q. When you say that there were multiple reports, do we have a figure for those reports?
A. We have one later on. We do, yes. The citation I was using here, which was the British National Formulary,

doesn't particularly mention the figure, but there is a figure, yes.
Q. Yes, okay. We will come to that in due course.
A. Yes.
Q. And as you say, as from early 2023, AstraZeneca was not routinely supplied in the UK, and Janssen, as we've already heard, was initially procured but has never been supplied.
A. Yes.
Q. The second area of safety you look at is myocarditis. Again, if you would just read through that, please.
A. Yes:
"Myocarditis
"The mRNA that is delivered to cells [messenger RNA that is delivered to cells] following challenge with COVID-19 nucleic acid vaccines (i.e. mRNA vaccines) is said to be normally degraded within a few days.
"There have been reports of vaccine-associated myocarditis [that means inflammation of the heart muscle] with all COVID-19 mRNA vaccines."
Again, the quote there is from the British National Formulary:
"Although the mRNA monovalent Spikevax (Moderna) is licensed in the UK for use in children aged [greater than or equal to] 6 Years, current guidance is that the

preferred COVID-19 vaccine for use in children is the mRNA monovalent [Pfizer vaccine], due to a lower reported rate of myocarditis [with the Pfizer vaccine]."
Q. Right. You then go on to what are described as other adverse effects.
A. Yes.
Q. And we will come in due course to look at the Yellow Card system and the report on that.
A. Yes.
Q. What we have here is, I think, a reference to it which is:
"A two-year analysis of Yellow Card reports (i.e. spontaneously-reported vaccine adverse effects), published in 2022 by the [MHRA], documented 2,362 spontaneous reports suggesting a fatal outcome following COVID-19 vaccination; while of concern, the association does not prove causality."
So you are noting there that, through the use of the Yellow Card report, there have been documented over 2,000 spontaneous reports of a fatal outcome. Of course that, as you say, is of concern. It does not prove causality. Is that --
A. It doesn't, no.
Q. Yes.
A. Still, that would be a high number that one would not

expect to see with certainly most vaccines.
Q. Yes. The other adverse events commonly reported are -- include, and you have listed them there: Bell's palsy; Guillain-Barré syndrome, which is ascending paralysis; transverse myelitis, spinal cord inflammation; and menstrual disorders and vaginal bleeding.
A. Yes.
Q. Could you go on then and I think I'll ask you just to read through the final section in this part of your report, the future course of COVID-19.
A. The future course of COVID-19:
"On 5 May ... the World Health Organization declared that COVID-19 [was no longer] a public health emergency of international concern.
"Epidemiological surveillance suggests that SARS-CoV-2 [which is the cause of COVID-19] is now becoming endemic (i.e. the virus is circulating at about the same incidence over a long period of time); endemicity is a feature of the four coronaviruses that have been known for many years to cause mild to moderate respiratory tract illness, including the common cold. Potentially, SARS-CoV-2 can still cause severe illness in those not previously exposed to the virus, either through natural infection or through vaccination.
"It is possible to model the possible future

behaviour of SARS−CoV−2 infection; no model however will be better than the assumptions on which it was built, and disease models are typically phrased in mathematical terms which can make them difficult to understand for the non−mathematician, and also lend them an air of exactitude that they seldom merit."

That's a quote from a great Swedish epidemiologist, Johan Giesecke, who has written a really very good book about infectious disease epidemiology.

Q. I think that's the final footnote, footnote 264.

A. Yes, it is.

MR GALE: Right.

My Lord, I wonder if I could take five minutes simply to rearrange papers and −−

LORD BRAILSFORD: Of course, absolutely.

MR GALE: Before we go into the next set. It will only be a few minutes.

LORD BRAILSFORD: Surely.

Five minutes then, ladies and gentlemen.

(12.37 pm)

(A short break)

(12.39 pm)

MR GALE: Dr Croft, going on to part 3 of your report.

A. Yes.

Q. You deal here with physical measures taken against

COVID−19. To a certain extent, this has already been prefaced in what we've been looking at.

A. Yes.

Q. You begin with a section in block here, and it's headed "Specific knowledge − pre−pandemic". This, I think, derives from the Jefferson 2011 paper Cochrane review.

A. Yes, that summarised this state of knowledge, yes.

Q. I wonder if you would just, first of all, take us through the key messages −−

A. Yes.

Q. −− and then we will look to a certain extent at the supporting statistics. But if you take us to the key messages, they are set out in the block section at page 54 of your report.

A. Yes. Yes. Yes. Thank you, my Lord.

These key messages I cut and pasted from the review itself, in the plain language summary of the review, which is on page 355.

Q. Yes.

A. I think it's right at the very bottom paragraph, page 355, six lines up:

"Respiratory virus spread can be reduced by hygienic measures (such as handwashing) ..."

That's where all of that comes from. So these aren't my words; these are word−for−word transcriptions

of the plain language summary key messages for laypeople, essentially.

Q. Well, perhaps before we do that, we can have a look at the Cochrane review.

A. Yes.

Q. So this is document number 8 within the bundle, and it's at page 354. I think we can see there that the lead author is Tom Jefferson, who was at that time at the Centre for Evidence Based Medicine at Oxford.

Do you know Tom Jefferson?

A. Yes, I do know him. I haven't seen him for 20 years or so, but I know him. He trained at Glasgow in medicine.

Q. I think, just looking at some of the detail of this, the publication status and date, it says that, albeit it's 2011: "Edited ([but] no change to conclusions), published in issue 4, 2020". So there has been some, presumably, iterative updating of the document, but as it says, no change in conclusions.

A. Yes.

Q. Then if we go on to the abstract, we can see that the background is, it says:

"Viral epidemics or pandemics of acute respiratory infections like influenza or severe acute respiratory syndrome pose a global threat. Antiviral drugs and vaccinations may be insufficient to prevent their

spread."

That's the context in which the authors of the Cochrane review were proceeding.

A. Yes.

Q. Then set out are the objectives, and it seems a single objective:

"To review the effectiveness of physical interventions to interrupt or reduce the spread of respiratory viruses."

A. Mm−hm.

Q. We then have a passage on search methods, and I think we can, with respect, pass over that.

Then some information about selection criteria, which may be quite interesting just to read. It says:

"In this update, two review authors independently applied the inclusion criteria to all identified and retrieved articles and extracted data. We scanned 3775 titles, excluded 3560 and retrieved full papers of 215 studies, to include 66 papers of 67 studies. We included physical interventions (screening at entry ports, isolation, quarantine, social distancing, barriers, personal protection, hand hygiene) to prevent respiratory virus transmission. We included randomised controlled trials ... cohorts, case−controls, before−after and time series studies."

And then we go on to see, on the following page at 355, the main results.

Now, these are obviously preparatory to what is contained within the plain language study. I think it might be useful, doctor, if you would just read out what were the main results, so far as the authors were concerned at that time.

A. Yes. So "Main results". This is on page 355, my Lord:

"We included 67 studies including randomised controlled trials and observational studies with a mixed risk of bias. A total number of participants is not included as the total would be made up of a heterogenous set of observations (participant people, observations on participants and countries (object of some studies)). The risk of bias for five [randomised controlled trials] and most cluster–[randomised controlled trials] was high. Observational studies were of mixed quality. Only case–control data were sufficiently homogeneous to allow meta–analysis. The highest quality cluster–[randomised controlled trials] suggest respiratory virus spread can be prevented by hygienic measures, such as handwashing, especially around younger children. Benefit from reduced transmission from children to household members is broadly supported also in other study designs where the potential for

confounding is greater. Nine case–control studies suggested implementing transmission barriers, isolation and hygienic measures are effective at containing respiratory virus epidemics. Surgical masks or N95 respirators were the most consistent and comprehensive supportive measures. N95 respirators were non–inferior [meaning they were the same] to simple surgical masks but more expensive, uncomfortable and irritating to skin. Adding virucidals or antiseptics to normal handwashing to decrease respiratory disease transmission remains uncertain. Global measures, such as screening at entry ports, led to a non–significant [meaning statistically non–significant] marginal delay in spread. There was limited evidence that social distancing was effective, especially if related to the risk of exposure."

Q. Right.

Just as a matter of detail –– and I think we will come to look at this in some detail later on –– N95 respirators, what are they?

A. I would hope they would define them, but they're clearly some kind of advanced respirators which isn't just a cloth mask. They must incorporate some kind of screen.

Q. So it's not sort of an advanced form of surgical mask;

it is something ––

A. No.

Q. And clearly they have the potential, as is observed there, to be uncomfortable and irritating.

A. Yes.

LORD BRAILSFORD: What does respirator mean?

A. A respirator is a gas mask, basically. So it's not just relying on cloth; it's relying on some kind of screen or mesh. Certainly military gas masks, which I'm familiar with, there's a charcoal filter as well. So the air goes through a filter. But it's one step up from surgical masks.

But in any event, they are no more effective than surgical masks.

MR GALE: Right.

Doctor, we go on, then, to the authors' conclusions, which are just in two lines. Would you read those out, please.

A. "Authors' conclusions

"Simple and low–cost interventions would be useful for reducing transmission of epidemic respiratory viruses. Routine long–term implementation of some measures assessed might be difficult without the threat of an epidemic."

Q. Then we have the plain language summary which forms the

basis for what is on page 54 of your report.

A. Yes.

Q. So from whichever source you want, can you read out what the plain language summary and the key messages are.

A. Yes. Shall I just go to the second paragraph, Mr Gale?

Q. Yes.

A. The first paragraph is the background. So second paragraph:

"We included 67 studies ..."

Mixed risk of bias for the observational studies, and that's a reference to the hierarchy of evidence. They are saying there are observational studies, but they are likely to be biased; some of them very biased, some of them slightly biased.

Then they go on to say, third line down –– this is where I take up my direct quotation:

"Respiratory virus spread can be reduced by hygienic measures (such as handwashing), especially around younger children. Frequent handwashing can also reduce transmission from children to other household members."

