GENERAL MEDICAL COUNCIL

FITNESS TO PRACTISE PANEL (MISCONDUCT/PERFORMANCE)

On:

Friday, 13th July 2007

Held at:
St James's Buildings
79 Oxford Street
Manchester M1 6FQ

Case of:

GORDON ROBERT BRUCE SKINNER MB ChB 1965 Glasg SR
Registration No: 0726922
(Day 10)

Panel Members:
Mrs S Sturdy (Chairman)
Dr M Elliot
Mr W Payne
Mrs K Whitehill
Mr P Gribble (Legal Assessor)

MR A JENKINS, Counsel, instructed by RadcliffesLeBrasseur, Solicitors, appeared on behalf of the doctor, who was present.
MR T KARK, Counsel, instructed by Eversheds, Solicitors, appeared on behalf of the General Medical Council.

Friday, 13th July 2007

(9.30 am)

THE CHAIRMAN: Good morning, everyone. I'm Sandra Sturdy, chairing the Fitness to Practise Panel hearing enquiring into the allegation against Dr Skinner. Just to remind you, it is important to maintain the anonymity of the patients, and should they be mentioned in error, please do not refer to them out of the room.

Again, mobile phones, I'm sure everyone has got them off, and if do you need to talk, please leave the room.

We will proceed, Mr Jenkins, with the evidence of Dr Hertoghe.

Housekeeping

MR JENKINS: Madam, thank you very much. Can I just raise a couple of matters of housekeeping. I don't know whether Dr Hertoghe's evidence will be finished today. If it is not, we're trying to make arrangements to ensure that the Panel can receive his evidence next week. He has other commitments, both family as well as professional, and it would be his wish to return to Brussels today, plainly, to be with his family over the weekend.

We are trying to make arrangements to ensure that there could be a video link if he has not finished

giving evidence today. That video link facility can be in place for Monday. I know that would require the GMC to consider what resources are available at this end but we will try, during the morning, to ensure that we can identify a suitable video link facility in Brussels.

THE CHAIRMAN: I think that could be a good solution, but we are aware of how they do break down, and I do think that would cause quite an interruption to the hearing if it didn't work. Thank you.

MR JENKINS: I agree and I understand that we can test it this afternoon, once we find a suitable link at the other end.

The other point is Dr Hertoghe's travel arrangements for today. It is not at this moment entirely clear whether he should be taking a flight to Brussels or whether it would be more efficient, in terms of the length of time he can evidence, to do the whole journey by train, London and then the Eurostar. I wonder if we can review that during the day. Having raised those matters I'll keep going, if I may.

DR THIERRY HERTOGHE (continued)

Examination-in-chief by MR JENKINS

MR JENKINS: Dr Hertoghe, we were dealing with your report looking at the reports of Dr Lynn and Professor Weetman, the document that the Panel have is D13, and we were on

page 6 yesterday.

A. Yes. Do you want me to continue?

Q. Please do, yes.

We had talked about the UK consensus and that Professor Weetman's view was that the goal of treatment is to normalise the TSH level in blood, and you've pointed out to us that that is going against the UK consensus, which he had mentioned in his report, which said that the correct dose is the one which relieves symptoms but may result in a high T4 and a low TSH.

A. Yes. There are too many conditions that influence the TSH to make it test that on its own. It's sufficient to follow up. I'll even show a slide again that I showed yesterday, but I'll not show it now, where you see that even if you have a good TSH during the day, during the night, it is test again hypothyroid, but during treatment if we use only the TSH during the day as a test.

Now, it's also in contradiction with Dr Weetman's view. I showed it yesterday anyway, that he talked about that we shouldn't rely actually on the TSH only. So he made an editorial, one or two months before testifying here, that says actually the contrary.

So also his publications, I think I had those two publications where he talks about actually where the

reference range one publication says that the reference range should probably be narrower, as a hypothesis, that was years ago, and now he talks that we should also treat patients, whatever their TSH is, if they have complaints that could be a possibility.

Q. Could you pull the microphone slightly closer to you?

A. Yes.

Now, I showed also on the TSH most of the information yesterday, so I don't think it's necessary to go through, I just repeat that study on biochemical test of thyroid function, any value in monitoring patients receiving thyroxine replacement where they saw that the tests were poorly useful, but I also mentioned that probably one of the reasons is that the patients take in the morning their thyroid hormones before, and that falsifies all the results later on, so you may not take the thyroid hormones before a blood withdrawal, it's a standard, and it's also in the official textbook for thyroid function. I think you have found that part.

Q. We can look at it, it's page 865 of Mr Lynn's textbook, but we may look at it later.

A. He went later on to talk about the fact that the TSH is useful is that there is an increase in atrial fibrillation and bone loss if the TSH is too low. There's a big difference between a low TSH during

treatment and a low TSH that is permanent. The studies that have shown a link, like I showed yesterday, between atrial fibrillation and TSH, were studies done where the TSH is always low because they have a permanent situation of excess thyroid hormones.

When you take thyroid therapy, during the day you have a decrease of the TSH, but in the study that I showed yesterday, when you look well at the study, the whole night and even the whole evening, their TSH is back to the usual condition, that means with a dose of 121 micrograms -- I'll probably show the study because it's too important, in my judgment.

I will give you a copy of that. It's important to show because it will make a lot ...

The dose where we suppress the TSH, the mean dose, I didn't indicate that here, it was 121 micrograms per day. And we look at those patients, they are clearly above the TSH even for UK, but we see that it lowers and it becomes still in pathological values. You still have here a sort of situation where there are increases in risk factors. You get easier fat, you have more insulin resistance, you have arteries get stiffened so they have a permanent atherosclerosis in this range.

So these patients are under treated during the day, but still they are better than without treatment. And

it's only 121 micrograms but remember Dr Lynn said we should probably not exceed 100 micrograms per patient. So these patients were exceeding that value.

It's still clearly hypothyroid and still remained in a sort of situation that increases risk factor, but look what happens after 7 o'clock in the evening. Their value is higher.

So just doing a test during the day without looking at the patient, without taking into account -- and I showed you that the clinical signs are easy to recognise. They're very typical for low thyroid.

I could have given you a lecture where I would have compared deficiencies in male hormones, in female hormones, in cortisol, in growth hormone, in melatonin, all these hormones, you will see that the clinical picture, the physical appearance is different from patient to patient because of the different deficiencies.

Q. Can I just read to you one sentence from this book, the chapter is "Treatment of hypothyroidism", the heading is "Maintenance and monitoring of treatment", and the sentence I want to read is:

"Because serum TSH and free T4 levels are influenced by T4 administration, blood should be collected before the T4 is taken."

Is that the point you were making?

A. Yes.

Q. Again, without marking the book --

A. And it would probably have had much less problems to understand why this happened now.

I just wanted to show you how misleading it is, just the TSH during the day, during a treatment. Clearly a dose of 121 micrograms for these women, 12 women, is insufficient because they remain almost half of the time in overt, clearly clear-cut hypothyroidism.

But you would have checked that, if the physician would have examined the patient and knows the signs -- because it's not taught any more at university. I didn't get any course of that. They showed maybe one or two pictures but they did not really go into details, but if you know very well the clinical picture, you would have known that the dose is too low.

So I think this is a fundamental point.

Q. If we move on in your report to paragraph 14.5 --

A. That I already mentioned that there were several studies I showed where the TSH test didn't seem to be an interesting test.

Q. I understand.

A. And you saw that Professor Franklyn, in her patients, 71 per cent, had suppressed TSH.

Q. The next paragraph, 14.6, again you have taken us through yesterday, issues about atrial fibrillation and what is said to be the risks of bone loss.

A. The only study I saw where treatment with T3, hormone in cardiac operated patients, patients operated for heart problems, cardiac valves, and they had a decreased risk in atrial fibrillation after the operation if they were treated. So those are not necessarily hypothyroid patients, just patients undergoing cardiac operation, probably euthyroid biochemically and receiving a supplement to improve the heart function.

Q. The next paragraph that you have, 14.7, has the heading "A low TSH can be associated with a reduced risk of major disease". I think we have covered that in your presentation yesterday.

A. Yes. I showed that ideally, to be sure you have to be under 0.4 of TSH, in order to avoid recurrence of thyroid cancer or occurrence of thyroid cancer in patients with goitres or nodules.

Q. The next paragraph, 14.9, you discuss the fact that the serum level of TSH undergoes large fluctuations during the day, and that's the point you've just made for us.

A. Yes, the younger the patient, the greater the fluctuations will be. In elderly patients the curve flattens. Part of aging is caused by deficiencies in

hormones, but the other part is caused by chaotic endocrine, so hormones have to be secreted at certain times of the day more than in other times. That's called a circadian rhythm. For many hormones more hormones in the morning, less in the evening, but for TSH it's the other way around, there are higher levels.

These fluctuation, are healthy, but that doesn't mean that the TSH at night may be at 6.00 like in this study I showed you.

Q. Your next paragraph, paragraph 15, on page 8 of your report, you agree with Dr Weetman that overtreatment with thyroid hormones can be associated with adverse consequences.

A. Completely. Excess thyroid hormones is actually a medical emergency. Not always because the risk for your body is so important but because the patient is so anxious with it.

Again, I repeat he's not euphoric. He gets too anxious and this is an unbearable situation for many, anxious from anything. In the long-term, it can cause heart failure, the heart doesn't work enough because when there's too much thyroid hormones, there's a melting of the muscles, the muscles melt, and the heart is a muscle, so they will decrease the structure.

If you stop the treatment that was clearly -- but

you already have to have very high doses, really very, very high doses to cause heart failure. You won't have that at 200 or 250. Normal people, I would say, are in an average hypothyroid patient. We have to have much higher dose.

Q. The question that you raise is whether the levels in Dr Skinner's patients were really excessive levels or whether the readings can be taken as true readings.

A. In my experience, if a patient doesn't take his thyroid hormones before the therapy and all his hormones are high, except the TSH is suppressed, he really has overt hypothyroidism.

But you certainly have to have a high T3, which has not been austere, I would recommend really Dr Skinner to ask if the T3 is really useful, because that is the active hormone. If I have a high T3, I'm sure the person is hypothyroid, and I will see it also clinically, it will be confirmed clinically, I will be able to see.

Here in these patients, the reason I'm so sure that the patients have taken the thyroid hormones before is that they were clinically not hypothyroid. Any doctor would have clearly seen they were hypothyroid. Even, I would say, with the less education that they seem to have here, the GPs that were in the reports and

the endocrinologists.

Q. What you say is that the probability is high, that all or most of the levels in the patients that the Panel have to deal with were not really excessive because there were no clinical signs of excess reported.

A. Yes, but if there were clinical signs, you would have the same sort of picture. So what I mean to say is that the clinical symptoms are extremely important. If they are not present, you're almost sure there's no hypothyroidism, and this is in contradiction with the views of Dr Weetman and Dr Lynn.

But they are not clinical doctors. Dr Lynn is a very good expert for cancer, he's a surgeon and things like, that but we have seen in the reports that they are not knowledgeable about the information about treating patients who are clinically -- also based on the clinical. So it's two schools of thinking, but I think we can say, and I will show, that the experts didn't have the experience to really with knowledge be able to report on these cases of these four patients.

And I'm sure, I'll ready to have all the BTA here, or the ten endocrinologists of the BTA, I'm ready to discuss, but I'm almost sure they will not be able to prove something contrary.

Q. Next paragraph, I think you've already dealt with that

topic, about serum T3 and T4.

A. Yes. Well, I didn't really. Dr Weetman says that serum T3 and T4 on their own would not be recommended to monitor thyroid hormones. I would agree with him, alone they are not enough but you do have to ask them in a follow-up. Again I repeat, if I have a patient who did correctly the blood test, which is apparently not the case in any case, I can have no proof of the contrary that they did their test like they should without take the thyroid hormones before.

But if they had a high T3 and high T4 and low TSH, it's almost sure they were clinically overdosed, and they would have clinical symptoms. In my experience I only had one patient who was normal with high levels of T3 and T4 after taking the blood correctly, but I know she took very high doses. But it's only one patient in my career.

Q. You refer again to the you United States National Academy of Clinical Biochemistry and their guidelines.

A. It's a proposition of guidelines to change the guidelines, it's not an official, but they say evidence begins more and more to tend that thyroid treatment should consist of maintaining the TSH level in the lower half of the reference range, that is between 0.5 and 2, and sometimes it's also cited by other guidelines to put

under 1.5, and I don't know why we should make a difference.

If a patient under treatment has to have his TSH under 2 or 1.5, why do other people who have a TSH above that value may not be treated? Patients between 2 and 4. There's no scientific evidence to my knowledge to make a difference.

I know that many patients, if you examine them very well and they have a TSH above 2 or 2.5 are clinically hypothyroid. We know that if it's in that range, you have a high risk of developing a full-blown clear-cut hypothyroidism. That would be accepted by all endocrinologists.

Q. You then deal with that guideline. I'm looking at paragraph 18.

A. Dr Weetman puts the guideline in question that he himself helped to make. The guideline of the National Academy of Clinical Biochemistry.

He says the evidence would be so far equivocal. There's, I think, 495 references, not only for that topic but for all the guidelines, which is more than the British Thyroid Association. So he puts that into question to strengthen the theory that we should not treat patients with a TSH at 2, at 2.5 and 3.

He says, for instance, that NACB thyroid guideline

would have been subject to detailed international scrutiny prior to the publication. But when you read his report, you have the impression it is against the guideline, that means that there was a lot of discussion.

There is always a lot of discussion to put good guidelines on the Internet or in an official guidelines of an official institute. I think this is normal. This just makes you understand that these guidelines have been thoroughly screened, by 95 examiners.

The British Thyroid Association guidelines have been reviewed by nine endocrinologists that are probably diabetologists.

Q. In paragraph 20 you deal with subclinical hypothyroidism and you comment on Dr Weetman's view that subclinical hypothyroidism without clinical signs, just chemistry outside the reference range, should not be treated.

A. Yes. So the definition of subclinical hypothyroidism is normally hypothyroidism without any clinical signs and symptoms. So it could be that that patient in the cells has a very good situation. But the evidence shows that it's different, that those patients have -- if they don't complain, they do have abnormalities and there's a lot of evidence that I showed yesterday, a whole series of slides, one after the other. I know I went

a little bit quick but you have those references in your copy of slides.

Q. I'm going to move you on, because I don't know that we need to discuss subclinical hypothyroidism.

A. It doesn't really concern here, but it's just to show you, in order to reinforce his statement he gave wrong information. He gave the impression that subclinical hypothyroidism had no pathology, which is not the case, evidence is against that, much evidence, and that the treatment of subclinical hypothyroidism doesn't work, and it does. The majority of the studies show it does. And it's not a small majority, it's a big majority.

Q. Let's come on to the wellbeing of patients, paragraph 23.

A. So again a misinterpretation of a study by Dr Weetman, I don't know why he does that. Dr Weetman states a study that contradicts his position that shows people to be better with low TSH than high TSH within the reference the reference range, and he tries to show the alternative explanations, but the best explanation is the patients felt better.