My third bullet point was the next one:

"Implementing barriers to transmission, such as isolation, and hygienic measures (wearing masks, gloves and gowns) can be effective in containing respiratory virus epidemics or in hospital wards."

I think there are some words missing. I think there were probably some words there, "in clinics or in hospital wards", because they're quite emphatic that the evidence for these barriers to transmission is strong for healthcare environments, but it isn't strong in the community generally.
Q. So we shouldn't just restrict it to hospital wards?
A. Well, we should restrict it, but hospital wards and clinics.
Q. Yes.
A. I think that's what they are saying, yes, but they are saying there's not much evidence that these type of barriers are useful in the community, although of course they're relatively cheap.
"We found no evidence that the more expensive, irritating and uncomfortable N95 respirators were superior to simple surgical masks. It is unclear if adding virucidals [chemicals that kill viruses] or antiseptics to normal handwashing with soap is more effective."
So they're saying probably handwashing with soap is probably as effective as the more complex agents:
"There is insufficient evidence to support screening at entry ports and social distancing (spatial separation of at least one metre between those infected and those

non-infected) as a method to reduce spread during epidemics."
And the reason there is insufficient evidence is probably because high-quality randomised controlled trials haven't been done.
Most of the data is, they admit, taken from case-controlled studies, level IIb evidence, and studies that are even less powerful evidence than case-controlled studies; this is in contrast with the later review, which they focus very much on randomised controlled trials.
MR GALE: Perhaps we can look at the later review after lunch.
My Lord, it might be an appropriate point to pause.
LORD BRAILSFORD: Very good.
Again, we stop a little early, so we will come back a little earlier. About 1.40, please.
(12.53 pm)
(The short adjournment)
(1.40 pm)
LORD BRAILSFORD: Right, good afternoon, everyone.
Mr Gale, when you're ready.
MR GALE: Thank you, my Lord.
Dr Croft, we were looking at the Cochrane review, the Jefferson paper, and we had looked at the plain

language summary which you replicate. It's page 54 of your report.
I wonder if we could just look a little more at that Jefferson paper.
Could you go, please, to page 367 within the paper.
A. Yes.
Q. I think we can see there that there's a summary of the evidence. Again, it's perhaps a slightly arduous task, but I wonder if you would just read through that, so we have that into the notes, please.
A. Yes. So the evidence seems to be mainly based on cluster randomised trials, and we know what they are now. So:
"The highest quality cluster-randomised trials indicate most effect on preventing respiratory virus spread from hygienic measures in younger children. Perhaps this is because younger children are least capable of hygienic behaviour themselves ... and have longer-lived infections and greater social contact, thereby acting as portals of infection into the household ... Additional benefit from reduced transmission from them to other members of the household is broadly supported by the results of other study designs where the potential for confounding is greater."
Shall I carry on?

Q. Yes. Just carry on reading for the extent of the evidence summarised.
A. Sure.
"The pooled case-control studies [and those are level Ib evidence], which focused on the SARS coronavirus ... suggest that implementing barriers to transmission, isolation and hygienic measures are effective with the use of relatively cheap interventions to contain respiratory virus epidemics. We found limited evidence of the superior effectiveness of devices such as the N95 respirator over simple surgical masks. This evidence is supported by a high quality hospital-based trial ... which reports non-inferiority between face barriers [meaning face masks are the same as N95 respirators]. Overall masks were the best performing intervention across populations, settings and threats. More expensive and uncomfortable (especially if worn for long periods) than simple surgical masks, N95 respirators may be useful in very high-risk situations but additional studies are required to define these situations.
"It is uncertain whether the incremental effect of adding virucidals or antiseptics to normal handwashing actually decreased the respiratory disease burden outside the confines of the rather atypical studies,

upon which we reported. The extra benefit may have been, at least in part, accrued by confounding additional routines.

"Studies preventing transmission of [respiratory syncytial virus] and similar viruses appeared to be closer to real life and suggest good effectiveness. However, methodological quality concerns of the controlled before and after studies, mentioned previously, suggest benefits may have been due to population differences, especially virus infection rates. These were poorly reported in most studies.

"Routine long-term implementation of some of the measures assessed in this review would be problematic, particularly maintaining strict hygiene and barrier routines for long periods of time. This would probably only be feasible in highly motivated environments, such as hospitals, without a real threat of a looming epidemic. Most of the trial authors commented on the major logistic burden that barrier routines imposed at the community level. However, the threat of a looming epidemic may provide stimulus for their inception.

"A disappointing finding was the lack of proper evaluation of global and highly resource-intensive measures such as screening at entry ports and social distancing. The handful of studies (mostly conducted

during the SARS epidemic) do not allow us to reach any firm conclusions."

They end by saying:

"It is remarkable that despite a long lead time to the declaration of a pandemic, an international, prospective study to evaluate entry screening practices was not set up. The study by Cowling et al is a good contribution to our evidence base but no substitute for a well designed and conducted trial ... Finally, few studies reported harms from the interventions studied. Harms affect compliance, which may decrease even if the intervention is merely cumbersome (such as a mask) and the threat is unclear."

Q. I think one of the interesting points to draw from that summary is what is contained in the penultimate paragraph in the left-hand column; that the authors note that routine long-term implementation of some of the measures assessed in this review would be problematic, particularly maintaining strict hygiene and barrier routines for long periods of time.

A. Yes.

Q. I think they then go on to say that, in a way, attention might be focused were there to be a more urgent and pressing pandemic problem.

A. Yes.

Q. Obviously, that perhaps leads us conveniently on to where we are now.

A. Yes, indeed.

Q. Would you look, please, at the authors' conclusions at 367 on the right-hand column.

A. Yes.

Q. I think what the authors say there -- they divide it into two sections. One is "Implications for practice" --

A. Yes.

Q. -- and then they go on to "Implications for research".

For present purposes, can we just look at the implications for practice.

A. Yes.

Q. I think what they highlight there:

"The following effective interventions should be implemented, preferably in a combined fashion, to reduce transmission of viral respiratory disease ..."

They then listed:

"1. frequent handwashing with or without adjunct antiseptics;

"2. barrier measures such as gloves, gowns and masks with filtration apparatus; and

"3. suspicion diagnosis with isolation of likely cases.

"Special efforts should be focused on implementing the three above interventions in order to reduce transmission from young children, who are generally the most fecund sources of respiratory viruses."

I think anybody who has had a child will know that getting a child to wash their hands is probably one of the larger difficulties of parenthood.

A. Yes.

Q. For completion, doctor, can we go to pages 464 to 466 within that, so the last few pages in the first volume, my Lord.

A. Yes.

Q. I think there we see the summary. Table 2 is the summary of main events.

A. Yes.

Q. I think we can see the events that were considered by the authors utilising the randomised trials went from handwashing, handwashing with an antiseptic, surface disinfection, gargling with iodine, nose wash, etc. I'm not going to go through them all. But that's a summary in tabular form of the results which they obtained.

A. Yes.

Q. Right.

A. Just commenting on that, obviously the left-hand column is the most reliable evidence, and then there's slightly

less —— the left two columns are the randomised controlled trials, and then as you go to the right, you get less and less reliable evidence.

Q. Could we go to the other Cochrane review, which is document 9. Could we go to page 477. It's the first document in the second volume, my Lord.

Now, this is the Jefferson Cochrane review of 2023.

A. Mm—hm.

Q. And, again, we can see, at page 477, that its publication status and date is that it has been edited, no change to conclusions, and it was published in 2023. No precise date is given.

Again, looking at the abstract, we can see there that there's something that we've already read, but I think it goes further, where it says:

"Viral epidemics or pandemics of acute respiratory infections ... pose a global threat. Examples are influenza (H1N1) caused by the H1N1 ... virus in 2009, severe acute respiratory syndrome (SARS) in 2003, and coronavirus disease 2019 (COVID—19) ..."

So just getting the context, we have material in here which has regard to the circumstances of the COVID pandemic.

A. Yes, we have now. Yes, indeed.

Q. It goes on to say:

"Antiviral drugs and vaccines may be insufficient to prevent their spread."

I think that's something that was also observed in the Cochrane 2011 abstract.

It says:

"This is an update of a Cochrane Review last published in 2020 [which I think we've seen]. We include results from studies from the current COVID—19 pandemic."

A. Yes.

Q. So we have information informed by the experience of that pandemic.

A. Mm—hm.

Q. Again, the objectives:

"To assess the effectiveness of physical interventions to interrupt or reduce the spread of acute respiratory viruses."

A. Yes.

Q. I'm not going to read through them, but there's then selection criteria and data collection and analysis.

If one goes over to the next page ——

A. Yes.

Q. —— 478, we see a passage that begins "Main results". Again, I don't intend to go through that in any detail at this stage, but I think the potential measures are

then grouped: "Medical/surgical mask compared to no masks", "N95/P2 respirators compared to medical/surgical masks", and then "Hand hygiene compared to control".

Then we find the authors' conclusions there, and perhaps you would read through that, Dr Croft, so again we have it in the evidence.

A. Yes. Authors' conclusions from the January 2023 Jefferson updated review:

"The high risk of bias in the trials, variation in outcome measurement, and relatively low adherence with the interventions during the studies hampers drawing firm conclusions. There were additional RCTs [randomised controlled trials] during the pandemic related to physical interventions but a relative paucity given the importance of the question of masking and its relative effectiveness and the concomitant measures of mask adherence which would be highly relevant to the measurement of effectiveness, especially in the elderly and in young children.

"There is uncertainty about the effects of face masks. The low to moderate certainty of evidence means our confidence in the effect estimate is limited, and that the true effect may be different from the observed estimate ..."

I think they mean the calculated estimate of the

effect:

"The pooled results of RCTs did not show a clear reduction in respiratory viral infection with the use of medical/surgical masks. There were no clear differences between the use of medical/surgical masks compared with N95/P2 respirators in healthcare workers when used in routine care to reduce respiratory viral infection. Hand hygiene is likely to modestly reduce the burden of respiratory illness, and although this effect was also present when [influenza—like illness] and laboratory—confirmed influenza were analysed separately, it was not found to be a significant difference for the latter two outcomes. Harms associated with physical interventions were under—investigated."

Finally ——

Q. Just pausing on that, the reference to, "Harms associated with physical interventions were under—investigated", it's not expanded upon there. What do you understand by that comment?

A. Well, "harms" is a broad term encompassing everything from inconvenience to death, and probably also including economic costs and societal costs. That, I think, is what they're getting at; that some of the interventions carry a very modest or almost no risk of harm, and they would probably include —— and almost none —— they would

include, probably, handwashing. They say you don't want to wash your hands too often at one point. So they would carry almost no risk of harms. But other interventions —— and here I think they would include social distancing and —— would have a greater risk of harm.

They talk about the harms of closing international boundaries, and there I think they are just talking about the economic and the harms to people wanting to travel. That's a basic —— a fundamental restriction on our civil liberties. So those are harms that are in a different category.

The specialised respirators, it comes up again and again in the trials that people don't like wearing these close—fitting N95 and P2 respirators. So the harms of that have to be taken into account, even though they might seem to be very effective. Nevertheless, are people really going to wear them when they are not in a highly disciplined environment like a hospital?

So it's a broad term, and this is a feature of randomised controlled trials. The authors want to stress the benefits of the new drug, the new vaccine, the new product that they've investigated, and they tend to downplay the potential harms because that's kind of human nature.

Q. And as I think you've indicated, the harms can range from matters of probably relatively insignificant harm, such as inconvenience ——

A. Yes.

Q. —— irritation, perhaps, looking at the N95 respirators ——

A. Yes.

Q. —— but across to more serious socio—economic harms.

A. Yes.

Q. Again, I think we discussed yesterday that you would see that as being part of your remit ——

A. Yes.

Q. —— as a public health consultant.

A. Yes. Yes. Indeed.

Q. I think we go on to see, again, a plain language summary, and the key messages that are derived from the research by Jefferson and others are those that are reproduced at page 55 in the block section of your report; is that right?

A. Yes, they are. Yes. They only have two key messages there, so I have just transcribed them directly into the report at page 55.

Q. Yes.

I think, again, utilising the plain language summary, there's a list of physical measures set out

there which I think, again, are reflected elsewhere.

A. Mm.

Q. And we also have a passage, "What did we want to find out?", and the authors say:

"We wanted to find out whether physical measures stop or slow the spread of respiratory viruses from well—controlled studies in which one intervention is compared to another, known as randomised controlled trials."

Can I take you, then, to page 510, which is a more expansive view or summary of the authors' conclusions in the left—hand column.

I think we can possibly —— well, again, can I burden you, doctor, with reading through what is said there. I don't think it's necessary to intersperse the various studies, but if you could just read through the text under "Implications for practice".

A. Yes:

"Implications for practice

"The evidence summarised in this review on the use of masks is largely based on studies conducted during traditional peak respiratory virus infection seasons up until 2016. Two relevant randomised trials conducted during the COVID—19 pandemic have been published, but their addition had minimal impact on the overall pooled

estimate of effect. The observed lack of effect of mask wearing in interrupting the spread of influenza—like illness ... or influenza/COVID—19 in our review has many potential reasons, including: poor study design; insufficiently powered studies arising from low viral circulation in some studies; lower adherence with mask wearing, especially amongst children; quality of the masks used; self—contamination of the mask by hands; lack of protection from eye exposure from respiratory droplets (allowing a route of entry of respiratory viruses into the nose via the lacrimal duct); saturation of masks with saliva from extended use (promoting virus survival in proteinaceous material); and possible risk compensation behaviour leading to an exaggerated sense of security ...

"Our findings show that hand hygiene has a modest effect as a physical intervention to interrupt the spread of respiratory viruses, but several questions remain. First, the high heterogeneity between studies [meaning different study characteristics] may suggest that there are differences in the effect of different interventions. The poor reporting limited our ability to extract the information needed to assess any 'dose response' relationship, and there are few head—to—head trials comparing hand hygiene materials (such as

alcohol−based sanitiser or soap and water). Second, the sustainability of hand hygiene is unclear where participants in some studies achieved 5 to 10 handwashings per day, but adherence may have diminished with time as motivation decreased, or due to adverse effects from frequent hand−washing. Third, there is little evidence about the effectiveness of combinations of hand hygiene with other interventions, and how those are best introduced and sustained. Finally, some interventions were intensively implemented within small organisations, and involved education or training as a component, and the ability to scale these up to broader interventions is unclear.

"Our findings with respect to hand hygiene should be considered generally relevant to all viral respiratory infections, given the diverse populations where transmission of viral respiratory infections occurs. The participants were adults, children and families, and multiple congregation settings including schools, childcare centres, homes, and offices. Most respiratory viruses, including the pandemic SARS−CoV−2, are considered to be predominantly spread via respiratory particles of varying size or contact routes, or both ... Data from studies of SARS−CoV−2 contamination of the environment based on the presence of viral ribonucleic

acid and infectious virus suggest significant fomite contamination ... Hand hygiene would be expected to be beneficial in reducing the spread of SARS−CoV−2 similar to other beta coronaviruses (SARS−CoV−1, Middle East respiratory syndrome ... and human coronaviruses), which are very susceptible to the concentrations of alcohol commonly found in most hand−sanitiser preparations ... Support for this effect is the finding that poor hand hygiene, despite the use of full personal protective equipment ... was independently associated with an increased risk of SARS−CoV−2 transmission to healthcare workers in a retrospective cohort study in Wuhan, China in both a high−risk and low−risk clinical unit for patients infected with COVID−19 ... The practice of hand hygiene appears to have a consistent effect in all settings, and should be an essential component of other interventions."

Q. I think I can stop you there, doctor.

The summary of the main results −− and I can simply give the reference to this −− is to be found at pages 770 to 772.

A. Yes, in the tables.

Q. In the tables.

A. Yes.

Q. It's an overall summary. Each area of study also has

a summary as well. So the detail is there and then it is aggregated into a final table.

A. Interestingly, these tables, compared to the previous ones, are focusing on randomised controlled trials. They don't include the others. Not in the tables.

Q. There's one matter that I would like to ask you about, and it takes us back to page 509 in the Jefferson review, which is quite close to where we were previously reading. I think in the right−hand column, towards the top of 509, there's a paragraph which begins:

"The two RCTs of medical/surgical masks during the SARS−CoV−2 pandemic found uncertain evidence of a small or no effect ... The study by Abaluck 2022 found a statistically significant benefit of masks versus no masks for COVID−like−illness, however, this study was rated at high risk of bias for five of the six domains due to issues including baseline imbalance, subjective outcome assessment and incomplete follow−up across the groups. Despite this study contributing 45% of the weight towards the meta−analysis of influenza/COVID−like−illness for masks versus no masks, the updated conclusions from the analysis strengthened around little or no effect of mask use."

A. Yes.

Q. Those two studies, I think you've helpfully reproduced

at I think it's appendix 5.

My Lord, if my Lord wishes −− and anyone else wishes −− the references, the study by Abaluck is in the bundle at pages 952 to 964, and the study by Bundgaard is at pages 965 to 974.

Perhaps we can shortcut that by looking to appendix 5, please.

A. Yes.

Q. Perhaps you could just help us by taking us through what was the Bundgaard −− the Bundgaard study was, I think, in Denmark.

A. It was carried out in Copenhagen, I believe. Yes.

Q. And the Abaluck study was carried out in Bangladesh.

A. Yes.

Q. Could you just take us through what you say, first of all in relation to the Bundgaard study.

A. Yes. Shall I just read it through?

Q. Yes, please.

A. "The Bundgaard study

"The ... study was a randomised controlled trial carried out in Denmark in April−May 2020 (i.e. at the start of the COVID−19 pandemic). At that time, mask wearing was not amongst the recommended public health measures in Denmark.

"There were 6024 participants in the study. They

were community−dwelling adults, previously uninfected with SARS−CoV2, who did not wear masks in their daily work. They were randomised into either (i) wearing a surgical mask outside the home for [more than] 3 hours, or (ii) not wearing a mask, i.e. control group. Testing for SARS−CoV−2 was carried out at 1 month."

So a very simple study, and a nice flow diagram that is in there:

"At 1 month, 42 (1.8%) of the mask−wearing participants tested positive for COVID−19, whereas 53 (2.1%) of the non−mask wearers tested positive. The odds ratio was 0.82 (i.e. suggesting a benefit from mask wearing) ..."

But once this was statistically analysed, the result was not significant. The confidence interval ranged from 0.54 to 1.23. So because the odds ratio crossed the line of no effect, this had to be considered an inclusive study −− an inclusive finding, but an interesting study.

Q. I think we can see that at page 109 of your report in the appendix, "Analysis 1.1: Comparison 1: Randomised trials: medical/surgical masks versus no masks", and I think we can see the Abaluck study is added into the list of studies −−

A. Yes.

Q. −− and is the first in that list. So we can see the material that they were working from at that stage.

A. That's right. The Bundgaard one that we were just looking about, it's in the second lot there, isn't it? The −−

Q. I'm sorry?

A. So Bundgaard, the second block, the third small red square, and you can see how the upper limit of the confidence interval does cross the line of no effect.

Q. Yes.

Go on to the Abaluck study, please.

A. Yes. The Abaluck study, this was a large group of investigators. Most of the investigators came from the United States. There were a few Bangladeshi investigators as well.

They carried out a cluster randomised controlled trial carried out in rural Bangladesh in November 2020 to April 2021. Here, the unit of randomisation wasn't individuals; it was villages. So they enrolled 600 villages in the study, and the villages were randomised into either wearing a mask and being shown a video and a brochure on how to use masks, or else no intervention, ie doing nothing. That was the control group. Then SARS−CoV−2 infection was determined in two ways: partly by self−reported symptoms that were consistent with

COVID−19, and partly by laboratory testing.

"The study authors concluded that the intervention [ie the wearing of a mask and also being shown a video and given a brochure] reduced symptomatic seroprevalence (i.e. a composite measure of positive symptoms and positive antibody tests). The all−age odds ratio was 0.91 ..."