In his explanation he implies that the serum TSH level indirectly implies that you wouldn't have such a lower TSH level where you feel better if you gave T3 and T4 treatment. So on the one hand he says T3 and T4

in those patients should probably be given, but he forgets one thing, if you add T3 to a preparation to T4 and you have an equivalent preparation, because you have to lower a little bit T4 and take a small dose of T3 and you get a sort of equivalent preparation as T4, you usually get a lower TSH. Studies show the TSH secretion is more suppressed by T3 than T4. So I added studies, but we had proposed not to give you too much of that data, but if you want you can get it, that shows those references, not in order, not to overload you.

Q. I understand. But from your paragraphs 24 through to 35 you are discussing the use of T3 together with T4 and also Armour Thyroid.

A. Yes.

Q. I think we've covered that fairly thoroughly yesterday.

A. Yes, it seemed to say that -- if I resume, that they are potentially harmful effects and they are potentially harmful effects to any thyroid preparation, and it's not more with dessicated thyroid than with the other ones. You may not overdose. You may not give thyroid hormones to somebody who doesn't need any.

If you give thyroid hormone to someone who doesn't need any, he gets overdosed and gets those unpleasant signs of anxiety, trembling fingers and sweating all over.

Q. I would like to come on to paragraph 34 on page 14 of your report.

A. So this is the fundamental point, the discussion on treating patients that have values within the reference range. I have shown you that there are a lot of references that show that there is in that reference range pathology, you must have optimal values. It's even more complicated, every person has a different optimal value, so you need to examine the patient clinically. I know it takes time, I take 15 or 20 minutes to examine a patient, but with that I have a lot of information, and that's only a physical examination. They have to fill in questionnaires before, so I know a lot about their complaints.

Q. You've taken us through the arguments that you raise in paragraph 35 in detail yesterday.

A. Yes. Showed that there is already at middle age, that starts at age 31, decrease in receptors and things. There's an increase in the blocker of T3, reverse T3 with age, and that makes that, even if the blood levels would look good, it may not be good in the cells. So the doctor has to examine the patient.

To resume a little bit this TSH question, I think you can compare; the T3 and T4 levels are like the food we take. We eat food to get energy.

The TSH is a sort of result, so you would examine for instance the stool to see if everything is absorbed. If you see food that is not absorbed, there probably is a problem that you have to solve. But in order to know how the person has energy, you have to examine the patient and interview the patient. We're doing something very specific, we cannot rely only on TSH. So I think this is very important.

Q. You make the point in the middle of paragraph 36:

"Physicians should not rely only on thyroid test for the diagnosis of thyroid failure but evaluate the presence or absence of hypothyroid complaints and physical signs too to reach a diagnosis."

A. All, but do of course also the test -- and this is a problem in the UK -- and do it on the right time without taking it before.

So the conclusion of Dr Weetman that he repeats inadequate symptoms in assessing whether patient is hypothyroid is made on his personal interpretation of a study that says exactly the contrary.

Q. This is paragraph 38, the study you talk about is the 1997 Swiss study from Basel. We saw it yesterday in one of your slides.

A. Yes, it says the conclusion of the study is exactly contrary, and what is very good in the study, it says

that we shouldn't only rely on clinical symptoms, we should also do testing, but next to testing don't forget the clinical symptoms for the diagnosis.

Q. So the point is, looking at paragraph 38.2 --

A. Well, in this study about 63 per cent of the patients who were outside the reference range, who were normally for classical diabetologists, I would say, with a TSH above the reference, 63 had severe hypothyroid complaints, but also 6 per cent within the reference range had severe hypothyroid complaints.

Dr Weetman said it means it's not clear-cut difference. No, it probably means that people within the reference range can also be severely hypothyroid because they have low receptors. The hormones do not go in the cells easily.

Q. Poor conversion is something you told us --

A. Especially if they are treated with thyroxine because thyroxine is just a precursor hormone. This is a bit what I said in ...

Q. That's what you say in paragraph 38 and 39.

A. And I confirm that in 40, and things like that.

I would normally make a big map where you have all the references but I think you're not here to be overloaded with information, but everything I say I can prove it here.

I have taken some copies out of major textbooks. We have here the thyroid. There are other major textbook. There's the Williams Textbook of Endocrinology, and very important, the Groat Textbook of Endocrinology, which is the biggest, three big volumes, and the Groat is very interesting. I'll talk about it later, because he had made an article called "Dangerous dogmas", and he showed that a low T3 -- so here the T3 wasn't really checked initially -- with normal other tests should be treated. He suggested that should be treated because now for the moment endocrinologists tend not to treat it, and he showed a fantastic article, I would say, because it had a lot of references and very clearly written, that there's a need at least for a trial of therapy in those patients.

Q. Paragraph 42, you repeat Professor Weetman's suggestion that he never heard of visual perceptions as a symptom of hypothyroidism or a sign of it.

A. Yes.

Q. You say in the next paragraph, paragraph 43, that you weren't aware of it.

A. I wasn't aware of it, and I found it at least a little bizarre indeed as a symptom, but by looking up the literature, I found and I showed you yesterday the articles that 71 per cent of patients with clear-cut

hypothyroidism have visual field effects. That means when they look, part of what they see is defective, it does not work well, so that was for me quite amazing, but when you think it's quite evident, because their's myxoedema in the head, so it is a sort of compression in the head of tissues, and also the nerves that go to the eyes can be compressed, that can give those visual field effects, and I can understand they clear up with treatment.

Q. On paragraph 44, again referring to Professor Weetman's report, it's the Stobhill study.

A. It's a double blind placebo controlled study, but with a very small sample, 25 patients and 19 controls of patients who were within the reference range for thyroid hormones but had complaints, and the complaints did not improve with thyroxine.

Q. We also know, because the Panel have considered this study, that only 100 micrograms of thyroxine was given to patients.

A. You see what happens with the 100 micrograms. I showed you the slide where at 120 micrograms of patients with overt hypothyroidism, half of the day they remained overt hypothyroid. So the dose may be not enough, but also thyroxine itself may be not the good medication. Patients do sometimes much better with T3 and T4

combinations and dessicated thyroid, that is considered by endocrinologists as outdated preparation, but that I use also regularly when patients do not respond on classical medication.

Q. You then comment on Professor Weetman's suggestion that the study was compatible with other studies showing no objective benefit from thyroxine in mild subclinical hypothyroidism.

A. Yes. So once again I repeat the information and I give all the references that there is efficacy by giving thyroid hormone in subclinical hypothyroidism. I don't know why he mentions this, because if he's really a thyroid expert, this is really fundamental knowledge to know that there are studies that do have effect and that there are more of those studies than the opposite, that it has no effect.

I didn't see really studies that treated subclinical hypothyroidism patients gives problems. So even if it's neutral, why not permit a therapy trial when patients have complaints or have some sort of problem that could be improved by thyroid hormones safely.

Q. You say towards the bottom of your paragraph number 45 that there are trials and most of those trials showed significant beneficial effects.

A. From 33 studies showing beneficial effects.

Q. And you say:

"It is, for example, an accepted practice to provide biochemically 'euthyroid' patients with thyroid nodules and goitre to reduce the nodules and goitre."

A. All endocrinologists, I think also Dr Weetman and Dr Lynn, normally have patients who have thyroid nodules and values within the reference range, give thyroid hormone, and for them it seems not to be a problem, and here suddenly it is a problem.

Q. You then go on to attack Professor Weetman's conclusion that the existing data shows that there's no significant benefit from thyroid hormone replacement in biochemically euthyroid patients?

A. I showed you yesterday where in depression you give to euthyroid patients T3 and it helps them get out of the depression. Where patients with a TSH above 2 but within the reference range and having thyroid antibodies, if they have a high cholesterol, it is lowered by a small dose of 50 micrograms of thyroxine. So it does mean there is some biochemical abnormality caused by a sort of thyroid deficiency, and that giving thyroid hormones improved the situation.

So there are many study actually, but not very many. I would say there are enough studies to go forward and permit in patients who are within the reference range

and have complaints to be treated, because there's no real study that shows it has adverse effects.

Q. Can I remind you --

A. There's only one study that they did that showed that some patients had heart palpitations, but some, a little group, it was not really significant but they mentioned it in the article.

Q. Can I remind you, Professor Weetman agreed that his view that there's no benefit from thyroid replacement in biochemically euthyroid patients that that view was based on that one study, and his view was the fact that there were small numbers of patients and controls in the study didn't matter.

A. Here we're talking about subclinical hypothyroidism. I found four studies with no benefit but against 33 with benefits.

Q. You deal again with his second conclusion that --

A. Yes, you're talking -- I'm sorry, I was talking again about subclinical. You were talking about biochemical euthyroid patients. It's true, it was only on one study.

Q. You deal with his second conclusion where he says:

"We have seen there are also good physiological reasons to believe that such treatment [that is of biochemically within the reference range patients] is

futile."

A. Yes, I think he adds this to accentuate his point but, again, I cited a whole lot of articles where you can see that it does help, it's worthwhile, et cetera. So I am very surprised that an endocrinologist of his level takes the risk to put all this sort of information in this way, that is actually erroneous. I don't understand.

It's not me to judge why he did. This, it seems to me unbelievable for a person who writes in the thyroid textbook to put information that could be so easily checked and the contrary can be shown.

Q. You deal with, in paragraph 46.2, a recent retrospective study, which I think hasn't been published?

A. It hasn't been published yet. It's a retrospective study so it has less important value. What is a retrospective study --

Q. How do you know of it if it has not been published?

A. I just received it from the person who did the study, and so those patients had received a thyroid treatment -- and actually you can have a copy, if you want, of the study -- and it showed that they had significant benefit, I think 70 per cent improvement by treating clinically hypothyroid patients, biochemically euthyroid patients. It is a practice that many more

doctors do.

Q. You then go on in paragraph --

A. It's patients who have been initiated in treatment, I think it was in November or December, for one or two months, consecutive patients.

Q. You then go on to talk about reference ranges and I think we've covered that in detail yesterday.

A. Yes. So again, Dr Weetman says these are normal results implying that they would be healthy results, but it's just statistical reference ranges. If you have a population that's completely hypothyroid, you will still have reference ranges with 2.5 per cent under, and 2.5 per cent above. If you have a complete normal population for a certain parameter, you will still have reference ranges. So you don't know very well, you know where other people have their values but you still need to have a clinical assessment of the patient to determine really if he's hypothyroid or not.

Q. Paragraph 50 on page 19, you talk about secondary hypothyroidism. I don't know that there are issues there that concern the four patients that we're dealing with.

A. Well, it does in the sense that secondary hypothyroidism means that there's a weakness of the pituitary gland. Dr Weetman says this is a very serious condition when

you have that, it's a drama, it's a tumour. Actually the most classical sign is progressive weakening of the pituitary gland with less and less able to secrete TSH, because it goes very slowly. So the older you are the more likely you are that your TSH of 2 would be in a young person 4 or 5, because there's ...

So many of the patients who age, aging starts maybe at 30 or 40 years, they get into a partial secondary hypothyroidism, and I showed you the data yesterday. One after the other at all levels. But that doesn't mean we have a 70 per cent decrease in secretion of TSH. On the average it's 50 per cent with age.

Q. Can I take you to your summary of Dr Weetman's report. You say that his:

"... belief that there would be no reliable scientific support for treating individuals whose thyroid function tests are within the reference range with thyroid hormones, is contradicted by over 80 studies."

A. Yes. So I have shown I think over and over the studies; you have the copies the references. Of course, it's very useful to have even more studies and more studies, but I think there's data enough to say now we have to pay attention to these patients, and when those patients say: doctor, I don't feel good; and they have those

values within the reference range but I would say still if it looks perfectly normal with a TSH under the 2 or the 1.5, you might have to be extra careful. But if they have a TSH above 2, above 2.5, I think you have to consider treatment, and a trial of treatment that has to be done safely in a very progressive way.

Q. You give your conclusions at paragraph 52 and the Panel can see those. I think we've heard them throughout your evidence.

You then set out a warning that's on the home page of the British Thyroid Association, which is in these terms:

"There are a number of websites with unhelpful and inaccurate information about thyroid disease which should be ignored. Our links [it says] are for recognised patient support and information science recommended by the BTA."

The pages may have got mixed up, and you may have to jump to page 20. Sorry about that.

You say:

"I believe that most of those websites are in support of treatment of all forms of thyroid deficiency, including patients who are clinically hypothyroid, but with thyroid tests still within the reference range."

You go on to say that:

"A check of those websites shows much more scientific evidence than the British Thyroid Association where scientific evidence is almost completely lacking."

A. I'm especially referring to the scientific references that they put on their statement on Armour Thyroid. There are about 300 references for their guidelines on thyroid diagnosis.

For me, for the website, the British Thyroid Association to say that, "We are the best and the only ones that should exist in a certain sense", is a little presumptuous, and I feel a little bit uncomfortable that a person would say, "I know the truth and I know the only truth".

Q. We then have to go to page 21, back a page, I think. Where you deal with Dr Lynn's report.

A. I want to repeat that my position is not to have it against a person. The impression I have from Dr Lynn is that he's a very honest person, and I was really impressed by his honesty.

I was not impressed by his report. I think the quality of his report is -- I just will, like a doctor would say, repeat what I know but I won't challenge this knowledge with what I have as information before me.

He says, for instance, in page 3.7, he's very clear: "The most diagnostic tool in diagnosis of

hypothyroidism is checking the blood level of thyroid hormones."

Q. That was his evidence, that --

A. Yes.

Q. -- blood results define hypothyroidism.

A. He says:

"The diagnosis of hypothyroidism is based exclusively on the measurement of thyroid hormone in the blood. These tests are highly accurate and are reliable."

I showed that there are some problems with these tests:

"Their use has not been questioned in any reputable medical journey."

Well, maybe it's time now to question and see if it gets published:

"It is considered that patients with TSH levels above the 5.0 milliunits per litre are likely to have thyroid distinguish contrast."

That I don't understand but I suppose it's just likely to have thyroid deficiency.

Q. Again, we have dealt with at some length --

A. Most the points dealt in Dr Lynn's report, happily I've answered them in Dr Weetman's report who states much more, I would say, inaccuracies, and it's much more

oriented.

So there's no evidence at the moment that the combination of T4 -- it should be T3 and T4, there's a little error here in my printing, it superior to giving T4 alone. Again, we showed that there is evidence. I didn't show all the evidence because some of that is not really problematic here.

Q. Can I take you on to page 22 of your report and paragraph 60. Dr Lynn was suggesting, certainly for one of the patients, that the problem was depression, not hypothyroidism.

A. Yes. Many patients often, when the doctor suggests that, have the impression that: doctor doesn't really listen to me. He puts me on (inaudible), I'm depressed but I feel something else on the problem.

That is one of the problems that there's more than a sort of fight between endocrinologists of the British Thyroid Association and patient groups than there is with doctors.