Meaning that it showed a reduction in COVID−19 with an odds ratio of 0.9, so a modest reduction. The confidence interval there just touches 1, so they're only just significant, because an odds ratio of 1 means no effect.

Then I've got here the assessment, what the Cochrane reviewers had to say about these two studies. Shall I carry on?

Q. Yes.

A. So the Cochrane reviewers looked at these two studies, which were obviously of great interest because they were carried out in the pandemic. They interpreted the studies in the context of pre−existing evidence, rather than the standalone studies, which is the correct way of doing it, and they applied the standards of scientific rigour that are routinely used in Cochrane reviews. They used the Cochrane risk of bias tool and, using this, the Bundgaard study was found to be at low to

moderate risk of bias, and the Abaluck study at high risk of bias.

I included in this section of my report part of that −−

Q. Yes.

A. So the Abaluck is at the top, and you can see, my Lord, the five red signs that indicate high risk of bias, and then coming down towards the bottom, Bundgaard is pretty good; it's got three greens, two reds and one uncertain.

Carrying on:

"When the findings of the Bundgaard and Abaluck studies were combined through meta−analysis with the findings of other [previous, pre−existing] medical/surgical mask [randomised controlled trials], they contributed modestly to the overall finding that mask wearing may be effective in preventing the acquisition of SARS−CoV−2 infection − but statistically [using statistical rigour], and because the confidence intervals for the various pooled effect measures, shown below as black diamonds ... in all cases include 1, the results are not significant."

And then I show the forest plot which compares medical/surgical masks versus no masks using only randomised controlled trials, so very high−quality evidence, and the outcome here is viral illness −−

various sorts of viral illness, but including SARS-CoV-2 illness.

So Abaluck is the very top line. It shows they had -- they seem to -- they disaggregated the villages into a number of participants. So they had 111,000 villagers who were wearing the masks and 155,000 who weren't wearing the masks, and you could see there seemed to be a modest effect. It's a large red square indicating a large number of participants. But even then the final -- if you go down eight rows, the black diamond is the pooled estimate of effect, and that plainly does cross the line of no effect.

So pooling all the evidence from all the studies shows with a degree of reliability that there's really no effect, statistically, from wearing a mask versus no mask in the community. That's what we're talking about. We're talking about community studies. For hospital studies and clinic studies, there's no dispute about it; they're good. But in the community, the benefit hasn't been shown.

Q. Now, the reference to Abaluck and Bundgaard are significant, obviously, for inclusion within the 2023 Jefferson Cochrane review --

A. Yes, that's right.

Q. -- in that they are randomised trials taken during the

COVID pandemic.

A. Correct.

Q. You said on a number of occasions that the Cochrane review process is a dynamic process.

A. Yes.

Q. Would one expect there to be further such trials emerging, either at present or have already emerged, and in the near future?

A. Yes. I am pretty sure other studies are emerging. They may even refer to some. Sometimes Cochrane reviews say, "We are aware of other studies going on but we haven't got the findings yet, but we will report about them later on".

Q. So, effectively, what one has, based on Abaluck and Bundgaard, and indeed the other, is a conclusion drawn by the Cochrane reviewers which is static as at 2023.

A. Yes.

Q. As at this year.

A. That's right. Yes, indeed. Indeed.

In fact, it's not as at this year, really, Mr Gale. It's probably whenever the last day they did a search.

Q. Yes. Yes.

A. It's July 2022. But at that point -- that's the point at which they stopped searching and then they present their conclusions.

Q. Yes, point taken. Thank you.

Could we go back to your report, please, doctor, and go back to 3.1. You've listed there -- this is at page 56.

A. Thank you.

Q. You head it as, "Physical measures taken in Scotland against COVID-19".

A. Yes.

Q. You have utilised the list that I think we've already looked at from Jefferson.

A. Yes.

Q. Perhaps you would read on from the bottom of page 56, beginning, "When the COVID-19 pandemic was declared".

A. "When the COVID-19 pandemic was declared, in March 2020, the response of most governments around the world was to safeguard their citizens by simultaneously advocating multiple protective physical measures (sometimes referred to as a 'layered' approach to population protection) that had been deployed in earlier epidemics of acute respiratory illness. This section [of my report] describes how in Scotland, as in most count[r]ies, a wide range of physical measures against COVID-19 was either recommended or else mandated, from early 2020 onwards. Some of the measures were undoubtedly effective. Others were harmful."

In fact, they probably all were harmful to some degree, because even having to buy a mask, you know, harms you. I think they cost a pound. But the harms were obviously more intense with some of the interventions.

Q. Yes.

A. So I give a little comment there.

Q. So your comment, please.

A. My comment is:

"As the pandemic struck, in early 2020, SARS-CoV-2 was treated as an acute respiratory virus. At that time, the best evidence for the effectiveness or otherwise of physical measures to prevent the spread of respiratory viruses was from a decade-old Cochrane review, Jefferson 2011 [we called it Jefferson 1]. This review was updated as the pandemic progressed, and was reissued in revised form towards the end of the pandemic, Jefferson 2023."

Q. Now, what you then go on to do, doctor, in 3.1.1 is list the physical measures advised or mandated in the period from March to July 2020.

A. Yes.

Q. I think, for the sake of brevity, we will take those all as read. I think everybody of our generation will remember the event of 23 March 2020, which is on

page 55, when the then Prime Minister said, "You must stay at home. I give you this simple message", I think is how he prefaced it.

You make a comment about that towards the bottom of page 58.

A. Yes.

Q. Perhaps you would just read that and perhaps expand on it, please.

A. Yes. This, of course, is my own comment as a professional public health physician:

"See the first 'Scientific knowledge' box, above. During March to July 2020 there was limited scientific evidence and in some cases no scientific evidence (e.g. as regards lockdowns) to support the physical measures that were mandated in Scotland against COVID-19. Such evidence as there was (e.g. for mask wearing) mostly came from hospital settings, rather than community settings – and arguably was not applicable to the general, non-hospital population."

Q. You say "arguably"; can you explain the basis of that surmise?

A. Of course, yes. Well, perhaps some policymakers might have extracted one randomised controlled trial that showed quite a strong effect in a hospital and thought: well, our people are disciplined or can be trained to be

disciplined and can be trained or educated to use masks properly, so we will apply that to the general population.

The Cochrane review authors were quite insistent that hospital environments are not the same as domestic environments, and hospital staff could be educated and, to some extent, monitored in the correct use and the consistent use of these inconvenient and uncomfortable measures.

Q. I suppose also it could be considered that there would be an ongoing education process in relation to the general population.

A. Yes.

Q. Particularly with the seriousness of the situation in which we were in.

A. Yes. Yes, that's true. They talk about the need to emphasise the importance of it and the gravity of the threat that was being faced. Yes.

Q. Thank you.

You then go on, in 3.1.2, to list the physical measures advised or mandated in the period between August and December 2020.

A. Mm-hm.

Q. Again, I think we can just take that as read.

Effectively, your comment at the bottom of page 59 is

really the same.

A. It's the same, yes.

Q. Then you talk about, in 3.1.3, the temporary easing of physical measures in the run-up to Christmas, and I think we all remember that there was a great deal of pressure on the policymakers and the politicians --

A. Yes.

Q. -- to allow us to have some form of Christmas --

A. Yes.

Q. -- in 2020.

A. Yes.

Q. I think you've indicated those measures that were put in place by the Scottish Government.

Then at page 60 you comment again, which again is a comment in relatively similar terms to what you've already said.

A. Yes.

Q. Perhaps you would just read it out.

A. Again, my professional comment:

"The easing of the centrally-mandated COVID-19 restrictions over the 2020 Christmas period differed, in different parts of the UK [the different nations]. It is not clear to what extent, if at all, the easing of the restrictions was based on a better understanding of the pathogenicity and transmission characteristics of

SARS-CoV-2."

Q. Moving on, the next period that you're looking at is the period in early 2021, and you list those up to page 61, and obviously this is bearing in mind the terms of the Inquiry's remit, taking it into the date on 22 June when the then First Minister, Nicola Sturgeon, announced:

"... a new indicative date for the whole of Scotland to move to level 0 on 19 July 2021, provided all necessary vaccination and harm reduction measures [were] met."

Again, can we just have your comment on that.

A. So my comment:

"Physical measures intended to restrict the spread of SARS-CoV-2 remained in place in Scotland throughout 2021, and some were still in place in 2022."

There are more milestones, more bullet points, and they are at the appendix to this report. All of those milestones I took from the official timeline that's on the Inquiry website, which is extremely helpful.

Q. Right.

We move on now to part 4 of your report dealing with vaccines, and I think we need to have in mind on this the material that you've already provided us with in section 2 of your report on vaccines.