The patients say: all those websites say they don't listen to me, they don't understand me. I showed you that if you have a hypothyroid patient, you can probably help the depression, but you probably cannot cure the depression. You will be able to teach the patient through psychotherapy to live with this depression, to

accept it.

If you give the thyroid hormone to a patient who's hypothyroid and has depression because of hypothyroidism, you will take out the depression of the patient more easily than with psychotherapy. If you stop the treatment, she will get depressed again.

Typical for depression caused by hormone deficiency is that it is chronic. It's all the time there. With thyroid deficiency it's more in the morning than the rest of the day but it's there, every day it's there, because the thyroid deficiency is not cured.

The problem of this approach to quickly put a label, it is psychic, it is nothing to do with an endocrine problem, it's just purely in the head, is that you have then to cure the head with psychotherapy and antidepressants, and as I said, also the other symptoms. For the constipation you get laxatives. You give for the pain they may have anti-inflammatory medicine, or pain reducing medication. It's more expensive and less efficient because you get also the side effects of all those other drugs, which you shouldn't have with thyroid hormones.

What is the risk of thyroid hormone? The classical risk is to give too much. So we should be aware of that. I always give the information to the

patient: what are the symptoms of excess? So they can also participate in the control of their treatment. They should always know the symptoms of overdoses. I give a sheet for that.

Q. You have heard that Dr Skinner has a sheet from June 2004, which is available for patients --

A. And it has all that information.

Q. -- and prior to that he would give that information verbally.

A. That's perfect. That's how it should be.

Q. I'm going to move on from your commentary on Dr Lynn's report, he'll understand no disrespect is meant if we don't spend a great deal of time on his report because you've dealt with the criticisms globally of Dr Skinner.

Can I take you on, please, to page 23, where you come to the third section of your report, Analysis of the Patient Files. You deal with a number of issues on page 23 that we've already discussed.

Can I take you to paragraph 68. You say that an analysis of the files or the patient records shows that the patients with higher serum T4 didn't show any psychic or physical signs of overdose.

You say:

"To be sure that there is an overdose of thyroid

hormones at least some minor signs or expects should be found and reported, which is not the case with any of the patients here."

A. No. I don't exclude 100 per cent that there was hypothyroidism, but with the data I have here I cannot confirm any of those that they would have hypothyroidism. It's most likely due to a wrong timing of the blood vessels, the wrong condition.

Q. You say there's no mention of the time when blood was taken in those patients.

A. No.

Q. You've dealt with your charts, with the concept that the serum level of thyroid hormone thyroxine is increased during the nine hours or so after a patient has taken the medication.

A. Yes, and the higher the dose, of course, the more it will increase.

Q. You then go on to deal with interpretation of biochemical testing during therapy. I think we have dealt with those issues in what you told us yesterday.

Then you go on to deal with lower levels of TSH and again, these are issues that you've discussed before. You deal with, in paragraph 71, aging and other conditions which tend to lower the TSH in testing.

A. A point that is very important to repeat is that every

individual has actually a different individual range.

Q. This is a point you make at 72b on page 25.

A. I've given also references that show that. So if you want you can have it.

It's very important in how do we know the ideal reference range by a patient, is again by examining him clinically, it's the end effect of the hormone. What we see and what we hear from the patient is complaints or absence complaints, give the end effect of the treatment. We cannot do a good endocrine therapy without clinical examination, and I would also suggest always to do blood tests, it always is helpful.

Q. Let's come on to the patients again. We dealt with the first part of your comments on Patient A. It's page 26 of your report, and we looked at everything on that page yesterday.

Your view was that a therapeutic trial of thyroid replacement therapy was justified with this patient.

A. Yes.

Q. Because of the medical history and the findings on clinical examination, and you had the view as well that it was beneficial for the patient.

A. Well, the patient says herself, "I feel much better", in her witness statement before the GMC.

Q. We have it in the GP records as well, and the Panel will

recall -- perhaps not need to be given the page references, from tab 1 of bundle 1 -- where Patient A was reporting improvements to her general practitioner, and you refer as well to improvements in her constipation.

A. What I have done is I have grouped all the slides together so we can go in the order that this report is done to the information.

Q. That's fine but the Panel will want to follow this on the page at the same time. We can certainly see the slide at the same time.

A. Okay.

Q. We've looked at that.

A. I will present it with showing on the slides, if it's okay for you.

We saw that Patient A had on all levels a suspicion of hypothyroidism, and then she got a treatment and she responded well. What is the treatment she received?

Q. She received a prescription from Dr Skinner for 25 micrograms a day of thyroxine, increasing after a week to 50 micrograms a day, and the evidence suggests that once she was on the higher dose, 50 micrograms a day, she had headaches and felt strange, if I can put it that way. She was feeling aggressive and violent, I think was the word she used.

She told us that Dr Skinner prescribed Armour Thyroid, and when she took that, her headaches stopped immediately. You deal in your paragraph 77 with headaches.

A. So Dr Lynn had said that for him it was probably not due to the thyroxine because it was such a small dose.

Q. Yes. That was his view, what's yours?

A. That is a possibility, certainly. Sometimes they don't tolerate because they are adrenal hormones -- the cortisol is a little low, but there was no information so I cannot really comment in this case.

Q. You said you agree with Dr Lynn that it's unlikely that the headaches were due to dose.

A. With the information I have it's unlikely. But anyway, because also a well-balanced thyroid treatment takes out the headaches in the patients who are hypothyroid with headaches. In 8 to 9 cases in my experience or 10.

Q. Could I just correct one thing that is in your report and may be on your slide, that this patient was started on half a grain of Armour Thyroid a day for a week --

A. Yes.

Q. -- going up to one grain and then 1.5-grains. The reference is bundle 1, tab 2, page 17. But I don't know that people need look at it now.

A. Well, I didn't have place enough to write everything.

Q. Don't worry.
A. I wanted to remain schematic.
Q. So the patient was treated. What's your view of the prescribing by Dr Skinner for this patient?
A. Well, if thyroxine does not work, prescribing Armour Thyroid is actually my first option. I don't go to a T3 and T4 combination, I go directly to Armour Thyroid if they do not tolerate thyroxine, because it's more likely to work in my experience.

Looking over, it seemed, following Dr Skinner, she seemed less hypo and in the report of Professor Franklyn she seemed to be euthyroid. So it didn't make her hyperthyroid apparently, so I would say it's a safe treatment.

It may not work sufficiently. If she is less hypo, she should be euthyroid. So I cannot comment on that because we have two different evaluations here from Dr Skinner and Professor Franklyn.

When she was on 1.5 grains, which is I don't know by heart exactly the dose that makes the T4 and T3, but which is a classical dose given in hypothyroidism, you see that the levels of TSH is 0.4, which is not totally suppressed but it has decreased, and the free T4 is 12.6, so it's borderline low.

What I know here is that I'm almost sure she didn't

take the thyroid hormones before the blood tests. This is one of the blood tests that was done correctly. That's almost sure.

I would say that it could be that Dr Skinner is correct because it's still quite a low T4, we saw being in this range has risk factors: increased insulin resistance, so you get easier fat, et cetera. And the diagnosis of Professor Franklyn I don't understand, she says she's euthyroid so stop the medication, and she actually in blood borderline low in T4, so if you stop the medication it will be even lower.

So I do think that there's in the UK -- it's not only in the UK but it's more in the UK than others, a sort of phobia, you have to treat patients within the reference range because there's so much aggressiveness from endocrinologists who do not agree, and probably that has pushed Professor Franklyn to give this advice that it probably should be stopped but that the patient could continue on the treatment.

Here normally the patient should have remained on the treatment.

Q. We know that her treatment was increased, of thyroxine, to 75 micrograms a day and then to 100 micrograms a day, and that was the treatment that the patient was on when seen by Professor Franklyn, I think.

A. She was on thyroxine and not on Armour Thyroid.
Q. It's not entirely clear. We have prescriptions from the general practitioner in the period running up to Professor Franklyn seeing the patient which suggests --
A. I thought it was switched over to thyroxine after seeing Professor Franklyn, and this blood test was before seeing Professor Franklyn.
Q. Professor Franklyn certainly says she thinks the patient should be taking thyroxine rather than Armour Thyroid.
A. Yes, but there are two blood tests, and I thought the second blood test that we have here, I don't know if it's correct, maybe Dr Skinner can correct here, but the second test showed a little higher T4 on the thyroxine, and the free T4 here with Armour Thyroid, what I thought was Armour Thyroid was a little low, which is what we would expect.

With Armour Thyroid we wouldn't expect the same level of T4 than with thyroxine alone. But here it seems that the patient didn't take the thyroid hormones before the blood test, and then you see a normal blood test.

So here there's, I would say here, 100 per cent for me at least, and I do think that I'm the only expert who has talked here actually in this sort of topic in this sort of section of endocrinology. I see absolutely no

reason to stop the treatment, and just to continue the treatment. It can be discussed if the patient has to go on thyroxine or Armour Thyroid, but she needs thyroid treatment because with thyroid treatment she's not overdosed, clearly.

Q. Right. Can I bring you to the criticisms that are made and which the Panel have to consider of Dr Skinner. It is said that he took an inadequate history and undertook an inadequate examination of the patient.

A. Well, we go back to the history. That's quite a lot of history that I have found. I can repeat the history, but I don't think it's necessary.

You see there's a lot of information. I have been a long time a medical board member, and I've seen files from GPs and there was just one or two words written on. This is much, much better. It's far better than that. So I wouldn't think there's an inadequate report. I think that the one who has written that probably has not really read the files, truthfully.

Q. It's said that the examination of the patient was inadequate as well. You --

A. There's a lot of symptoms here and there are often other things that are not written, cracked heels and yellow tint, it's very clearly symptoms that are compatible with hypothyroidism. So I wouldn't say that it's

inadequate. I see absolutely no sign.

Q. Let's come on to what may be the major criticism that in prescribing at all for Mrs A Dr Skinner was acting in a way that was inappropriate, unnecessary and irresponsible.

A. You see here that if she didn't do that, if she didn't receive treatment, there are increases of risk of memory loss, metabolic syndrome means being fatter, having a high sugar level, higher cholesterol, higher triglycerides, high lipids, abnormal cardiovascular risk markers in certain subcategories of patients heart, and coronary heart disease, I think.

So I don't share that view. Also possibly an increase in breast cancer risk, even mortality, so --

Q. Do you say it was appropriate or inappropriate for --

A. I think this was appropriate. I certainly would have given, and certainly in face of the important complaints of the patient that extreme fatigue, the constipation and the fact that by giving the treatment, the patient felt much better.

Q. Do you have any other explanation for why she improved?

A. I don't see how you can improve chronic depression in a person who is hypothyroid. She has so many hypothyroid symptoms and she probably improved on -- also the bowel movements were better, so constipation

since here teens were improved, it's almost pathognomonic, it's typical for low thyroid function, and that improves with treatment.

Q. Professor Weetman's view as to why she may have improved was that she found Dr Skinner to be a nice doctor. A sympathetic doctor, and Dr Lynn's view was the same.

A. He must have some magic powers then, to cure an extreme fatigue. We have probably a new guru then. But I think it's just -- we should stay matter-of-fact. She received thyroid hormones, she did not see any more -- I think she even complained, wasn't it so.

Q. She was the woman who said she didn't really like Dr Skinner or his manner.

A. She wasn't happy, so how could she be influenced by a doctor she doesn't like? She would rather be more fatigued and more depressed. I don't think it's proper information.

Q. She said she was complaining to the General Medical Council about him.

A. Yes.

Q. Let's turn to Patient B, if we may, please. Page 29 of your report, and you've drawn up a slide for us?

A. Again, we see here a patient with a medical history that is possible due to hypothyroid depression, but depression can also be found in other endocrine

deficiencies. But she's also menopausal. This, I would say, this patient is important because we'll see later.

She has symptoms that can be caused by low thyroid function, or at least by an endocrine deficiency, so how can you know if a symptom is caused by hormone deficiency or by another cause?

Well, usually if you have stress you get depressed for a short time, but if the cause is endocrine deficiency you remain depressed all the time. Maybe not all the time of the day, but every day you will have the complaint, and that is very typical for an endocrine deficiency, chronic symptom.

So she seemed to have a lot of those, no enthusiasm, lack of energy, falling asleep all the time is typical. Thyroid hormones are wake-up hormones. If you have too much thyroid hormones, you're too much awake, you don't sleep well. So when it's not enough, it's the contrary. Memory lapses.

Thyroid hormones increase connections between brain cells. So the memory's not good when you don't have enough because there's not enough connections. Those are the hormones of intelligence.

If a child has very low levels, he won't be able to study, or do good studies, and it's the same for elderly persons, they get memory lapses.

Breathlessness can also be the heart doesn't always pump very good in hypothyroidism.

Joint pains, but it's a diffuse joint pain. That I don't know from the information.

Fibromyalgia, in my experience those are pains in muscles and in the tendons that people have. There is an incidence of hypothyroidism and it often is partially relieved at least when you give that to hypothyroid patients, the thyroid hormones.

Hoarse voice, that is quite typical.

Puffiness around the eyes, face, hands, and feet is rather quite typical of low thyroid function.

And under the slow heart, that's typical, when you have too much thyroid hormones you're above 80 per minute. Here it's 56. And dry skin --

Q. 58 --

A. Yes. So there's a lot of very typical symptoms of low thyroid function, especially if you group them together, you're almost sure that those symptoms will improve with thyroid hormone even if you don't have the blood test result yet.

Overweight, brittle nails, that is typical for low thyroid. It improves with thyroid hormones in swollen ankles, et cetera.

So there's a lot of good physical examination,

complaints were abundant, and they are suggestive of hypothyroidism. So we have a medical history where it's possible hypothyroidism, but it's much more clear at physical examination and the complaints, and we have a TSH at 2.3.

You have seen that there are several studies that have shown increased pathology, for instance you have easier autoimmune antibodies, the people are easier obese or arterial stiffness, the familial hypertension, there's more hypertension in families. In family people have higher 2.3 so they have a predisposition to hypertension, these patients.

Increased cholesterol, it's more likely in such a patient.

Q. We see that.

A. You see that.

Q. Take us to the T4 level. Again, you don't need to read out what is in your right-hand box.

A. Again, it's a T4 that is apparently in the lower quartile, in the lower 25 per cent of the reference range. This is also linked with a whole list of diseases we have seen, or disease factors.

Of course, that doesn't mean that this patient has all these increased risks, but some of those probably she does. If she doesn't get the treatment. If she

gets the treatment she will probably be relatively protected against those.

Q. We've heard that Dr Skinner prescribed thyroxine at an increasing rate as he might do for such patients, first at 25 micrograms, then 50 --

THE CHAIRMAN: Mr Jenkins, I'm sorry, could I just interrupt. On the screen the T4 is 12.2 and in our notes it's 16.7.

MR JENKINS: I'm grateful, thank you.

A. Patient B, you're sure?

MR JENKINS: It is Patient B. Let me check the result, forgive me.

A. That's probably an error. I thought it was another patient.

THE CHAIRMAN: So it should be 16.7?