A. Yes.

Q. Give me a moment.
(Pause)
My Lord, bear with me for a moment.
(Pause)
Thank you, my Lord, apologies.
Right, Dr Croft, we are at page 62 of your report.
A. Yes.
Q. Again, there is a block section in which you note the position post-pandemic, and it's headed, "What are the benefits and risks of vaccine for preventing COVID-19?", and there are certain key messages.
A. Yes.
Q. I would be grateful if you would just read through that, please.
A. Yes. These are taken from the Cochrane review by Graña and colleagues, 2022:
"Key messages
"▪ Most vaccines reduce, or probably reduce, the number of people who get COVID-19 disease and severe COVID-19 disease.
"▪ There is insufficient evidence to determine whether there was a difference between the vaccine and placebo in terms of death because the numbers of deaths were low in the trials.
"▪ Many vaccines likely increase number of people

experiencing events such as fever or headache compared to placebo ([defined as] sham vaccine that contains no medicine but looks identical to the vaccine being tested). This is expected because these events are mainly due to the body's response to the vaccine; they are usually mild and short-term.
"▪ Many vaccines have little or no difference in the incidence of serious adverse events compared to placebo.
"▪ Most trials assessed vaccine efficacy over a short time, and did not evaluate efficacy to the COVID variants of concern."
Q. I think we can find that material at the Graña Cochrane paper, which is paper number 7 and is at pages 50 and following.
I think if we go to page 53 within the bundle, can we just look at some of the accompanying text. Going to page 53, I think we can see the objective of the research at the bottom -- well, let's start logically with the background:
"Background
"Different forms of vaccines have been developed to prevent the SARS-CoV-2 virus and subsequent COVID-19 disease. Several are in widespread use globally."
And then the objective was:
"To assess the efficacy and safety of COVID-19

vaccines (as a full primary vaccination series or a booster dose) against SARS-CoV-2."
So that's the objective.
We then have the research materials and, I suppose, methodology.
If one then goes on to the main results on page 54, I think we can see that the authors:
"... included and analyzed 41 RCTs ..."
A. Yes.
Q. "... assessing 12 different vaccines, including homologous and heterologous vaccine schedules and the effect of booster doses. Thirty-two RCTs were multicentre and five were multinational. The sample sizes ... were 60 to 44,325 participants. Participants were aged: 18 years or older in 36 RCTs; 12 years or older in one RCT; 12 to 17 years in two RCTs; and three to 17 years in two RCTs. Twenty-nine RCTs provided results for individuals aged over 60 years, and three RCTs included immunocompromized patients. No trials included pregnant women. Sixteen RCTs had two-month follow-up or less, 20 RCTs had two to six months, and five RCTs had greater than six to 12 months or less. Eighteen reports were based on preplanned interim analyses."
There is then an indication the overall risk of bias

was low in eight RCTs, while 33 had concerns for at least one outcome.
We then see that, in the main results, the authors divide this up into confirmed symptomatic COVID-19, severe to critical COVID-19, and serious adverse effects. We can obviously read the data if we wish in respect of each of those trials.
But then we have the authors' conclusions at the top of page 55, and I wonder if you would just read that, please.
A. Yes:
"Authors' conclusions
"Compared to placebo, most vaccines reduce, or likely reduce, the proportion of participants with confirmed symptomatic COVID-19, and for some, there is high-certainty evidence that they reduce severe or critical disease. There is probably little or no difference between most vaccines and placebo for serious adverse events. Over 300 registered [randomised controlled trials] are evaluating the efficacy of COVID-19 vaccines, and this review is updated regularly on the COVID-NMA platform ..."
Q. Then I think, significantly, the "Implications for practice". Would you read that?
A. Yes:

"Implications for practice

"Due to the trial exclusions, these results cannot be generalized to pregnant women, individuals with a history of SARS−CoV−2 infection, or immunocompromized people. Most trials had a short follow−up and were conducted before the emergence of variants of concern."

Q. To a certain extent, implications for practice there is excluding certain matters. What would you take from the authors' conclusions and your review of what is contained in the Graña Cochrane review? What would you take as the message for the implication for the practitioner?

A. What I take is the key messages that I extracted and put my report which follow in the plain language summary.

Q. Yes.

A. So I could go straight to them or read them from here.

Q. Yes.

A. So the first message:

"– Most vaccines reduce, or probably reduce, the number of people who get COVID−19 disease and severe COVID−19 disease.

"– Many vaccines likely increase number of people experiencing events such as fever or headache compared to placebo (sham vaccine ...). This is expected because these events are mainly due to the body's response to

the vaccine; they are usually mild and short−term."

Q. Yes.

A. "– Many vaccines have little or no difference in the incidence of serious adverse events compared to placebo."

However, I think there ought really to be a qualifying phrase for that little bullet point, as there is to the next one, so if I read the next one.

Q. Yes.

A. It says:

"– There is insufficient evidence to determine whether there was a difference between the vaccine and placebo in terms of death because the numbers of deaths were low in the trials."

So in the same way, I think really they should have qualified the previous phrase by saying the numbers of severe adverse events were low, and so therefore we can't make a firm conclusion about that.

Q. Other than to note that the numbers were low.

A. The numbers were low, yes. So the numbers were low, so therefore the confidence intervals were wide.

Yes, and then finally:

"– Most trials assessed vaccine efficacy over a short time, and did not evaluate efficacy to the COVID variants of concern."

That's really because many of the trials started before the variants of concern had even emerged so, therefore, they're not to be blamed for that. It was just the way that history worked out.

LORD BRAILSFORD: Can I take you back to page 54, under the heading "Main results".

A. Yes.

LORD BRAILSFORD: You told us, when you were talking about protective measures, that Cochrane reviews did report on the trials underway about which there was no result available at the time of authorship.

A. Yes.

LORD BRAILSFORD: I see in this particular Cochrane report there is an entry under "Main results":

"We identified 343 registered RCTs with results not yet available."

Is that what it's talking about?

A. It is, but if you look at the number of trials that they looked at, they initially identified 600, of which they narrowed it down to 41. So that's not to say that we're suddenly going to be confronted with 343 eligible trials in a year's time. Those numbers will be whittled down because many of them will actually be looking at something else. But they have potentially randomised controlled trials that will provide results in the

future. So there will be ––

LORD BRAILSFORD: You guessed where I was going.

A. Yes. There will be an increase in the evidence. The evidence base will be enlarged.

LORD BRAILSFORD: But not by 343 ––

A. No, maybe by four or five or ten. And, of course, not all those randomised controlled trials are really relevant to the people of Scotland because they are looking at vaccines that were used elsewhere. They look at the whole range of vaccines.

LORD BRAILSFORD: Notwithstanding those caveats, is it likely –– you may not know the answer to this –– because of the likely emergence of a number of randomised controlled trials, that there will be an update to this particular Cochrane review at some stage?

A. Yes, I'm sure there will be a hard copy update, and they are the important ones, because they are the ones that are peer reviewed before they are launched. So there will be, I would guess in maybe two years' time.

LORD BRAILSFORD: Two years' time. Thank you.

Sorry, Mr Gale.

MR GALE: No, not at all, my Lord, thank you.

Let's perhaps indulge in a little speculation, Dr Croft.

The key messages, the first of which is that:

"Most vaccines reduce, or probably reduce, the number of people who get COVID-19 disease and severe COVID-19 disease."

Further research -- and my Lord has just indicated the extent of that that is noted as ongoing, and I appreciate you saying that that will be filtered for those that actually input into a subsequent Cochrane review, as edited.

A. Yes.

Q. Would one anticipate that it may be possible that that terminology might be altered so that it could be increased in its strength, perhaps by removing the word "probably"?

A. Yes, that could be the case, yes. Yes, if enough trials come along which are measuring that particular outcome and are judged to be suitable to include in the meta-analysis. That's part of the catch-22. Not all of them are suitable. What we might be looking at might be the mashed-up vaccines, for example -- not wishing to denigrate them -- so they might not be suitable to compare to genetic instruction vaccines.

But, in general, when looking at an estimate of effect, it doesn't tend to vary -- it doesn't jump around. It tends to sort of move down the same trajectory, but with the confidence intervals getting

narrower and narrower and narrower as more evidence comes in. That's generally what Cochrane reviews find.

Q. Right.

Could you go back to page 62 in your report --

A. Yes.

Q. -- and, "Vaccines procured against COVID-19".

I think, again, this is somewhat repetitive of what you've already said and what we have looked at.

A. Yes.

Q. There are a number of individual -- I think they are called pivotal studies in relation to vaccines, and I think we can see those referred to at page 63.

A. Yes.

Q. My Lord, for my Lord and for the benefit of others, the Folegatti report is paper number 4 --

A. Yes.

Q. -- and is at pages 27 to 34 of the bundle; Sadoff is paper number 17 and is at pages 917 to 930 of the bundle; Polack is number 13 and is at pages 864 to 876; Baden is number 2 and is at pages 5 to 18; and I'll also give the reference to Ramasamy, which was a follow-up report on the AstraZeneca vaccine, which is number 15, and that's at pages 887 to 901.

Now, again, it may be useful, doctor, without actually going through what you actually say in your

text, to go to page 64 and your comment, because you do make a comment on the AstraZeneca study.

A. Yes.

Q. Perhaps you would just read that.

A. This is a comment on -- it's really on Folegatti, the pivotal study, and my comment is:

"Strength of the AstraZeneca study [Folegatti] are that it is a randomised controlled trial. It appears not to be sponsored by industry."

It seems to be an academic study emerging from Oxford University.

"Limitations include (i) the relatively small number of participants ([1,077 in total] 543 people in the vaccine arm); (ii) the use of a different vaccine as the control, rather than saline (this would tend to result in an unrealistically favourable assessment of the study vaccine's true tolerability); (iii) the short period of follow-up ([28 days or] 4 weeks); and (iv) the lack of a study flow chart in the published report (even though this is mandatory [nowadays when you report an RCT])."

Q. Yes. I think that's a reference to a document we looked at yesterday, Altman.

A. It is, yes, and that makes it very difficult to understand what they were doing.

Q. The Janssen vaccine which you refer to at 4.1.2, I think

probably again, with respect, we can ignore, because I think as you've already indicated, it was not a vaccine that was made available in the UK or in Scotland.

A. No, that's right.

Q. One goes on, if I may, then, to the Moderna vaccine at 4.1.3. Again, we can read what is said there. The pivotal study was the Baden paper which I have given reference to. I think we can see there were 30,000-plus participants, and you make a comment on that at the top of page 66.

A. Yes.

Q. Again, if you would read that, please.

A. Yes. So:

"Strengths of the Moderna study are that it is a randomised controlled trial [that's the gold standard of evidence]. It uses a true placebo [the placebo was saline]. The trial incorporates a [nice] study flow chart [so very easy to see what was done]. Limitations include (i) it is an industry-sponsored study ..."

It's sponsored by Janssen, which is a Dutch study. So inevitably, with industry studies, there will probably be some reporting bias:

"... (ii) the differing follow-up periods for different participants groups is confusing."