A. Yes, 16.7.

(Pause).

MR JENKINS: There was a date on 21st August 02, and the Panel have it at bundle 1, tab 4, page 2.

A. I think it's from another patient.

Q. If you go come to your report you've got it correctly, I think.

A. It should be correct. The 16.7 is good, that's a good value, and as I have pointed it here, I didn't put any remarks, it's only the TSH above 2.1 actually, so not

this result that I put there that is correct. The 2.3 is correct.

Q. But you say in your report on page 29 that a therapeutic trial of thyroxine was justified because of the history.

A. Yes.

Q. And the clinical signs with this patient.

A. Yes. It must be also some error here or an omission, that I'm talking of familial history of Hashimoto.

Q. I think that's another patient, Patient D.

A. I think there are some errors there so you should take that away. But there is depression, there's clearly clinical hypothyroid, take out this Hashimoto, and there's non-optimal thyroid test, but it's only one test that is not optimal. Page 29.

Q. The family history relates to Patient D, I think, and it may be --

A. Yes. I'm going to take it away here.

THE CHAIRMAN: I'm sorry, could I just question here? I'm on page 29.

MR JENKINS: That is right.

THE CHAIRMAN: I don't see Hashimotos on this page.

MR JENKINS: Under "Therapeutic trial, justified".

A. That's an error.

THE CHAIRMAN: Thank you.

MR JENKINS: We're putting a line through "Familial history

of Hashimoto's and dysthyroidism, unoptimal thyroid test."

THE CHAIRMAN: Are we saying that there could be a confusion --

A. With Patient D.

THE CHAIRMAN: Or the history.

A. Yes. For the family history.

MR JENKINS: It relates to Patient D and it has clearly crept in.

A. It is actually the technical detail. I used probably slide D to make this one and I omitted to take that away.

MR JENKINS: You say that the trial is justified because of the history --

A. Yes.

Q. -- of depression. And the --

A. Clinical hypothyroid, and one test was not optimal. One thyroid test was not. So it's always a combination. One factor is for me not enough. You do have to have a certain confirmation by medical history, by thyroid test, and there was notable improvement, so less depression, following Dr Skinner and the GP, Dr Blair.

Q. I don't think that Dr Blair reported much by way of improvement. But Dr Skinner certainly did in his notes.

A. He did in one of his letters, in the other it wasn't any

more. In one of his letters he did, it was the letter of 27/2/2004. I don't know exactly which page it is.

MR KARK: 4/19.

A. But as Dr Blair suspected that the treatment would harm his patient, he did everything to take his patient off, if I understood.

MR JENKINS: It's page 20 where Dr Blair says:

"When she consulted me on 15th May she had started on the thyroxine tablets, having stopped her antidepressants and at that stage she was feeling better."

A. Yes.

Q. Yes, thank you.

A. I can only rely on the reports.

Q. Of course.

A. This was under treatment, so I didn't put that Dr Skinner gradually increased the dose, I think it's a very good technique to gradually increase the dose. Why do we have to with thyroid hormones to increase gradually the dose? This is very simple.

When you have a low thyroid function, your blood volume is reduced, so there's less blood, so you give thyroid hormone, it's concentrated, and there's also something very particular to low thyroid function, is that patients have a lot of adrenalin in their blood,

a lot more, sort of compensation, but it doesn't work well. Because there are much less receptors, so the molecules for it to work are not there, not enough there.

When you give thyroid hormones quite quickly there's much more receptors, and then with this excess of adrenalin there can be palpitations and bad reactions. That's why we have to start always on low doses.

The methodology used by Dr Skinner was in my option perfect, I do it a little different, but it's if I understood 25 micrograms of thyroxine during one week, the smallest dose, then 50 micrograms during three weeks, and then 75 micrograms, 3 to 6 weeks, et cetera, so it's quite slow, and that's the best to avoid harm.

You may never give very suddenly the dose. It will create bad reactions.

So I didn't put that, but it's here, the fact that he climbed up to 75 micrograms per day of thyroxine and then later switched over to T4 and T3 preparation, so Tertroxin was added, 20 to 40 micrograms per day T3. It wasn't clear to me if it was given once a day or twice or three times. I thought it was given once a day from what I had in the reports. So the patient was hypothyroid, still on the treatment, following Dr Skinner, but the GP talked about an episode of

thudding heart, of a sort of heart palpitations, and we've discussed that.

Now, what sort of heart problems do you get when you get a too high dose of thyroid hormones or you don't tolerate it? It's a 24-hour on 24-hour problem. You get the whole day and the whole night your heart goes too quickly, and you feel it even more in the evening, because in the evening you rest down in your bed and then you feel your heart beating stronger.

Q. So you're saying it's not an episode if the patient is overtreated?

A. It should not be an episode, no.

Q. It should continue?

A. It could have been because I see no information here. It could be alcohol but it also could be caused by caffeine. If my patients take caffeine, two cups a coffee with the thyroid treatment, they will not tolerate it, because it's like a supplementary dose of adrenalin, and they have already, once they're well treated, so many receptors.

So the recommendation, and that is generally given in endocrinology, is to avoid caffeinated beverages during treatment of thyroid deficiency. One cup of coffee maybe, but two is generally too much. There could also be another cup but there's no information on

that.

Q. What we know of this patient, and again the Panel will recall the reference at tab 3, page 21, is that ten years or so before in 1992 Patient B had had over a period of a month episodes of flutters in the chest off and on for about half an hour and a raised pulse.

She was asked to do a diary of those palpitations by her GP, and that indicated that she was having episodes quite frequently each day. Might that be relevant if she's having palpitations --

A. So she probably has another cause --

Q. -- without being on thyroxine at that point?

A. When I receive, because I don't always have everything in memory, but if she has heart beating at 52 per minute, you can almost 100 per cent exclude any hypothyroidism, so that's further confirmation.

Q. I'm afraid when we come on to the lab tests you've got two figures in the wrong box, and the Panel will want to look at tab 3 of bundle 1, page 118.

Just looking at the lab test results, the Panel I hope have tab 3, page 118. For the first lab test of two, 5th December 2003, the TSH result was below 0.1 and for the next lab test, 1st September 2004, the TSH was 0.2, so they're transposed.

A. And also the T4 or not?

Q. No, the T4's right. I'm going to ask you to look at that page, page 118 on tab 3, just to check.
A. I have here three blood tests.
Q. All right. So what you've said of the result on 5th December 2003 -- and perhaps we should look at the page in the GP records to see where that test was taken, it's page 28. Do you have page 28 of that same tab?
A. Okay, I have the two pages.
Q. The note on 4th December 2003 --
A. Apparently thyroxine 225 micrograms, and pulse is at 76, and blood pressure 150 on 60, 70.
Q. It's not clear whether it's 150, 60 or 80 over 70. It's not to easy to read Dr Blair's writing, but he said "TFTs [tick]", which implies blood was taken on that day.
A. Yes, so you cannot really comment and I don't really have real clinical symptoms of excess thyroid hormones. If was 100/80, 70, you could have that because of an excess of thyroid hormones, because there's a big differential, but 150/70, certainly not.
Q. But there's no indication that the patient was told before that day not to take her medication before the blood test was taken?
A. No, and if you really have -- this is in December, so -- in September or December?

Q. This one is December 03. The next one is taken nine months later in September 04.

A. If you really have a T4 at 39, which is very high, and a TSH 0.1, you should have a quicker pulse rate than 76 per minute. It should probably be around 90, 100, or 110. So it's likely the patient took the thyroid hormones before and that this test is due to too much thyroid hormones, it's very likely.

Q. Well, what you've said is in the right-hand box, you have said:

"Most likely cause, blood test taken hours after the intake of thyroid."

A. Yes: other possible causes that she may have some central hypothyroidism which is not really confirmed because if you have a TSH of 2.3, I wouldn't call it central hypothyroidism.

Low T3 and T4 receptors -- I don't really think we have to talk about the rest. It's for me so clearly that almost to 99 per cent only possible cause is taking the hormone before there is apparently no physician here who has raised that point. I do not understand. What I would have done is taken a second blood test to check and specifically asking not to take the thyroid hormones before. Then you get your confirmation.

Q. Let's go on to the next test in September 2004.

A. What is good is we have some information on the most frequent antithyroid antibodies (inaudible) are not there not detectable. Here we have borderline high free T4, which will be good to have a normal T3, but the TSH is slightly suppressed.

(Interruption due to technical problem)

THE CHAIRMAN: We will take our break now.

(11.05 am)

(A short break)

(11.25 am)

THE CHAIRMAN: Mr Jenkins.

MR JENKINS: We were looking at Patient B on page 30 of your report and we've just corrected two figures that have been transposed, the TSH figures for 5th December 2003 and that for 1st September 2004.

A. On my report there's a little correction to put, that I put a footnote on T4, the thyroid lab tests of 11th November 2004, 13.5. I think it was without treatment at that moment.

Q. It certainly was by November, yes.

A. So you have to take out the little footnote.

Q. You had an asterisk, and you said above reference range for that, but it's not.

A. No.

Q. If we look, and perhaps the best place to look at it is

page 118 of tab 3.

A. So that's a TSH that is slightly too high to be without risk factors.

Q. When she is being treated in December 2003 --

A. Yes.

Q. -- and there's no indication that she's told not to take her medication in December.

A. No.

Q. The T4 is 39, the TSH is less than 0.1.

A. Yes, that's most likely the --

Q. Nine months later, at about the time the treatment stops, from Dr Skinner, her T4 -- and again we don't know --

A. She's probably on the treatment at that point, she wouldn't have this level of TSH and free T4.

Q. But by November 04, when she's certainly off the treatment, her T4 level is down to 13.5.

A. Yes.

Q. And her TSH is back to 2.4.

A. Both values are not optimal. We have seen that based on the studies I have shown before.

Q. When you're saying both values are not optimal, do you mean the November 2004 values?

A. Yes, is not optimal. TSH is, in my experience, a little too high, and the free T4 is discussable, could you

accepted or is slightly too low. When we look before ...

Q. The early readings were --

A. Here you have to have 16.9, I believe.

Q. It's 16.7 or 16.8. The two pages the Panel will want to remind themselves of are tab 4, pages 2 and 3. One is a reading in August 2002 where the T4 level is 16.7, and the other is a reading in January 2003 where the reading is 16.8.

A. What I wanted to point out is the following: we are back to before. So it was suggested that giving thyroid hormones during a certain time might be detrimental. This is what the classical have, we come back to the previous values, and even if you give that to a patient who has received thyroid hormones during 20 years, usually tests show that it's back to initial values as before.

I have another study that I didn't include here, a very old study where you have patients since 30 or 33 years of treatment, they came back to initial levels, which is not normal because normally you have a certain aging of the axis, you should have lower values after 20/30, years and apparently in those studies the levels were back to normal. But what I wanted to point to you it is not only that the --

Q. Slow down a little, if you would, please.
A. What I wanted to point to you is not only that the risk factors cause problems here because of the blood test, but that if she stopped, she probably had again her symptoms back.
Q. Well, Dr Blair hasn't recorded --
A. And he had recorded in the beginning that there was a certain improvement, and so she normally will have back her symptoms. It becomes slowly and progressively back. It takes several weeks, several months, but they normally come back if they were relieved by the thyroid therapy, of course, because it's not as well noted here.

So what I've put in here also is Patient B, information on possible harm. So I think here the possible harm is not of doing a treatment trial, but the absence of thyroid therapy long-term. This is really what I experience in patients, that you do have to watch out. We are there to relieve the symptoms.

I think both Dr Lynn and Weetman agreed that there wouldn't be any problems later on because of this therapy trial, of what I understood of their reports.

Q. Can I take you back to page 29 of tab 3. It covers the period --
THE CHAIRMAN: This is tab 3/29?
MR JENKINS: Tab 3, page 29, yes.

Blood is taken at the top of the page on 1st September 2004, and the blood pressure recorded of Patient B is 150 over 80.

A. And later on I see also in November, it's 170 on 90.

Q. Well, I'm going to take to you earlier blood recordings as well, but if you go back one page, we've looked at the date when blood was taken in December 2003, pulse is recorded as 76, and the blood pressure, it's not clear quite what's written, whether it's 150 or 160 or even 180 over 70.

A. So it's a similar, I would say, blood pressure, so I do not think we have here a condition of excess thyroid hormones in December 2003.

Q. Can I take you to page 26, please. The same patient. This is years before Dr Skinner prescribes any thyroid replacement therapy for this patient. We're dealing with recordings in 1998.

There are blood pressure readings for this patient on 26th May 1998 of 160 over 86, lower down 170 over 80, and below that 160 over 85.

A. So it's equally the same, yes. Higher blood pressure, because she has high blood pressure, is quite typical of low thyroid function. When you have a low thyroid function the arteries, the walls of the blood vessels are thicker, they're full of myxoedema and it's very

hard, they don't extend easily, so they're less supple.

And typically in several studies that I've consulted, you see the blood pressure going down. And it's quite uncommon, I would say, that a patient has to continue anti-hypertensive medication if they get a thyroid hormones because they have a thyroid deficiency. So after six months to a year often we do have better results, the blood pressure goes down.

What is quite typical for low thyroid is a higher diastolic, it's the lower blood pressure's a little higher, but here it's 90 actually, so it's a little on the high side.

Q. So just looking generally at Patient B, the allegations against Dr Skinner that at his initial consultation he took an inadequate history and undertook an inadequate examination of the patient.

A. If you compare his examination with what I see from Dr Blair, I believe, there are almost no notes, so I think it's much more extensive and I see no problem there. But the notes here of the general practitioner are typical of what you see in many general practitioners.

Q. Well, Dr Lynn said of the history that Dr Skinner took and of the examination, Dr Lynn's comments were that they were perfectly adequate. Do you agree with him or

not?
A. I would agree with that, yes.
Q. And of the prescribing to Patient B at all stages, both the initial prescribing and subsequent prescribing when the levels were increased, what do you say of the allegations against Dr Skinner that his prescribing was inappropriate, unnecessary, irresponsible and risked putting the patient at harm, it wasn't in her interest?
A. I think it's the opposite. I can absolutely not confirm any risk here, and again the explanation is that the thyroid tests were not done in the right conditions.
Q. You say that of the readings in December 2003?
A. Yes, and you see also there's no real harm because lab tests are the same as initial, after two months of treatment, stopping of the treatment.
Q. Well, again --
A. It takes three weeks to put everything back and normally what I do with patients, I rather do tests six weeks later when treatment is stopped.
Q. If this patient had been made thyrotoxic by the medication that was prescribed for her, what would one expect to have seen?
A. At the moment of being thyrotoxic you do have to have hypothyroid symptoms and complaints, clearly when you know very well the clinical case it's so easy to see,

and with blood tests here you cannot know. You should do again the blood test. I would have advised actually to do again the blood test when this has happened.

Q. What about physical signs and symptoms of the patient if she had been rendered --

A. It's very well-known in medicine how to recognise. The ask the patient to put his hand out and they tremble a little bit. They have sweating, the skin is often like that of a baby, it's getting too soft but also wet. You have too dry skin when there's not enough. You have too soft skin when it's too much. They have in the eyes, just when they look at the eyes, they look very anxious, they have a lot of adrenalin and it gives what we can call an adrenalin look.

They tend to be quite nervous, you see that the blood pressure often is a big difference, the lower blood pressure goes down and upper blood pressure tends to go up. They can have for instance 14/4 of blood pressure, or 140/40, and not the classical 13/7 or 13/8, and 12/7, 120/70 et cetera. So they tend to get a greater difference between the two blood pressures, and their heart beats quickly but regularly, in a regular pattern.

Sometimes I doubt, but when you examine the patient it's so easy. You really can examine. Sometimes when

I'm not sure about the doses a little too much or normal, after examination of the patient I don't have any hesitation.

You hear the heart bounce also much stronger, too strong, and of course, it could be emotionally, it could be because they took coffee before, so you have to control that, but there's enough information to be sure if the dose is even slightly overdosed by clinical examination, in my experience.

Q. Let's turn to the next patient, please, Patient C. The Panel will want to turn to page 32 in your report.

A. Again, I have tried to combine everything because I -- it's too difficult to go from one page to the other of the report, so I prefer to group together what I have found as evidence. You see the diagnosis of hypothyroidism again should be based on the different aspects, the medical history, history of being unwell, that is any endocrine deficiency, since menarche with irregular periods and polycystic ovaries. When the thyroid function doesn't work enough, the ovaries cannot work enough either.

So it could be either a lack of sex hormones, the ovaries don't work well, or that lack could be caused by lack of thyroid hormones, which are hormones that stimulate all the endocrine glands.

Then typical when they take out the tonsils in childhood, it's a child who has had a lot of infections. That is suspicious of a thyroid deficiency.

Secondary amenorrhea, so she had her first menstruations probably quite early, and then after she seemed to have -- they have stopped because maybe she developed more hypothyroidism at that moment. So this is compatible with a history of a certain degree of hypothyroidism. Clinical examination, poor energy is very general as a symptom. Ongoing fatigue that can be caused by low thyroid function. Poor memory also, that is more typical, I would say, to low thyroid function. Difficulty in concentration.

And then what is quite typical of low thyroid function is the dry skin. That is as hormone deficiency number 1 deficiency. In my experience, what you have to watch for is thyroid deficiency.

Then the bradycardia, 60 per minute is normally too low as opposed to a person who does not do extensive sports.

Dry skin and hair is also pointing to the direction of hypothyroidism. So I would say that physical examination is more -- I wouldn't say mildly hypothyroid, I would say hypothyroid, and when you look at the lab test, again we have a TSH above the 2, so

several studies show increased pathology, I am not going to repeat it all the time.

THE CHAIRMAN: Mr Jenkins no date, am I right?

A. There's probably a date but I haven't put it on.

MR JENKINS: Can I give you the page reference, it's bundle 1, tab 6, page 3. That's the date of the lab test, which is 10th March 2004.

THE CHAIRMAN: Thank you.

MR JENKINS: If you were to go above to the medical history and clinical examination, the page reference should be 1, tab 6, page 1.

The date of the consultation of Dr Skinner is 6th March 2004.

You've analysed the TSH above 2.1 before and you've analysed having a T4 in the lower quartile, 25 per cent.

A. So it's quite close to that so I repeated the information a bit to 2.1, so you do have studies that show that there's a risk of increase.

Now, I'm talking about all those studies because that's what I found. But there's a lot of -- there can be a lot of complaints also that I don't talk about because I didn't find the studies. But you have to think that that patient already suffers, she does have complaints of low energy and probably much more.

I think here the uptake(?) was not as good as before

but still satisfactory for me. But what the patients most want is not that you decrease their risk factors but that they get as quick as possible relief of their problems, their symptoms, so you have to think that there are also problems related to a lower T4 and a little higher TSH.

So because of the clinical symptoms, the medical history, and the blood test, I think it's justified to give here a medical history, to give here a therapeutic trial. Was it beneficial or not, it's not as clear here in this patient. Following Dr Skinner it was for some improvements, less weakness, better concentration, the tongue decreased in volume, it was less swollen, and there was still some low pulse, 64 per minute. It could be acceptable but it's better to have 68 or 72 per minute, and energy slightly better. So it gave a certain effect.

Following the GP it wasn't so, the patient felt not better on thyroxine and feels a lot better off medication. But he did not specify what is better, so it's not so clear for me.

For Dr Rodin, endocrinologist, he felt no better and no worse on or off treatment, it did not work but it wasn't ... So there are three different opinions here.

Q. The other issue with this patient, Patient C, is that in

August 2004 when she changed from one practice to another she said she was taking three preparations and, again, we have seen it in bundle 2, tab 2, page 2.

When asked to include the name and dose of all regular medication, Patient C said she was on thyroxine, Armour Thyroid, and Tertroxin, the T3 preparation. The second of those, Armour Thyroid, was not prescribed by Dr Skinner. That's the evidence that we have heard and we've seen no documentation to suggest that he did.

If she was taking that in August 2004, as she said she was, in addition to thyroxine and Tertroxin, might one expect the laboratory tests to reflect Armour Thyroid and T4 and T3?

A. This is compatible, but again I think it's mostly that the medication was taken before the blood test. That's the most likely cause, but it could also be overmedication by self-medication by the patient. It's difficult for me to say. It could be both pictures.

Q. We have not heard from the patient, all we know of the patient is what's said in that document, the one she completed. But coming back to the issue of whether she should have been placed on a therapeutic trial or not and Dr Skinner's prescribing for her, what do you say? We see your slide says that it was justified to put her on a trial.

A. Again I don't justify it on one test or one group of symptoms but on the fact that the whole medical history was clinically hypothyroid or suggestive at least of being hypothyroid and TSH above 2.1, and a free T4 in the lower quartile. I think we can talk of a mild thyroid failure that needed treatment.

Q. Can we just deal with some references for the Panel perhaps to make a note of in your report. There is an entry at bundle 1, tab 5, page 1 for 5th October 2004. This is the GP's records, saying, "Feels no better on thyroxine".

There's an entry on page 2, an entry for 28th October 04 in the GP records, "Feels a lot better off medication".

At page 13 of that bundle there is a letter from Dr Allen to Dr Rodin in which, as you point out, the patient herself felt no better or worse on or off treatment, is what is said.

Dr Skinner though had noted improvements in her signs and symptoms when she was on the medication. But as to putting her on a trial and the suggestion that his prescribing was inappropriate or unnecessary or irresponsible, what do you say?

A. Irresponsible, certainly not. I would have put probably the patient also on a therapy trial. The fact that they

didn't seem to have a lot of results is maybe due to the fact that the dose was maybe not adequate or the type of preparation was not adequate.

If we look at the type of preparation, there was no dessicated thyroid. It could be that this patient might have been better with dessicated thyroid than a T4 and T3 preparation.

Because the problem, I will repeat, of synthetic T4 and T3 preparations is that it's given once a day and the T3 will go quickly in the body and quickly disappear. So the effect of T3 will be short term. It's better to have a preparation that releases more slowly its T3 like dessicated thyroid where you have a more 24-hour on 24-hour effect and not just a 4 or 5-hour effect of T3. So it might be due to the type of preparation.

The mild improvement that was talked about was the tongue was a little smaller, the heart was a little bit quicker, but that really shows that actually there was still some hypothyroidism, I believe.

We have the same here with the lab test, so I would say the first likely cause is a wrong condition of blood intake, but the second possibility could be an eventual overmedication if she took the thyroid hormone, if she did the blood test correctly. But this I cannot know

with the information I have here.

Q. I understand. Well, again, we've not heard from Patient C as to when --

A. There's a blood test I found, it was not clear to me if she was still on the treatment at that moment, of 21/9/04, where the TSH is too low so she probably was on the treatment, yes. Here it's not so easy to know if she took the thyroid medication before the blood test or not. But again, she rather is hypothyroid, I would say she's certainly still hypothyroid based on the information I have from Dr Skinner.

Q. Well, again, do you have any criticism of Dr Skinner's actions with regard to this patient?

A. I think it's completely following the guideline of the International Hormone Society, and I think it's an adequate treatment that didn't have the time to go off to maybe a more efficient medication.

After stopping we have the same, the TSH goes back to where it was before, so normally there's no harm to the thyroid gland for such a treatment. Again, I have to repeat there are studies where the patients were taking for more than 20 years and in 3 weeks or 4 weeks time it comes back to normal.

Q. Let's go to the last patient, if we may, Patient D.

A. So Patient D medical history was more abundant and this

was the patient with autoimmune disease in the family, and a lot of different sorts of thyroid dysfunctions in the family, hypo and hyper. When a person has been hypothyroid many of those patients end up by being treated -- end up later on with hypothyroidism.

So when there's already history of autoimmune disease there is generally -- because it's also personal, so she normally has autoimmune antibodies, and those patients are more likely to develop hypothyroidism. That's a well-known fact. So the medical history is quite strong for at least a mild form of hypothyroidism.

And the complaints: lack of energy since more than seven years, typical for endocrine deficiencies. How to spot the difference between a wrong food intake and have a lack of energy caused by wrong food intake on endocrine deficiency. Endocrine deficiency is every day you have the complaint.

Frequently sleeping during day time, a major symptom of hypothyroidism is sleepiness, you sit and you fall asleep, or you tend to have what you call somnolence. That is very typical, it's not in other endocrine deficiencies described as such.

Constipation, quite typical for low thyroid function.

Muscle and joint pains, but if it is caused by low thyroid function, it's all over the body, it's not just in one place.

Headaches, frequent in hypothyroidism, and they are often very well relieved through thyroid medication.

Lower pack pain. Why do patients with hypothyroidism have lower back pain? The ligaments are more relaxed. A child has flat feet typically on low thyroid, and later an adult will keep on the flat feet, and the back is more bendy and you have more pain and typically, in my experience, it really works to give thyroid hormones to relieve this sort of pain.

I've seen, not only in a child, but also in an adult with hyperlaxicity, a sort of bended back, hyperlodosis it's called, to bend it back, straighten up with thyroid hormones in one year's time.

So memory loss, typical, comes often in low thyroid function.

Depression, of course. It's more a morning depression, you get better when you move.

Decreased interest, typical. They get apathetic, they're not interested in things any more because they lack energy and they lack, I would say, spirit.

Q. We heard that vividly from Patient D.

A. She testified?

Q. She did.
A. Okay. Slow movement, slow speech. This is very typical for low thyroid function.

Physical sign, being overweight. This is a sort of massive obesity, that is not only caused by low thyroid function. Low thyroid function, the patients tend to be swollen up and to be obese, but not massive. If it's massive, there's also another problem.

Thyroid gland is enlarged. That's a reason often also to give, even if she has normal lab test, thyroid hormones, because it could enlarge further into a goitre, to a bigger thyroid, but it depends. I have no information on how large it was. So it depends on the volume.

I would have done here an echography of the thyroid gland. There's moderate bradycardia, 65 per minute.

Rough hair. The hair is rougher, it's thicker in low thyroid function, but dry. It's very typical, it's actually myxoedema of the hair, it's thickened by also myxoedema.

And swollen tongue by myxoedema. The waste products accumulate in the tongue.

So I would say clinically in history the real typical person, and we'll look at the test here.
Q. She gave the results and the Panel have them in the

documentation at bundle 1, tab 8, page 4, she gave some test results.

A. Yes, I found them also elsewhere.

Q. They're certainly in the GP records at tab 7.

A. The first results that you see here are normally results that the patient, if I well understood, had before taking any thyroid hormones. I just put them with the dates in 2003, 2004, and then I have a lab test where I didn't put the date here, so I can't help you there. That was what I thought was the initial lab test, before treatment.

You see that TSH is 1.9, so I would say it's near the 2, which is possibly associated with disease in subgroups of patients.

And the free T3 is 5.7, which normal actually.

And the T4 is 14.2, which could be near the lower tertile, so the blood tests are less conclusive in this case, this is really, I would say, a sort of borderline case where the lab tests are not 100 per cent bad, they're not optimal but you do have some hesitation, are you going to do a therapeutic trial or not.

Q. Can I say that the lab test that is that Dr Skinner did are at tab 8, page 16. Those are the TSH results of 1.9 and the T4 level of 14.2.

A. Yes.

Q. The earlier results, if the Panel want to remind themselves, are at tab 7, page 5, where in 2003 -- I don't think we have the month because it has been chopped off on the photocopy that I have certainly -- there's a TSH level of 1.1.

A. But to make a clinical diagnosis you take everything. If you have normal blood test, which is more or less the case here, but you do have severe complaints, I would say a therapy trial is justified in this case, because also it was severe symptoms, that were not released in seven years, she had terrible lack of energy.

Q. I think it was six, but perhaps the point is the same.

A. Well, it's five, six or seven, it would be the same. So I would say justified.

Also the familial history of Hashimoto, those are patients who easier will make hypothyroidism during their lifetime. So I think it was justified, and the fact that the therapy was given gave a sort of major improvement. Much more energy, less depression because they're awake during the day, this is what we typically see with patients.

I had also recently a patient like that, that had thyroid hormones before, was taking thyroid hormones but was still depressed and fatigued, and we searched over because she didn't respond so well, she came from

Germany, and after three months of treatment, the husband said to this doctor: I cannot believe this, she has laughed more during these three months than in the whole life I had with her. 30 years I lived with her.

She had laughed more. I think she must have been terribly depressed all the time, that woman, but it shows you, this is not because the doctor has charisma. That is not possible.

A placebo effect takes a certain time, but at the end the placebo effect will die off, but this person will stay in a good condition because the cause of her depression, of her fatigue, was treated by a better thyroid medication.

So it's a bit about the same enthusiasm that you see in this patient, although not everything is relieved, about 90 per cent seem to be relieved, if I could have the information I have found in the file.

I would say this justifies the treatment and the therapeutic trial itself. Then as a treatment, small doses were given up to 200 micrograms.

The patient is a good converter of T4 to T3 because with thyroxine she had major improvement. Again, we have blood tests that are above reference range also for the free T3, which I think is very good to ask.

Q. Can I pause you there. Just to give the Panel the

reference to the lab tests. The Panel will recall that at tab 8, page 44, the lab tests show all the results that you've recorded, the 27.2, the less than 0.1 for the TSH, and the free T3 at 7.7.

A. So here we have two possibilities with these lab tests. The patient is clearly overdosed, or she has taken the thyroid hormones before.

Apparently there's no recording of hypothyroid signs. I would opt that by far the most likely cause is the fact that again she took the thyroid hormones before, which had never been done during treatment. Before a blood test, no thyroid hormones.

Q. Let's come on to your view about this patient. The Panel did hear her evidence about how her life changed significantly once she was on treatment.

A. I think this is a very important -- you need in the UK to have an alternative. When you look at the position of the endocrinologists, official endocrinologists that are actually diabetologists, is that you only treat the lab tests, you do not treat the patients, you treat their lab tests at least for hypothyroidism, and such a patient would not be treated then. She would be considered fully normal and all the rest is psychological.