It's confusing to me, anyway.
Q. I think you said that it was sponsored by Janssen.
A. Yes.
Q. I think this is the Moderna ——
A. I'm terribly sorry. It's sponsored by Moderna.
Q. Top of 66.
A. I beg your pardon, yes. Moderna, which is an American company. I beg your pardon, yes. Janssen was the Dutch company. Moderna is an American company which, as we were saying yesterday, got a lot of money from the US Federal Government for this development of this vaccine.
Q. Yes.
You then go on at 4.1.4 to look at the Pfizer vaccine. The pivotal study is the Polack study, which we've given reference to.
A. Yes.
Q. There's a follow—up study to that, which is Thomas, which I'll give the reference to: it's paper number 19, at pages 937 to 949.
Could you just, again, go to the comment that you make in relation to the Pfizer vaccine.
A. Yes. So:
"Strengths of the Pfizer study are that it is a randomised controlled trial. It uses a true placebo

[which is described as saline, which is good]. It incorporates a study flow chart."
Nice study flow chart on the fourth page of the study, page 867.
Q. That makes it Altman—compliant, if I can put it that way.
A. It makes it Altman—complaint, yes, of course. You can see exactly what they are doing. But interestingly, in the next study, it's pretty much exactly the same flow chart, which isn't what I would have expected, but there we are.
"Limitations include (i) it is an industry—sponsored study ..."
Sponsored by Pfizer, a very wealthy company that invented Viagra. That's why they're so wealthy, and that's why they were able to fund this study entirely without outside interference:
"... (and hence its reporting is liable to commercial bias); (ii) short period of follow—up for the majority of participants."
Oh, shall I carry on with the further comment?
Q. Yes, you make a further comment.
A. Further comment. In September 2021, there was a follow—up study by Thomas, and this was meant to sort of carry the story forward, because as far as we can

tell, all of these four products were given authority to supply, approval to supply —— it's not the same as licensing in the UK —— on the basis that the participants would be studied after two years. So they are meant to be two—year studies. But what happened was that the study participants started to drop out in massive numbers. So what I put here:
"There were multiple drop—outs from the original 'pivotal' Pfizer study; the study in effect had shrunk in size. The follow—up study found that vaccine efficacy declined at 'an average ... of 6% every 2 months' [which hadn't been anticipated in the original study]. The lack of transparency in the data presented by Pfizer in their follow—up study was strongly criticised in an online editorial in the British Medical Journal ..."
Which I cite as Doshi ——
Q. Yes, you've given us that reference and, again, for my Lord and those following, that's document number 3 at pages 19 to 21 ——
A. Yes.
Q. —— of the bundle.
Perhaps, just to understand what the criticism was, can you just tell us what Mr Doshi was saying?
A. I'll refer to his papers.

Q. Yes.
A. It's quite hard to follow some of his arguments, but he seemed to be making a number of, I thought, rather valid points, which is why I included them, my Lord, in the studies, so ...
Q. Yes, I think it's at ——
A. Yes.
Q. —— pages 19 to 21?
A. So what he seems to be commenting on, my Lord, is a pre—print of Thomas 2021, when we would have expected the study flow diagram to continue for another six months, but in fact his study flow diagram is the same as the previous one, which is a bit odd.
So really what he's saying is that really all it's doing is —— all the pre—print is doing, according to Doshi, is measuring —— in fact, again, telling us what vaccine efficacy was at two months. Although it purports to be a study taking the story forward, because of the way they have presented the data, it doesn't actually add any more information.
Dr Doshi is saying waning immunity is a big problem with influenza vaccines.
"If vaccine efficacy wanes over time, the crucial question becomes what level of effectiveness will the vaccine provide when a person is actually exposed to the

virus? Unlike covid vaccines, influenza vaccine performance has always been judged over a full season, not a couple months."

Then Mr Doshi talks about the Israeli experience of ——

Q. Yes, I was just going to draw your attention to that.

A. Okay.

Q. Midway down page 19, just below halfway down page 19, Dr Doshi makes reference to the Israel experience, which I think we did touch on earlier this morning.

A. Yes, and then Dr Doshi says —— we have to take his word for it —— the FDA, the Food and Drug Administration —— I believe he's based in the United States.

Q. Right.

A. He says they expect an approvable vaccine to have at least 50% efficacy. So he says here that the Israelis have found that the Pfizer vaccine efficacy had fallen to 39% when the new variant came along, the Delta variant, and so he considered this to be pretty poor performance.

Then he says: well, okay, what's happened? We've now got this booster. He puts "booster" in inverted commas. I agree with him. I think "booster" is a misnomer. It's actually an odd—on to try and get the vaccine up into the stratosphere. The booster is what

you use when you've already achieved immunity, just to keep the immunity going.

Then he talks about US plans for all fully vaccinated adults to have a booster. Then he says more about —— he says:

"Waning efficacy has the potential to be far more than a minor inconvenience ... the bottom line is that vaccines need to be effective [especially when new variants are circulating].

"... it is unclear whether the 2—dose series ..."

Yes, so he goes back to the two—dose series, the original primary course, which was presented as the solution to COVID—19, and he says: would this even meet the FDA's approval standard of six or nine months for a vaccine?

So I think what he's saying is, in a way, we were sold a pup, in a way. They were sold a product that wouldn't have met the ordinary FDA regulations under normal circumstances.

Q. So I think essentially what Dr Doshi is doing is it's an online editorial ——

A. Yes.

Q. —— and, like many online editorials, the writer tends to like to pose questions ——

A. Yes.

Q. —— without necessarily providing you with the answer.

A. Yes. We looked yesterday at Fiona Godlee's editorial after the swine flu vaccine. She was pretty angry as well, and he's obviously quite angry.

Q. Yes, okay.

Can we go to 4.1.5 at page 67 ——

A. Yes.

Q. —— which is the COVID—19 vaccination timeline.

Just on the question of timeline, I think you previously indicated that you'd had regard to the Inquiry timeline. I think the timeline you've been given is what's called the SPICe timeline.

A. Yes, it is, yes.

Q. That's a timeline that was provided by the Scottish Parliament.

A. Right. Thank you, Mr Gale.

Q. So you begin at 4.1.5 by saying that:

"On 8 December 2020 the first vaccinations against COVID—19 were given in Scotland to those who would be carrying out the subsequent population—wide vaccination programme; this included both medical and non—medical personnel."

Then the milestone of care home residents and staff were vaccinated from 14 December 2020 onwards, and high—risk clinical groups were offered vaccinations in

early 2021.

A. Yes.

Q. Then you list the various milestones from then right through to the end of 2021.

A. Yes.

Q. I think we can see, as at December 2021, you make reference to it being the one—year anniversary of the first COVID vaccination in Scotland.

A. Yes.

Q. Since then, 4.3 million first doses have been administered, 3.9 million second doses and 1.9 million boosters and third doses have been administered from around 1,200 locations.

A. Mm—hm.

Q. So you then go on to make a comment, and I wonder if you would just read that out, please.

A. Yes. So:

"Comment. The COVID—19 vaccination programme in Scotland continued throughout 2022 and is still in place in 2023. In autumn 2022, MHRA approved bivalent vaccines from Moderna and Pfizer."

So those are vaccines that are designed to provide protection against the original strain of the virus and also the variant strain, one variant strain:

"The vaccination milestones are summarised in

Appendix 9 to [my] report."
Q. I think it's actually appendix 10 ——
A. Oh, is it?
Q. —— but it makes little difference. We can easily find it.
Right. You then go on at 4.1.6 to refer to vaccine adverse events reported in the UK.
A. Yes.
Q. I think we can probably work it out, but what do you regard, or what do you and other public health practitioners regard, as a vaccine adverse event?
A. Yes. There's a fine distinction between an adverse event and an adverse effect. An adverse effect is considered to be indisputably linked to the drug or vaccine being considered.
An adverse event will be something that's reported, usually in the early days, but often later on, in the history of a drug or a vaccine. An adverse event is simply practitioners —— healthcare practitioners, doctors or nurses —— and nowadays members of the public can report any untoward physical or psychological or mental problem that they consider is linked to the prior taking of the drug or use of the vaccine.
Q. And that's, in part, the Yellow Card referral.
A. It's done through the Yellow Card system, yes. For

doctors there was always, at the back of the British National Formulary, a Yellow Card pre—printed, and if a patient to whom you prescribed a drug —— and it would usually be a kind of novel drug —— had a funny reaction to it, and you thought, "This is odd, I need to report that to the precursor of the MHRA", you would fill out the card and send it off. There was always a lot of under—reporting, but responsible doctors would try and report adverse events, even though they may not even become aware of them. Some patients would die —— you might give them a drug, they might die, and you might just never really link it to your having given them a particularly powerful drug. That's part of the difficulty of spontaneous reporting systems.
Q. Go to the bottom of page 69 of your report and what is said at 4.1.6. Perhaps you would just read from the beginning of that. Some of it, I think, is material you have now already covered or alluded to, but perhaps you could just read what you say.
A. So "On 1 December" onwards:
"On 1 December 2022 the UK Medicines and Healthcare Products Regulatory Agency (MHRA) published a summary of the spontaneously—reported adverse events (Yellow Card reporting) that had been received by the agency between 9 December 2020 to 23 November 2022 ...