What were her symptoms again ... Let me see here.

Her headaches, memory loss, decreased interest is psychological, because the thyroid hormones are normal. So she would remain her lifetime with those symptoms without the treatment.

Imagine what a miserable life and what tragedy to continue with those symptoms. If the treatment is a placebo effect after six months, it dies off, it does not remain, and it will never get that sort of improvement.

So I think it's very important to consider that we're not talking only about lab tests here, we are talking about living patients and living humans, and I think this is the classical case that motivates physicians to say: it's not all lab test, it's also clinical symptoms and we have to treat.

Q. It motivates the clinicians.

A. It motivates the clinicians very, very much.

Q. Again, the criticisms of Dr Skinner are that he should never prescribe for this patient, that it was irresponsible for him to do so, unnecessary, and inappropriate. What do you say?

A. I would say not treating her in this case and not doing a therapy trial is inhuman, it's not human, humanitarian. You at least have to do a therapy trial in this case. There's enough data to do it, and the

fact that it worked is a good indication.

There is the problem that because of this controversy that she is now not any more on the guidance of a physician. This is not good, but what other choice does the patient have? Remain with all those symptoms or be a normal person by taking her thyroxine through the Internet? And it's the responsibility of the physicians to have an ear open for those patients and to have the possibility of being able to do therapy trials with those patients without overdosing them, of course.

Q. The other criticism of Dr Skinner in relation to this patient is that he didn't examine her properly, didn't take a proper history. What do you say about that?

A. I don't see this, when you look at this, compared to the GP's notes I think it's very evident that there is data written.

Q. I should have asked the same question in respect of Patient C, and I didn't. Did you see anything in Dr Skinner's examination or history taking with Patient C which falls short of what he should have done?

A. I have found less information about the complaints, the physical signs, maybe I didn't look good in the files, it could be. But there was enough information to justify therapy trial, so I think we can say it's satisfactory and it was, I would say, even better in

Patient D.

Q. Right. Thank you. You deal with other matters in your report that follow on, but I think we've dealt with them all in the other aspects of the evidence that you gave yesterday.

A. I summarise a bit about the four patients, of what I see.

Q. Do.

A. Is it useful?

Q. Do summarise it, but I'm sure you can do so briefly.

A. Yes. Diagnosis of hypothyroidism in the four patients has a high likelihood of being correct and the therapy trial was therefore also justified.

So I think diagnosis, there was enough indications to do a safe therapeutic trial.

The fact that he treated I think three of the four patients at a later stage with T3 and T4 medication, including Armour Thyroid, I think can be considered as acceptable, and I don't think here it has really been argued any more that there should be a place open for T3/T4 medication (inaudible) T4.

About the follow-up, I cannot prove any real excess of thyroid hormones based on the information I have. There is never a real clinical sign of hypothyroidism noted wherever, nor by the GP, nor by the

endocrinologist, nor by Dr Skinner. On the contrary, there are symptoms often that the patient is still hypothyroid or is good, and feels good.

So biochemically I have no proof where I can really say there I have no doubt. It's most likely a wrong condition of blood withdrawal, that is very typical, when I see that the first thing I think is it's a wrong blood intake. And that misled Dr Skinner, the GPs, and the endocrinologists, and diabetologist. You'll get a copy of this, but there seems to be problem to make copies for everyone.

Q. I imagine the photocopier's feeling exhausted but we'll sort it.

A. What about alleged risk in these patients? The thyroid treatment had no effect or reduced in this case the risk of atrial fibrillation. We have seen this yesterday, that T3 can reduce the risk of atrial fibrillation, and there is no indication that it would have caused this, because based on the blood test we cannot -- the blood test was done not on the right condition.

Only one patient, I would say, could have a mild or no risk of reduced bone density following the study. That is the patient with post menopause. I don't remember which code --

Q. Patient B it would be. She was the eldest.

A. So she is at risk of having a transitory, because it's the first year that you have a reduction in bone density apparently following the study, but she does not have a risk of increased risk of fractures, because this is in the loss of bone the major problem, the bone fractures. So there is no proven risk in two studies, and I think Mr Kark showed a study that is somewhere here, where you had this difference.

And there is no proof that thyroid treatment may damage or atrophy the thyroid gland in these patients. On the contrary those who had stopped had again the same lab test as before which is indicative that there was no damage.

Now, the real risk, I really think, and this is really my belief and also my experience, is that there is a risk of stopping the treatment, and the absence of treatment of the four patients has a high likelihood, in my experience, to continue or to reappear again the hypothyroid symptoms and complaints they had, and for a lifetime, and increase also the risk of disease.

We saw that with the studies. We have seen the certain reference ranges you can have an increased risk of disease, and it possibly may also shorten their lifespan because many of those diseases like coronary atherosclerosis, that means that the blood vessels for

the heart are blocked with plaques of atheroma, and the stiffening of the arteries are linked with increased mortality.

So it is not an easy thing to do. A good diagnosis for hormone deficiency takes time. At my place we take one hour with the patient. I heard that Dr Skinner at least for the first consultation does also one hour.

But once you get all those collection of indications, I think a therapeutic trial is worthwhile, also for the long-term consequences. Not only for the short-term happiness or wellbeing of the patient but also to avoid or to prevent or to delay other age-related diseases.

So I think this risk is far greater of an aggravation of her health situation or of a maintenance of her poor health situation, than the risk we could have of an atrial fibrillation or reduced bone density, which in this case is almost not the case in any patient.

So I do have a remark I think on the practise of Dr Skinner. I think he should continue doing blood tests in patients during follow-up because there seem to be some indications that might not be so, but without intake of thyroid medication before. I think really this is the ABC of endocrinology.

I think also it's useful to have a free serum T3 because you do a normal blood withdrawal in good conditions, non-thyroid hormones before, and the patient could have high T4, and you would say then she's overdosed because she didn't take the thyroid hormones, but, no, it's not the real active hormone.

Q. Slow down a little, if you would.

A. It's not the real active hormone, the T4.

So you do need to know if the T3 is also not too high. If the T3 is too high, the T4 is too high, and TSH is depressed, and the patient did not take the thyroid medication before, she's almost certainly hypothyroid, and it will be seen on the clinical symptoms, on the physical examination.

So I do recommend that this would be done and also to take at least one of the tests for thyroid antibodies. Anti-thyroid peroxidase or possibly also anti-thyroglobulin, it does help, they have fluctuating activities of thyroid glands those people, so you do have to follow up them more closely.

To increase -- that it would have increase in tension when patients have a lower TSH or high T4, and in a particular high T3, like I just said, otherwise if there is some doubt about the conditions of the test to re-do the test. I think it has not been done but

I think it's worthwhile to re-do the test if there's any hesitation.

I think also certain notes can be improved, but maybe mostly the lecture of the notes, but also the notes when a phone call has been given. This is more information, I did not get from the reports but I get from the exchanges we had here, that notes should be improved and maybe it would be interesting to follow even the basic course, I would say, of international non-speciality because at least you get those basic principles of how you do the test. We will get a copy of this.

Q. I'm sure people will want a copy of this.

A. There was some resistance to photocopying. I asked a little too much.

Q. You clearly have one more slide.

A. I have a comment on the refusal of Dr Skinner to submit to a practice assessment.

MR KARK: Sorry, how could this -- I have let him range far and wide.

MR JENKINS: I didn't know this was coming. Take the slide away, if you would.

Are you in a position to comment on Dr Skinner's refusal to undergo an assessment?

A. I thought that the fact I was an expert, I could comment

on facts, but here it's not so?

Q. I don't ask you to comment on that aspect of the case. But if you're asked questions about it, certainly you can reply.

A. Sure.

Q. Those are all the questions I have for you. You will want to take that slide away.

A. I will take it away.

MR JENKINS: Just take it away. Move it off the screen is what I'm asking you to do. Just click on another slide. That's fine.

Thank you very much, would you wait there because you will be asked questions by others.

THE CHAIRMAN: Mr Kark?

Cross-examination by MR KARK

MR KARK: Do you understand, Dr Hertoghe, the duties in this country of giving expert evidence --

A. Yes.

Q. Either in court --

A. I do. I think I do.

Q. Good. Would you tell us what they are, please?

A. I think it's to give truthful information at every step, and the way I see it is also only to give it when I have evidence to back up what I say.

Q. It's to be independent of the side that pays you, you

understand that?
A. Yes.
Q. It's to reflect the research accurately.
A. Yes.
Q. And it's to indicate if your opinion has changed.
How did you come to be giving evidence in this case?
A. I've been asked by -- it was a patient group who said there's a doctor who might have problems.
Q. Which patient group was that?
A. Actually there were two patient groups. I think it's two patient websites. I think it's -- I know the names of the persons who control the websites. I think it's Lynne Minott and Sheila Turner.
Q. So what did you do when you got that information from those two patients?
A. I said I could eventually come and intervene as an expert, if that was the question.
Q. So you contacted Dr Skinner, did you?
A. I did not contact Dr Skinner.
Q. What did you do?
A. I got contacted, I think, by Dr Skinner's assistant to see -- Afshan Ahmed -- if I could intervene as an expert.
Q. Had you met Dr Skinner previously?
A. The first time I met him was at the World Congress of

Anti-aging Medicine in March.

Q. March of this year?

A. Yes.

Q. Was that before you wrote your report?

A. Of course that was before. Before it has been recently written, and I got no information before.

Q. When you met Dr Skinner what were the circumstances of meeting him?

A. As he was a lecturer, we went eating with several lecturers and we talked.

Q. And this was an anti-aging hormone --

A. Anti-aging medicine world congress, there are 3,000 participants and two sessions, so in one of the sessions Dr Skinner spoke in a session on thyroid therapy. There was also, I think, a workshop he did.

Q. Have you had direct contact with Dr Skinner in the compiling of your report?

A. No. The second time I saw Dr Skinner was here.

Q. Right. Can you help us: how did his endocrine survey end up in your report?

A. Which endocrine survey?

Q. Do you remember, you flashed up a screen earlier that Mr Jenkins said you should take that off?

A. No, this is just because I heard here about this endocrine survey, and I think there was also in the

papers I received for the report --

Q. There was something about that, was there?

A. -- an explanation where he was proposed and he was also refused -- he was proposed a practice assessment, and it seemed that he refused afterwards, yes. So this was reconfirmed when I heard it here.

Q. Doctor, you've been pretty scathing about endocrine practice in the UK.

A. Yes. That is true.

Q. You have attacked Professor Weetman.

A. Report.

Q. Well, you've attacked him actually on the basis, I think you said of his education, his learning, his approach.

A. Yes.

Q. Is that fair?

A. I think it's fair, because it's so evident that something is -- maybe I have been harsh, I tried to be diplomatic because it's not a personal attack.

Q. It will be a matter for the Panel how diplomatic you were. But effectively you have suggested that he has misrepresented research to this Panel.

A. Yes, I think so, and I reconfirm this.

Q. Can we just deal with your qualifications, please. We've got at appendix 1 of Professor Weetman's report, his qualifications. I just want for a moment to look

at the man who launched that attack on one of the leading endocrinologists in this country.

Are you actually an endocrinologist in Belgium?

A. No, I'm not an endocrinologist, I have a specialisation of general practition and specialisation of anti-aging medicine.

Q. Let's look at your background, please, in Belgium. We have your CV.

THE CHAIRMAN: We are on D12.

MR KARK: It is indeed, thank you.

You graduated in 1986 in Belgium.

A. Yes.

Q. That was your medical degree, was it?

A. Yes.

Q. And then you did a specialised degree, you call it, but it was your three years in general medicine, yes?

A. Yes.

Q. And then you went on to --

A. Two years, at that time, specialisation after the normal education because I was doing a specialisation in psychiatry but stopped it after four months, and then I went to specialisation of general medicine, which is the one I obtained.

Q. Right. Then you obtained your certificate, as it's called. For two years you studied anti-aging medical

therapeutics, is that right?
A. Yes.
Q. Which took you up to 2005.
A. Yes.
Q. Just help us a little bit about anti-aging medicine, would you? It may be self-evident, but what is its purpose?
A. Okay. The purpose is to lessen or decrease aging or delay aging, and it's actually the new name for what was considered before as preventive medicine. But anti-aging medicine has something more, it's that it's not only preventive, it's also therapeutic or curative.
So you try to lessen the burden of aging. For instance, when you give to a woman female hormones in the post menopause, you do lessen the signs and consequence of aging if you do it safely.
Q. Is it something that really could relate to all of us?
A. I think this indeed relates to everybody. Everybody ages sooner or later.
Q. We're all aging all the time on one view.
A. Voila. So that means you do have to have a certain good lifestyle, correct your nutrient deficiencies, et cetera.
Q. So after you had done your two-year certificate in anti-aging medicine, what did you go on to do then?

A. I'm since several years lecturer, so I'm building up programmes of lectures, seminars.

Q. Let's have a look at that. If we look at under the heading "Actual scientific organisation responsibilities", first of all:

"[Membership] of the International Board of Scientific Advisers of the American Academy of Anti-aging Medicine.

"President ..."

Does that mean you were the president?

A. Yes.

Q. "... of the European Academy of the Quality of Life and Longevity Medicine."

A. Yes.

Q. What does "Eaquall" mean?

A. Eaquall, European Academy of Quality of Life and Longevity Medicine, the society started the first -- it has another name, but anti-aging medicine congress in Europe. We had a big sponsorship of the European Union, so it had a sort of official recognition.

Q. Sorry, European money went into this, do you mean? What do you mean big sponsorship of European --

A. It's a venture, so the European Union selects certain programmes or congresses where it gives its official approval, and we also get a budget to be able to run the

conference.

Q. So European money went into the conference.

A. Yes.

Q. Excellent.

A. And we had the Prime Minister, the previous Prime Minister also as honorary president.

Q. Then 2001, you were the supervisor and course lecturer of the post university course of anti-aging medical therapeutics.

A. Yes.

Q. 2003, you did another course lecturer of the international post university anti-aging medicine specialisation, co-founder.

2004, you were the co-founder of the International Hormone Society. I'm going to come back to that because you've told us quite a bit about that. I think you said it was one of the most important endocrine societies in the world.

A. It's in membership for the moment the third and growing very fast so we hope to be in the leading position later on.

Q. Right. President of the World Society of Anti-aging Medicine, WSAM, in Rome?

A. Yes. You'll find a website also.

Q. I expect we will.

"Member of the directory Board of the European Organisation of Scientific Anti-Aging Medicine.

"Founder and member of the editing staff of a Journal of European Anti-Aging Medicine."

And president of the International Hormone Society.

A. Exactly.

Q. So that was the one that you had co-founded, and then you took over the presidency of it, did you?

A. Yes.

Q. Right. In Belgium is there a similar system to the one in England about specialisation and having to be on a specialist register?

A. Yes, there's I think a similar system. Specialisation of anti-aging medicine is not yet recognised.

Q. I expect that's right. But I think endocrinology is recognised in Belgium.

A. Yes.

Q. And there is a specialist register for it, isn't there?

A. You know it is the same way as I suppose it is in England, mostly diabetology.

Q. You are not on that register, are you, in Belgium?

A. No, I am not.

Q. And you couldn't practice as an endocrinologist in Belgium, could you?

A. I can prescribe hormone therapies without any problem,

but I cannot call myself an endocrinologist because I don't have the paper.

Q. Right. Would you be entitled to act as a general practitioner in Belgium?

A. I could, but I don't have to. As a general practitioner you can generally go in several directions.

Q. All right. So far as your lectures are concerned, we see on page 2 that almost straight after you'd finished your degree, you were off lecturing to the Broda Barnes Research Foundation. Dr Broda Barnes is one of the leading proponents of the use of thyroxine in the United States, isn't he?

A. Not of thyroxine, of dessicated thyroid.

Q. I am sorry, of Armour Thyroid?

A. Yes.

Q. Then we can see in 92 you --

A. It's quite evident that I will speak at that time because I was at that moment already practising with my father, who was an official endocrinologist. So it was, I would say, evident that I would start lecturing and providing information, because I had one of the -- I would say, a good teacher in endocrinology.

Q. The topics in which you were lecturing, let's have a look, androgen deficiencies, multiple deficiency syndrome.

A. Meaning multiple hormone deficiency syndrome.
Q. And then in 92, pituitary gland and aging?
A. Yes.
Q. 96, Broda Barnes again, adult growth hormone therapy.
A. Yes.
Q. Is that an anti-aging therapy as well, or not?
A. What I do is I treat hormone deficiencies and one of the deficiencies with aging -- when you see a person age much more, the big wrinkles, the spine atrophy with the bent back, that is typical for growth hormone deficiency and I'm one of those who treat this deficiency and am quite well-known for this deficiency treatment.
Q. Right? 97, the effects of diet, et cetera.
You lectured in Abu Dhabi, therapy of andropause, growth hormone therapy. I'm just looking for any of these on treatment of the thyroid.
A. Well, you skipped something because in 1996 there was adverse endocrine consequences of radioactive iodine therapy for hyperthyroidism. That's very important. And you probably are skipping out all the seminars I gave, but that's later where there's abundant talking of thyroids.
Q. Sorry, can you keep your voice up? What did you just say?
A. That you understood, I think, adverse endocrine

consequences of radioactive iodine therapy for hyperthyroidism. That's one of the subjects. You should not forget that. There's also a lot of topics that are not on this paper that are related to thyroid. Each time there is a workshop or a hormone therapy, many of those contain an hour or two on thyroid therapy.

Q. Coming back, please, to anti-aging and in fact you have a whole list of the seminars that you have --

A. Yes.

Q. -- dealt with. Page 5, I think. Medical educational seminars on hormone replacement therapies in aging adults.

A. Yes.

Q. What are the drugs that you use in that field of practice?

A. Well, you have thyroid therapy, female hormones.

Q. Take it slowly. Would that be thyroxine?

A. Thyroxine, Armour Thyroid, T3 and T4 combinations.

Q. Right. What else do you use in anti-aging medicine?

A. What I do is I treat deficiencies in persons because many people confuse anti-aging medicine with aesthetical medicine, so the word itself is a little unsatisfactory. We're treating patients, so what I use is thyroid therapy in general, cortisol therapy if it's a deficiency in cortisol. I use growth hormone if

there's a deficiency in growth hormone.

The female hormone therapy, that's oestrogens and progesterone, transdermal estrogeon, which is much less at risk of breast cancer and progesterone. Testosterone, male hormone. That can be transdermal, that is through the skin or injectable.

Q. Does anti-aging therapy have the effect of appearing to halt the aging process?

A. It does. There are several studies that show that this helps. I have published myself a study that cancer incidence was decreased in those therapies, all the patients who came in 2005 in my practice.

Q. And anti-aging medicine an enormous business, particularly in the States, isn't it?

A. Yes, the States have a sort of commercial outlook. That's why I think it's so important that Europeans who are, let's say, much more evidence-based and a little more careful -- I think it's better that they get more importance in the movement.

Q. All right.

A. It has always been my goal.

Q. Could I just ask, you are in private practice, are you?

A. I am in private practice. I work with six physicians who I supervise.

Q. So you don't receive, as it were, a salary from any

company?
A. No, I never have been receiving a salary from a pharmaceutical company or whatever.
Q. You told us quite a bit about the International Hormone Society.
A. Yes.
Q. You described that as one of the most important endocrine societies in the world?
A. I think it's going to be really one of the most important.
Q. How many actual endocrinologists do you have?
A. I think that we have something around 30, 40 endocrinologists.
Q. Have you got any from Great Britain?
A. No, we don't. We have doctors from Great Britain, but we don't have any endocrinologists. I think it would be quite difficult with the positions of the British Thyroid Association, but it would be a good idea to have a section here of international ...
Q. From this book, there are contributions from endocrinologists all over the world, aren't there? Saudi Arabia, Canada, America, United Kingdom, Netherlands, Denmark, I think there's one from Belgium. Are any of these going to be members of your society?
A. Well, the goal is that once, let's say, we are quite

a big society is to try to approach those people to make a sort of major congress together, and that is true that it's one of the goals in the next two or three years.

Q. Where is the society based, as it were?

A. Now we have changed location and we're putting it in Brussels.

Q. Where was it based?

A. It was based in Kansas City.

Q. Did you go over to Kansas regularly?

A. No.

Q. So how did it work?

A. Well, it didn't really work in the beginning and one of the reasons I went out to practice is to really reinstall and make a strong society. That's why now there are references, consensuses are published, and I turn around to get the signatures on the therapies' consensuses.

Q. Who was the president before you?

A. The president before was a person named Dave Johnson, who is not a doctor.

Q. He wasn't a doctor? What was he then, was he a businessman?

A. No, he's not a businessman, he works as a computer specialist, I think, and in America the system is different. In America it seems to be better to have

a sort of neutral person than a doctor at the head of a society. That is what I was told. But I didn't find it a good idea and thought it was a better idea to -- it was a transitory post for him.

Q. So he was a computer specialist?

A. Yes.

Q. Were there any doctors involved in the hierarchy apart from yourself?

A. In the what?

Q. Sorry, at the top of the foundation.

A. Do you mean in the past or now?

Q. When you started the thing.

A. When I started it was with Dr Jackie Springer, who was co-founder of the society.

Q. Right. And is she still part of the structure of the society?

A. Not any more because we "re-found" the foundation in Brussels.

Q. Sorry, is she still part of it or not?

A. No, not in the directory board or not in the foundation.

Q. Why not?

A. Because she didn't work, she didn't do anything for it, so I think it was important to get only people we can count on and are active.

Q. How did you come to found it with her if she was your

co-founder? Were there just two of you?

A. She is an endocrinologist. She was very active -- I thought she was going to be very active, but except of the foundation of the society she didn't do really very much, so I thought it was not a good idea to keep it located where -- because she lived in Kansas, where people don't do a lot.

Q. People don't do a lot in Kansas? All right.

A. Yes, but not Dr Jackie Springer.

Q. When did you found this society, when was that?

A. I have to go back to my CV. Some years ago. But we only really started to open up for membership recently, and it goes very quickly, so in 2004.

Q. Wasn't one of the problems that in November of 2004 she had her licence to practise removed from her?

A. It is also one of the reasons but she had her licence suspended for prescribing Armour Thyroid and prostamol.

Q. She had her licence suspended, I suggest, because she was prescribing growth hormones without performing adequate examinations of her patients, is that right?

A. Yes, and I don't know if it was true, but that was unacceptable.

Q. And she was running a weight loss clinic at a place called Overland Park in Kansas City, which got closed down.

A. That I didn't know actually about the weight loss clinic, that it closed down.
Q. You didn't know that?
A. Not about that, no.
Q. Really? She was charged with illegally importing a banned drug called Fenfluramine, it's the weight loss drug?
A. I heard about that. That is also unacceptable, yes.
Q. You didn't mention that earlier as one of the reasons that she wasn't any longer --
A. I didn't think about that. What I had in mind is that she had her licence suspended and that was not acceptable also. But the reason was not that, because I think that some of the arguments that made her licence suspended are not correct. It's a sort of trial like we have here. So I really think she was not correct.

I have no problems of keeping a person in the society. I even asked her to be a founder of the society, even if she lost her licence, because I work for truth. I think that's the same thing that you're striving for. But the main reason not to take her up again -- she didn't want anyway -- is that she didn't work.

Q. Who else is on the board of the International Hormone Society?

A. Now we have a Vice President, Ron Rothenberg, who is an assistant --
Q. Sorry, Ron Rothenberg? Where is he based?
A. In San Diego. He's normally assistant professor in, I think, preventive medicine.
Q. Is he an endocrinologist?
A. No, I don't think he is. Don't forget that this society is not a society only for endocrinologists, but open to all medical specialities. What we claim is that there's adequate hormone therapy that can be done inside of, I would say, other medical specialities. That's the critical point.
Q. Dr Hertoghe, the reality is you've never trained as an endocrinologist and you have nobody at the top of your society which you are --
A. The what?
Q. You have nobody at the top of this society. You are putting this society forward to this Panel as one of the leading -- let me remind you of your words: one of the leading endocrine societies in the world.
A. It's the leading society of the other medical specialties. I said this very clearly, I don't think I have withheld any information, and it really is going quickly.
Q. Would it surprise you that Mr Lynn and Professor Weetman

have never heard of your society?
A. It doesn't surprise me. They look in another direction.
Q. They're looking in the other direction? Contrary to what you --
A. They have never heard, neither, of all this evidence that they stated that didn't exist.
Q. We're going to look at that and look at how accurately you portrayed that.
A. Good.
Q. You seem to think that training of endocrinologists in this country is a year or a year and a half. Is that really your view?
A. It's the view I explained that was internationally like this and most of it is in internal medicine. In France I know it's three years of endocrinology, but the problem is still mainly with diabetes patients.
Q. I accept that endocrinology and diabetes are very closely linked, aren't they?
A. Yes.
Q. And I'm going to suggest that the training is a minimum of three years and it normally requires a period of research, leading to a masters degree?
A. Yes.
Q. Can we just look, please, at your uses of the drug thyroxine. Do you use thyroxine to treat obesity?

A. No, never. I treat thyroid deficiency and this is also something I teach to all my students, only to treat a hormone deficiency, not to treat a symptom. Don't give a hormone for a symptom.

Q. Page 31 of the printout that we've got of your report. It's not actually in the report, it's in the slides.

THE CHAIRMAN: Mr Kark, can you give us the reference?

MR KARK: D14. Thyroid and obesity, weight loss and T3 treatment.

A. I don't have the right pages.

Q. Sorry, page 31. Do you see thyroid and obesity?

A. Yes.

Q. Weight loss and T3 treatment.

"Greater weight loss if Tri-iodothyronine is given." So that would be T3?

A. Yes.

Q. Do you use that in treatment of obesity?

A. No. It just shows that when you treat a thyroid deficiency you can have many effects, the T3 that you add with the treatment can help to decrease obesity, et cetera. But it's just one of the side consequences, I would say, of treating the main problem, which is the thyroid deficiency. If you see very well the references, it's a reference I have found actually in The Thyroid, the big book you have there.

Q. You didn't, actually, because it's not in this one, it's the last one.
A. It's the recent edition you have.
Q. I think the one you're using may be something like 10 years out of date. This one doesn't make reference to it, does it?
A. But that doesn't mean that the study is not good. That just means that you have here an idea of what importance it can have, thyroid hormones, because it can help on different levels. You never can treat one symptom or one at a time.
Q. Depression.
A. Which page?
Q. Page 88, I think, is where you mention this topic.
A. Yes.
Q. There are a number of reports there that deal with the use of thyroid treatment for bipolar effective disorder and depressive disorders.
A. Yes.
Q. Just help us about that. I want to try and understand what you're saying. Are you saying that clinical depression can be treated with T4 or T3?
A. No.
Q. Or are you saying that people are depressed because they're hypothyroid, and therefore, if you treat the

hypothyroidism, that will cure the depression?

A. Hypothyroidism can be associated with depression. Not every depression has hypothyroidism. Depression can be caused by other endocrine deficiences or by stress situations.

This is even totally different. I don't even know if the patients were normal thyroid or hypo. It's just to prove that certain patients who were probably euthyroid, very probably euthyroid, receives by -- this is therapy that I do not recommend. This is a therapy that has been done, and I just cite the references to show that evidence by the experts you have has been omitted, and that this is one of the evidence that shows that people with normal blood tests are treated with thyroid hormones, it's very different from what you have suggested.

Q. If you have somebody who is clinically euthyroid but depressed, let's say depressed and overweight and lethargic --

A. If the patient has a lot of symptoms of deficiencies, and there's history suggestive and the blood tests are like you say in a near or in the reference range --

Q. Within the reference range.

A. -- where there's pathology, that part of the reference range that has pathology, I think it's a therapeutic

test -- a trial, a therapeutic trial, is justified. But if the person only has depression I never will give thyroid hormones. I need more data, that's not enough.

Q. Let me ask the question again. Let's take an example. We have got depression, overweight, and lethargy. Feeling tired all the time.

A. I need a clinical examination. If I don't find any signs that are suggestive of hypothyroidism, they're very typical, you have dry skin at the elbow, you can have swollen eyelids, the rough hair. So there are many signs. You can have slow Achilles reflexes. This is a sign that is very typical to low thyroid function only. You need to have much more data. I wouldn't treat that, that's not enough for me.

Q. One of the problems is that a number of these symptoms that you're mentioning are also symptoms that could be the result of many, many other problems.

A. Not slow Achilles reflexes, no, that is very typical, that is standard known. The slow Achilles reflects this is typical for low thyroid function.

Q. Are you saying that's diagnostic of hypothyroidism?

A. It's diagnostic, not on its own but that's an important symptom, yes. It's an important physical sign.

There are things that are very typical. You cannot have, I would say, cold extremities or fully keratosis.

If the vitamin A is normal, you still have full keratosis, it is very typical for low thyroid function. They are very typical signs.

Q. Let me just finish off the list that I'm going through. Anti-aging you've dealt with already. Certainly something that thyroxine would help with.

A. If the person is low thyroid, otherwise it would create a hypothyroid situation, which would increase aging, so it's only if there's a deficiency.

Q. It follows from what you have said about the attitude of endocrinologists in this country that your belief would seem to be that the use of thyroxine should be made much more widely available to people who are at the moment regarded as chemically euthyroid.

A. But hypothyroid symptoms and blood tests in the part of the reference range that may have link to pathology, yes.

Q. We'll look at that but we've just been looking at patient who was, as I think you said yourself, certainly they were well within the reference ranges, but nevertheless --

A. Patient A to D, you mean?

Q. I think it was C actually. But in any event, we will go through the patients. In any event, you thought a trial of thyroxine --

A. And certainly that patient she had severe symptoms. You really can have cellular low thyroid function in people who may have normal blood tests, but most of those do have at least borderline low thyroid hormones and borderline high TSH.