"The December 2022 MHRA publication ..."
I've got this here. This is what it looked like, my Lord. So it:
"... lists 2,362 Yellow Card reports with a fatal outcome ..."
In other words, where the person had received a vaccine and they had died, and somebody, maybe a doctor or a relative, reported it as linked to the vaccine.
Of these, 1,044, or 47%, were in females, and 1,189, in other words 53%, were in males. Of these reported fatalities, 809, 34%, occurred in people who were aged less than 69 years. So relatively young people.
And then:
"During the 2—year period of assessment [by the MHRA], vaccine—associated adverse events (other than fatal events [so non—fatal events]) were reported ... as follows ..."
With AstraZeneca COVID—19 vaccine, a huge number of reports: 246,866; with Moderna, there was fewer: 47,045; and with Pfizer, there was somewhere in between: 177,900. So a very large number of reports.
What I couldn't get from the report was how many doses had been given, what's the proportion of the number of doses given. But just taking those as ball numbers, very high numbers, and far more than you would

expect to see with vaccines.
So my comment here:
"A reported adverse event from a drug or vaccine (e.g. sudden death) does not prove causality — although adverse events that are reported frequently and consistently often do point to a true causal association. The risk of vaccination need to be weighed against the risks of severe COVID—19. Historically, the UK's Yellow Card system for reporting adverse events has resulted in under— rather than over—reporting; the MHRA's December 2022 report on the potential harms of COVID—19 vaccines may therefore have underestimated the scale of the vaccine—associated harms, rather than overestimating it."
Shall I carry on.
Q. Please carry on, yes.
A. "Aside from fatal events, analysis by the MHRA of two consecutive years ..."
So basically they were looking at 2021 and 2022 up until November, and then basically they stopped analysing:
"... suggests [strongly suggests, I'd say] that COVID—19 vaccination may cause an increased risk of the following serious adverse events."
And then I list ——

Q. You list them.
A. And these are ones that they themselves have highlighted as coming up a lot. So:
"▪ Anaphylaxis (i.e. immediate−onset, life −threatening allergic reaction) ..."
They received 990 reports, mostly with the AstraZeneca product.
"▪ Bells' palsy (i.e. unilateral facial nerve paralysis) ..."
So half of your face becomes paralysed. They seem to imply that they get a lot of reports of that, but the numbers weren't in the report; at least I couldn't find them. They say it's continuously reviewed. That is what they are saying. Although at the end of the report they tell us they're stopping routine reviewing, which is a bit odd.
"▪ Guillain−Barré syndrome (i.e. ... paralysis of the lower limbs) ..."
It's occasionally seen with other vaccines, but with these particular vaccines seems to have been seen quite often. Again, the actual numbers were not disclosed in this report; at least I couldn't find them.
Immune thrombocytopenia. That sounds pretty grave, because that means you've got very few platelets, so you're going to have severe problems with clotting. You

won't be able to clot your blood if you cut yourself.
Then major thromboembolic events. That means very serious life −threatening blood clots. There were 486 reports to the MHRA of these, mostly with AstraZeneca, although, again, we don't know what proportion of the vaccines administered were AstraZeneca. But assuming they were one−third, they clearly have been strongly associated with AstraZeneca.
Menstrual disorders. A huge number of reports to MHRA, 51,000, and the MHRA says they were "mostly transient [and we] will continue to review [this problem]".
Myocarditis. That's the inflammation of the heart muscle. Again, very large number of reports to MHRA, in my view: 1,241 in total, and 15 of these were reported as having a fatal outcome. The MHRA comment is the "reports ... are being monitored closely".
Pericarditis, which is inflammation of the fibrous sac surrounding heart. Again, a very large number of reports to MHRA which were associated with the vaccine, at least in the people reporting them. 954 of these.
Finally, transverse myelitis, which is rather rare −− in fact, very rare −− inflammation of the spinal cord, going right across the spinal cord, would cause paralysis of the limbs, and there were 179 reports in

total; again, most of these originating from vaccination with the AstraZeneca product. The comment from MHRA was −−
Q. Perhaps can we just −− I'm sorry, I think you're going on to the comment on transverse myelitis.
A. Yes.
Q. That, "the product information has been updated".
A. Yes. Yes, indeed.
MR GALE: Can we just stop there, doctor, because we have to be mindful of the burden on the stenographers, and I think we've passed our normal time. So perhaps we can just stop there and we will return to finish in a few minutes.
LORD BRAILSFORD: Very good. Thank you.
(3.11 pm)
(A short break)
(3.30 pm)
MR GALE: Dr Croft, just another few hours.
Can we go to page 71 of your report, please.
A. 71.
Q. You've got a comments section. I think that's as far as we had got −−
A. Yes.
Q. −− in your read−through. Would you read the comment section in its entirety, please.

A. Yes. Comment:
"See the 'Scientific knowledge' box, on Page 62 of this report."
And that was summarising the key messages from Graña ...
(Interruption to the live stream)
"... of COVID−19 vaccines states that it is unclear as to whether or not vaccination has made any difference to the numbers of deaths from COVID−19 [and there is a quote from the Cochrane review:] ('there is insufficient evidence to determine whether there was a difference between the vaccine and placebo in terms of death because the numbers of deaths were low in the trials'); future updates of the review may resolve this important point. Minor adverse events (e.g. fever, headache) occur commonly with many of the currently−available COVID−19 vaccines. For many of the currently−available COVID−19 vaccines, serious adverse events (e.g. cardiac and neurological events, and sudden death) appear to be few, based on the reported RCTs. However this apparently low number may be due to (i) the very short follow−up period in many of the reported vaccine RCTs, (ii) the fact that the candidate vaccine was not compared against a true placebo, (iii) the number of participants in the reported RCTs was small, (iv) the

RCT participants were optimally healthy at time of vaccination, or were otherwise unrepresentative of the majority of the UK population, or (v) a combination of any or all of the foregoing. In early 2023 MHRA announced that it would no longer be issuing special publications on the spontaneously-reported adverse events associated with COVID-19 vaccines."

Although they do say -- so this is the last of its kind -- that they will report on the autumn booster for 2023.

"The reasons for this announcement are unclear. Around the same time, and for reasons that are also unclear, the December 2022 report [that's the two-year report, this one] ... was removed from the agency's website."

At least I couldn't find it when I tried to find it on there. By good fortune, I downloaded it, I think, around about Christmas, when it came out, so I had a copy.

Q. Finally, in relation to this, can we go to the MHRA document, please.

A. Yes.

Q. This is a summary of Yellow Card reporting published on 1 December 2022, as you have just said.

Can we go to page 819 within the second bundle of

documents, please.

815 shows us the cover sheet:

"Coronavirus Vaccines.

"Summary of Yellow Card reporting.

"Published 1 December ..."

And the data included data from 9 December 2020 to 23 November 2022.

A. Yes.

Q. Can we go to page 819 within that document.

A. Yes.

Q. I think we can see there this is the summary section.

A. Mm-hm.

Q. Reading the first four paragraphs I think is perhaps of particular interest, and I'll just read those to you:

"Over the first 27 months of the pandemic over 178,397 people across the UK have died within 28 days of a positive test for coronavirus ... Vaccination is the single most effective way to reduce deaths and severe illness from COVID-19. A national immunisation campaign has been underway since early December 2020."

Now, again, as a public health consultant and epidemiologist, is that a general statement with which you would agree?

A. Thank you, Mr Gale. If I could refer you back to Altman and his statement. So that is quite a powerful

statement. Doug Altman says -- that's in the bundle of documents, my Lord -- only randomised trials allow valid inferences of cause of effect. Therefore, I would expect that statement there -- "Vaccination is the single most effective way to reduce deaths and severe illness" -- to be supported by the evidence from randomised controlled trials. But that isn't what the randomised controlled trials show at the moment. It may do at some point in the future.

Q. It may do at some stage in the future.

A. Yes, they may do.

Q. Right.

Then the document goes on:

"Three COVID-19 vaccines - the ... Pfizer/BioNTech ... AstraZeneca and ... Moderna - were used in the primary and booster vaccination campaigns up to the end of August 2022. All have been authorised for supply by the ... (MHRA) following a thorough review of safety, quality and efficacy information from clinical trials. In clinical trials, these vaccines showed very high levels of protection against symptomatic infections with COVID-19. Data are available on the impact of the vaccination campaign in reducing infections, illness and mortality in the UK."

Then it goes on:

"The MHRA confirmed on 9 September 2021 that the COVID-19 vaccines made by Pfizer and AstraZeneca can be used as safe and effective booster doses. Following a review of the data for the COVID-19 Vaccine Moderna vaccine, the MHRA and Commission on Human Medicine ... experts also concluded that this vaccine can be used as a safe and effective booster dose. All vaccines and medicines have some side effects. These side effects need to be continuously balanced against the expected benefits in preventing illness."

A. Yes.

Q. If one turns over the page to 820, there's separate comments on each of the vaccines, the Pfizer-BioNTech, AstraZeneca and Moderna.

A. Yes.

Q. And then towards the bottom of that page, if one can see penultimate paragraph:

"The MHRA continually monitors safety during widespread use of a vaccine. We have in place a proactive strategy to do this. We also work closely with our public health partners in reviewing the effectiveness and impact of the vaccines to ensure the benefits continue to outweigh any possible side effects."

A. I would like to know who these public health partners

are because it's not really explained, but it's interesting ——

Q. To a certain extent you take that on trust.

A. We have to take that that on trust, yes, because the MHRA isn't really resourced to conduct epidemiological analysis of the reports coming to it, not really. They count the reports. They are not like NICE, National Institute of —— who do. They can assess the benefits and harms of new drugs. MHRA isn't really like that. They will depend on other people to do that for them.

Q. If one goes to page 821, in the first full paragraph I think we can see that as at the data date, 23 November 2022, 17,965 Yellow Cards have been reported for the Pfizer, 246,866 have been reported for AstraZeneca and 47,045 for Moderna.

If one then goes to the bottom of that page, three paragraphs from the bottom:

"For all COVID-19 vaccines, the overwhelming majority of reports relate to injection-site reactions (sore arm for example) and generalised symptoms such as 'flu-like' illness, headache, chills, fatigue (tiredness), nausea (feeling sick), fever, dizziness, weakness, aching muscles, and rapid heartbeat. Generally, these happen shortly after the vaccination and are not associated with more serious or lasting

illness."

Again, to the bottom of that page, the last sentence:

"Overall, our advice remains that the benefits of the vaccines outweigh the risks in the majority of people. Further comments on use in specific populations and details on the specific safety topics can be found within Section titled Analysis of data."

Then we have a conclusion section there. Again, we have a very generalised statement, which is:

"Vaccines are the best way to protect people from COVID-19 and have already saved tens of thousands of lives. Everyone should continue to get their vaccination when invited to do so unless specifically advised otherwise.

"As with all vaccines and medicines, the safety of COVID-19 vaccines is being continuously monitored.

"The benefits of the vaccines in preventing COVID-19 and serious complications associated with COVID-19 far outweigh any currently known side effects in the majority of patients."

And then there's a reference to further information.

So, again, can I just ask you on the first part of that concluding section, is that something with which, in general terms, you agree?

A. Well, no, because again the first part —— it may be correct, but it's not demonstrated by the Altman rule, which is that only randomised trials allow valid inferences of cause and effect, and the randomised trials don't show that the vaccines have that effect in saving lives, in preventing deaths.

Q. Would it be right in saying, doctor, that it doesn't satisfy your test and standard, but it might satisfy others?

A. Well, it isn't my personal test. It's the professional test that all epidemiologists would use before making those kind of categorical statements, yes.

Q. But other epidemiologists might have a more liberal approach, if I can put it that way, to adherence to the Altman rule, so you might find others who disagree with you.

A. Possibly, yes. I'll just say again what the Cochrane review says, and that's:

"There is insufficient evidence to determine whether there was a difference between the vaccine and placebo in terms of death."

That has to be the —— a valid inference of cause and effect.

(Pause)

Q. I think we do have some data at pages 825 and 826.

Perhaps if we just look at that.

Table 1 discloses those who have received a first dose of the vaccine for COVID-19 up until 11 September 2022.

A. Yes.

Q. For Scotland, the figure is 4.5 million, second dose is 4.285 million, and those who have received a third or booster, again in Scotland, is 3.5 million. I think these are figures you've given already ——

A. Yes.

Q. —— in the terms of your report.

A. Yes.

Yes, just to clarify what I've just said, my Lord. So it's correct that deaths started to decline, if you like, as the pandemic progressed, but there may be other reasons for that, including what we talked about earlier, which was the better treatment protocols that weren't contributing to the patient's morbidity. That's just the obvious reason. So just to say, "Well, the deaths were declining at the same time as the vaccines were being given to people" is not scientifically sound, in my professional opinion.

Q. Could I, just in conclusion, ask you to look at page 855 within that document.

A. Yes.

Q. I think these are tables 11 and 12 which show, first of all, the number of UK reports with a fatal outcome ——
A. Yes.
Q. —— received for COVID—19 vaccines by a patient up to and including 23 November 2022.
A. Yes.
Q. So for all vaccines the total is 2,362.
A. Yes.
Q. But, again, causality is not being established here.
A. Of course. Indeed. And the true number may be higher. It's likely to be higher based on historical under—reporting. Or it may be lower because there may have been over—reporting.
Q. Yes.
A. But it's what has been reported. That's the data as we know it.
Q. It is possible that it's just simply a coincidence ——
A. Yes.
Q. —— between particularly, perhaps, an elderly person who has been vaccinated and subsequent death.
A. Potentially, although if you see very many of the —— very large numbers are in the middle—aged and young groups there, which wouldn't tend to give that kind of pattern of reporting.
Q. I see.

I think also in table 12 you have that stratified into male and female.
A. There doesn't seem to be much of a difference between male and females. Actually, slightly more males, actually: 53% are males, 47% are females. So there is a difference there, yes.
Q. I think we can inform ourselves on that if we look at the accompanying text at 854.
A. Yes.
Q. If one goes to the penultimate paragraph, you see:
"A report with a fatal outcome on the Yellow Card scheme does not necessarily mean that it was caused by the vaccine ..."
A. Yes.
Q. "... only that the reporter has a suspicion it may have been. Underlying or previously undiagnosed illness unrelated to vaccination can also be factors in such reports. The relative number and nature of UK reports with a fatal outcome are subject to many factors that influence ADR reporting. They should therefore not be used to directly compare the safety of the different vaccines."
A. Yes, I would agree with the last sentences.
Q. Could we just go finally to the conclusions on page 858. These are essentially the same conclusions that we've

seen in the summary and, again, I draw your attention again to what is said at the penultimate paragraph:
"The benefits of the vaccines in preventing COVID—19 and serious complications associated with COVID—19 far outweigh any currently known side effects. As with all vaccines and medicines, the safety of COVID—19 vaccines is continuously monitored, and benefits and possible risks remain under review."
Again, I understand the clarification that you've made in relation to that, a statement of that generality.
A. Mm—hm.
Q. And I presume you would make it again in relation to this?
A. Yes. Yes.
Q. Right. Doctor, you will be pleased to know we're nearly there.
A. Thank you.
Q. Can we go to section 5 of your report, please. This is at page 73 and following. This is a summary, "what do we know now?"
So do I take it really here you are giving, from your perspective of a consultant public health physician and as an epidemiologist in particular, what is your professional, informed opinion?

A. Indeed, yes.
Q. And these statements are statements which, in your view, are borne out by the material that you have considered ——
A. Yes.
Q. —— and looked at?
A. Yes, Mr Gale.
Q. So on that basis, doctor, would you just read through that section, please.
A. So:
"Summary — what do we now know?
"The COVID—19 pandemic of 2020—2023, caused by a novel coronavirus, was a national emergency which threatened the lives of certain groups in society: the very old, and the very sick [but especially the very old].
"Other groups (children [by which I mean healthy children], healthy young adults) were not ever at risk of severe disease.
"By early 2023 the pandemic had abated but there were reports of many people with long COVID and of other people with long—term cardiovascular sequelae of COVID—19 infection."
So some bullet points about physical measures against COVID—19. These, I hope, all arise organically

from what has been said earlier in the report. So the first bullet point:

"▪ From March 2020 onwards, and in common with many other governments, the Scottish government recommended or mandated a range of physical measures intended to limit of spread of SARS−CoV−2, the novel coronavirus which was the cause of COVID−19.

"▪ The physical measures recommended or mandate by the Scottish government ranged from simple public health practices (the encouragement of frequent handwashing, cleaning of environmental surfaces, the use of PPE in hospitals and care homes) to coercive and/or intrusive measures (face mask mandates outside of healthcare settings; lockdowns [and there's a definition for that]; enforced social distancing; test, trace and isolate measures).

"▪ In 2020 there was scientific evidence to support the use of some of the physical measures (e.g. frequent handwashing, the use of PPE in hospital settings) adopted against COVID−19.

"▪ For other measures (e.g. face mask mandates outside of healthcare settings, lockdowns, social distancing, test, trace and isolate measures) there was either insufficient evidence in 2020 to support their use − or alternatively, no evidence; the evidence base

has not changed materially in the intervening three years [even though there have been two additional reports about face masks].

"▪ It has been argued that the restrictive measures introduced during the COVID−19 pandemic resulted in individual, societal and economic harm that was avoidable and that should not have occurred."

And in fact that's now one of the textbook standard points that seems to be indisputable.

So "Vaccines against COVID−19". We have eight bullet points. Firstly:

"▪ Vaccines against COVID−19 became available to the UK general public in [January] 2020; initially only the high−risk groups (the very old, the very sick) [also healthcare workers] were targeted.

"▪ All the COVID−19 vaccines procured by the UK government during 2020 and 2021 were nucleic acid vaccines using novel gene technology."

But more recently they had procured a couple of conventional vaccines.

Thirdly:

"▪ As additional vaccine supplies became available, vaccination was extended to young, middle−aged and elderly adults, and to children."

Q. Can I just stop you, doctor. There is one small

typographical error but it might be seen as being significant. In the first bullet point I think the date should be January 2021.

A. Yes. Yes, thank you. Thank you, Mr Gale. Yes. Thank you so much.

So fourth bullet point:

"▪ Vaccination against COVID−19 became a prerequisite of travel to many countries, and some UK employers made it obligatory for their workforce.

"▪ It remains unclear as to whether or not COVID−19 vaccination has resulted in fewer deaths from COVID−19.

"▪ COVID−19 vaccines have been shown in randomised controlled trials to be effective, or probably effective, in reducing the number of people acquiring COVID−19 or severe COVID−19; however vaccine−induced protection against COVID−19 is short−lived."

So there's waning immunity which is not mentioned at all in the MHRA report, I should add.

The last point:

"▪ Because of the antigenic variability of all coronaviruses [this concept of them being a moving target], including SARS−CoV−2, it was foreseeable that COVID−19 vaccines would only provide short−term protection against COVID−19 (as is the case also with current vaccines against seasonal influenza).

"▪ Because the novel gene technology vaccines procured by the UK government had been tested on relatively small study populations, and had been assessed for safety over short follow−up periods only, rare and sometimes serious adverse effects (including reported fatal events) emerged, once the vaccines had been used on a mass scale in the UK and in other countries."

Q. Doctor, there's one point in your summary that I would like to touch on with you, and that goes back to that first section at page 73.

A. Yes.

Q. It's the second paragraph, the short paragraph.

A. Yes.

Q. "Other groups (children, healthy young adults) were not ever at risk of severe disease."

A. Yes.

Q. Now, I think I understand the context in which you're saying that −−

A. Yes.

Q. −− but I think you are saying that in the −−

A. In a population −− in a population −−

Q. −− context of a public health practitioner looking at large −−

A. That's right, yes.

Q. And of course we know ——
A. Yes.
Q. —— and we will hear and we know of instances where children and healthy young adults were at risk of severe disease?
A. Right, yes, of course.
MR GALE: Doctor, you will be pleased to know that is all I have to ask you. If his Lordship wishes any clarification, but at the moment, thank you very much for the report that you have provided and the work that you have done, and on behalf of the Inquiry, I thank you.
THE WITNESS: Thank you.
LORD BRAILSFORD: I'm happy to say I've got nothing I wish to ask you. Thank you very much. I simply repeat what Mr Gale has said.
THE WITNESS: Yes.
LORD BRAILSFORD: This is, of course, simply the first stage in the presentation of scientific evidence. There will be a considerable volume, at the moment we can't be sure exactly how much further evidence of science or epidemiology in relation to COVID—19, and what we've heard today will no doubt figure in the evidence we hear in the future because, of course, all subsequent experts who give evidence in whatever capacity will have the

opportunity to see what you've written. But in the meantime, thank you very much indeed. I'm very grateful.
So far as the audience is concerned, our thanks to you for attending for two long days. I'm very grateful. And, as I suppose they say in show business, I look forward to look seeing you on 28 August at Murrayfield.
Thank you all.
(4.00 pm)
(The hearing adjourned until Monday, 28 August 2023)

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