Q. Can we take a person who is within the reference range, and for the moment I'll accept a reference range of TSH below 2.5.

A. Okay.

Q. I know you are, I think, pushing to go even lower.

A. Yes.

Q. But the problems of the human condition are many and varied, aren't they? We can be too fat, too thin, we can all feel listless, lethargic and tired. Some people have transient depression and some have depression as an illness. Quite a lot of us find it difficult to get out of bed in the normal.

A. And you find that normal?

Q. Many of us suffer from back pain.

A. Yes.

Q. And we all, sadly, get old.

A. Yes.

Q. Can we all do with a touch of thyroxine?

A. If you're hypothyroid I think you will benefit from the treatment. If these are your symptoms, maybe we should

examine.

I mean, I don't want to make a personal question, but I think we have certain tools, we live at a certain time of our -- a time where we have tools where we can decrease some of that aging.

Q. We could decrease some of that aging?

A. Slow it down. That doesn't mean we are magicians and that we can do everything, but we can lessen some of the burden of aging.

Q. Are you content to use thyroxine for a host of problems of the human condition, which have nothing whatever to do with a thyroid gland being sick?

A. Yes. I wouldn't do thyroxine for things that have nothing to do with thyroid hormone. You do really have to have confirmation from several aspects. If you only have complaints and you have physical signs that are not conclusive, I wouldn't treat with thyroid hormones.

Q. But the list of what I described as problems of the human condition that I had just given you, you would regard as signs of hypothyroidism?

A. There are so many endocrine deficiencies that I wouldn't directly say, but what I showed you, I think you have seen those slides, is that all tissues age and the body stays the same size as it was at a young age when most of us had a good hormone level. So if the hormone

levels decrease -- and I showed you the receptors -- the cells get slowly more and more hypothyroid.

The big difference between the concept that you seem to, let's say, defend is that only a minority of people can have hormone deficiencies, and the position of doctors that work in anti-aging medicine are in, let's say, biochemical euthyroid, clinical hypothyroid patients, is that there is much more deficiencies. There are intimate degrees of deficiencies.

Q. Right. Just before we break for lunch, can you help us: where would you like the TSH level to be lowered to?

A. I would like TSH levels to be between 0.5 and 2 on the average.

Q. So if you lower it, you would have a host of people who would have no signs or symptoms of hypothyroidism --

A. Those I will not treat.

Q. Hold on a second. If you're outside your TSH reference range, so you're above your reference range of 2, the argument would be that they would be subclinically hypothyroid, wouldn't they?

A. No, subclinically hypothyroid is a TSH above 4, 5, or 5.5, depending on the reference range. So you mean --

Q. But why? If you're bringing your reference range down to --

A. Well, that's the definition of the -- oh, I see what you

mean.

Q. Dr Hertoghe, you want to bring the reference range down to 2, yes? So anyone who is over 2 is abnormal, is that right?

A. I understand what you mean. Again, it's written in the consensus, it concerns clinically hypothyroid patients. If you have a clinically euthyroid patient, and biochemical euthyroid, I wouldn't treat that patient, it's very clear.

Q. No, I am asking you -- I will try and get this in before lunch -- if you have somebody who's at 2.4, your reference range is 2.

A. Yes.

Q. They have no clinical signs of hypothyroidism --

A. I wouldn't treat the patient.

Q. Why not?

A. Because he has no clinical signs, so that means in the cells it's okay.

Q. So whatever their TSH is, you wouldn't treat them unless you see signs, is that right?

A. I'm almost sure that if the TSH is above 4, or 4/5, you certainly find clinical signs. I haven't seen a patient at 4, personally not, maybe endocrinologists here in this country have seen that, but I haven't seen a patient at 4 not having clinical signs of

hypothyroidism on physical examination. But I still would like a little safety zone that some people may do well at a TSH of 3 or 3.5, I would let this this safety zone. I wouldn't treat the patient.

Q. Help us with this: what proportion of the population, do you have any idea, would have a TSH of above 2?

A. The average mean of a population in general is around 1.5. So 50 per cent are above 1.5, and I don't know exactly the population that is above 2 or 2.5. It probably is something like 30 per cent or 25 per cent or 35 per cent. I don't know exactly.

Q. So just pausing there for a moment. If the 12 people sitting round these tables, I'm not, with respect, including the patients, four of us probably would be over your range at which you would treat --

A. Yes. And I can give you a very good comparison to understand that. How many people of us will not need glasses to see better to read? Do you know how much?

Q. Tell us.

A. Everybody after age 50 will need glasses to read. I don't need them yet, but I probably will.

Q. So we're all going to need thyroxine at some stage, will we?

A. If we age and live long enough, it is possible, but still there can be an individual that may resist better,

but the scientific studies show that. Everything ages all the way.

Q. We're aging perhaps if we don't get some lunch, so shall we pause there for the moment.

A. Sure. But you understand the comparison with people who have difficulties to see, if we would put reference ranges and only 2.5 per cent of the people who are over 50 may get glasses because they are definitely almost blind, I would say, we need to treat and to give glasses to people who also have a more moderate deficiency.

THE CHAIRMAN: Thank you, we will break for lunch until 1.50. Thank you.

(1.00 pm)

(Lunch Adjournment)

(1.50 pm)

MR KARK: I want to turn, please, to some of your criticisms of Professor Weetman, and obviously the Panel will have to make up their minds about that. I expect you would accept that his CV, which the Panel have at appendix 1 of his report, was impressive? (Handed).

Would you like to have a look through that. I presume you've seen it before?

A. No, I didn't. Is it here at the beginning?

Q. His CV is attached as appendix 1 to his report. Did you not have it?

A. I have it now.
Q. Did you not have it before? So when you commented on his education, his training, you hadn't read his CV; is that right?
A. I haven't read his CV, what I was commenting is on a part of the training, the practical training with patients' treatment of hypothyroidism.
Q. You have a look now, would you, at his CV?
A. Yes.
Q. When we start in 1974, when he qualified with first class honours, and just leaf through it, and tell us please, if you would, where it is, in your view, lacking.
A. He has been accredited in general medicine, endocrinology and diabetes mellitus, 1987.
I see no place where there's specific training in treatment of patients. I suppose it is -- I don't know if it's diabetes. There's no indication. There's only positions in hospitals and research centres. Neurology here. General medicine, previous appointments. No, I cannot see any -- maybe you can see some.
Q. You see the difficulty, I suppose, is that we don't know of his previous appointments. We see that started off as a house physician, then senior house officer, then registrar in general medicine, then Medical Research

Council Training Fellow, honorary registrar department of medicine in Cardiff, senior registrar, lecturer of medicine.

Over the page, top of page 4, Medical Research Council, travelling fellow. Wellcome senior research fellow in clinical science, honorary senior registrar at Hammersmith Hospital, senior lecturer and honorary consultant physician.

A. Where do you see that he specialised in thyroidology in that part?

Q. I think we can take it that he did. If we to the membership of editorial boards, Clinical Endocrinology, thyroid, 1993. Editor Clinical Endocrinology.

A. But that's mostly research papers. It's not practice with patients. Or do you see other information?

Q. Presumably you read the transcript of his evidence; did you?

A. I read of the fourth day, I read about half or two-thirds of his disposition. But I based my impression on his report where he talked about clinical symptoms of hypothyroidism without knowing them, and --

Q. Oh, I see.

A. And with embarrassing comments also.

Q. Hold on, let's get that right. You thought that Professor Weetman's evidence was embarrassing, did you?

Can you with just look --

A. In the sense that it didn't seem to be correct information.

Q. Right.

A. But it's not for me to put an opinion on why it is or why it may be so.

Q. Well, to repeat an expression we heard earlier in the case, I think you just have. Let's have a look at page 6 of his CV just to look at his internationally invited lectures. From 1991, I think it is, it is almost all specialising in endocrinology and diseases, problems with the thyroid.

A. But that does not mean he has clinical experience, that means that he does research or he does lectures.

Q. Okay. Let's look at your specific criticism and I think at paragraph 12 of your report you say this:

"Dr Weetman illustrates his point of view that 'none of the symptoms or signs just listed' would be 'sufficiently sensitive or specific for the diagnosis of hypothyroidism ..."

A. Yes.

Q. "... even [I think it should be] when combined together'."

THE CHAIRMAN: Sorry, Mr Kark, guide me to the reference.

MR KARK: Paragraph 12 of Dr Hertoghe's report.

THE CHAIRMAN: Is that D13?

MR KARK: Yes.

A. It should be page 5 of the report.

Q. Page 5, paragraph 12.

This is under the issue of the symptoms and signs of hypothyroidism. Can I just ask you this: was it your understanding that what Professor Weetman was saying was that all that you do is you look at the laboratory, the blood laboratory tests for the patient, and then decide whether they're hypothyroid or not, is that your understanding of his evidence?

A. So if I well understood what you said, is if it was my understanding that Professor Weetman would base mainly diagnosis --

Q. No, not mainly. Was it your understanding that Professor Weetman was saying, you look at the lab test and you decide if they're hypothyroid or not?

A. Yes, that was my understanding. He didn't find the clinical symptoms sufficiently interesting, I would say.

Q. No, not sufficiently interesting, sufficiently for a diagnosis of hypothyroidism. That's what he was saying.

A. I would share his view if he would say that they're not sufficient on their own. It depends a little bit on the severity and where also the blood tests are in the lab.

Q. Can I just ask you to pause for a moment because there may be a language issue, and I don't mean that in any rude sense. If you say that something is diagnostic of a particular disease, what do you mean by that?

A. When you say this is on its own enough for a diagnostic reasons, that means that based on this information you can make the diagnosis of the disease, and treat the disease.

Q. Right, okay, so we do have the same meaning of "diagnostic of".

Let's look at this criticism of his evidence or his report. You deal in your paragraph 12 with what Dr Weetman said about using symptoms and signs of hypothyroidism and you repeat this:

"The authors of the study conclude: 'Evaluation of symptoms and signs of hypothyroidism with a new score in addition to thyroid function testing is very useful for the individual assessment of thyroid failure and the monitoring of treatment', which contradicts Dr Weetman's analysis of the study."

Yes?

A. Because it means that you do have to take into account those clinical symptoms, which he seemed, in my way of interpreting his report, not to be important to add to the diagnosis, and it seems more in accordance with what

he later writes.

Q. I think you may return to this topic at page 15 in your report, paragraph 38.

You write this:

"The study Dr Weetman writes about [and that's the same study, I think, appendix 4] can be interpreted differently.

"1. It does not show that symptoms are inadequate for the diagnosis of thyroid function."

Let's just pause there for a moment. Are you saying that the symptoms are adequate for the diagnosis of thyroid function?

A. I'm saying they're symptoms -- the symptoms are adequate as one part of the diagnosis, maybe I should have precised that.

Q. Yes, I think that's important, isn't it.

A. It's an important difference.

Q. You are not saying, are you, or perhaps you are, that symptoms themselves can be diagnostic of hypothyroidism?

A. Not on their own.

Q. Right:

"The authors of the study rightly write that 'Evaluation of symptoms and signs of hypothyroidism with a new score in addition to thyroid function testing ..."

That may be important:

"... in addition to thyroid" --

A. Yes, that is fundamental I think.

Q. "... is very useful for the individual assessment of thyroid failure and the monitoring of treat."

Yes?

A. Yes.

Q. You were very critical, were you not, about Professor Weetman's use of this report?

A. Of Professor Weetman's report?

Q. Yes.

A. Yes.

Q. You said you didn't understand why he said what he did.

A. Why he said it differently.

Q. Yes.

A. Because it's not in the conclusion that -- what he said was:

"None of the systems or signs just listed would be sufficiently sensitive or specific for diagnosing hypothyroidism and this reinforced what he's talking about.

THE CHAIRMAN: Could you bring the microphone just more in front of you? That might help. Thank you.

A. I will repeat, maybe. So what he says, he illustrates his point of view that:

"None or the symptoms are signs just listed to be

sufficiently sensitive or specific for diagnosing hypothyroidism even with combined together."

What I understood is that he meant that symptoms were actually not that important and that it's not necessarily a contributing part of the diagnosis.

MR KARK: I don't think he said either of those things actually. You see, your criticism before this Panel, you made some comment, I'm rather cautious about my note, my recollection is you didn't understand why he failed --

A. Why he changed the meaning of the --

Q. Why he changed the meaning.

A. Yes.

Q. So you're suggesting that Professor Weetman has changed the meaning?

A. Yes.

Q. Right.

A. You read that in the context, that is really the impression I have.

Q. What Professor Weetman said to this Panel was this, and he's dealing with his report, I asked him:

"At the bottom of page 5 you're dealing with the same area, you say, 'In addition none of the signs and symptoms just listed is sufficiently sensitive or specific for a diagnosis of hypothyroidism even'" --

A. Sorry, I do not follow you. Where exactly is it?
Q. I'm reading from the transcript, which you don't have, so if you could listen.
THE CHAIRMAN: It is on page 5, the last paragraph, in about the middle if that would help.
MR KARK: That is right.
All right?
"In addition, none of the symptoms or signs just listed is sufficiently sensitive or specific for a diagnosis of hypothyroidism even when combined together."
Are you saying he's wrong about that?
A. This is when you read this sentence, it could be interpreted differently.
Q. I see.
A. What I read here, "In addition none of the symptoms or signs [so none, not one of them] just listed is sufficiently sensitive or specific for the diagnosis of hypothyroidism".
You could interpret it alone that the symptoms do not contribute to diagnosis together with the lab test or you can interpret anyway you don't have to take into account the signs and symptoms.
Q. And he also --
A. So the sentence could be interpreted differently than

the way I did interpret it.

Q. Are you --

A. But the context I would say would rather point in the direction in the way I would interpret it, that he in a certain sense rejects clinical symptoms as a part of the diagnosis of hypothyroidism.

Q. You were critical also, if we go to page 6 of Professor Weetman's report, of the last line:

"As the authors conclude, even this detailed scoring system could not be used to establish a diagnosis that the results are also important in quantifying how common the clinical problem of suggestive hypothyroid features in healthy individuals actually is."

A. Well, I think it's a different interpretation. It's interpreting a study that does not really say that.

Q. Right.

A. Because you can interpret it that on the contrary, that you do have some hypothyroidism in patients who are obviously in the reference range, so it depends where the point of view is.

Q. What you have quoted from the report, which we have at your paragraph 12, beginning with the words "the authors of the study conclude", is from the abstract of the report, isn't it?

A. Yes.

Q. Did you bother to read right through the report before you launched this attack on Professor Weetman?
A. In this case I based myself on the abstract because I found that the abstract was sufficiently conclusive in this conclusion.
Q. Would the answer to the question then be: no, I did not read the full report before I launched this attack upon Professor Weetman's conclusion?
A. I read the full report.
Q. Oh, you did?
A. I did not receive the article.
Q. I'm sorry, did you read the article? It's Zulewski, isn't it?
A. I didn't receive that article.
Q. Did you ask for it through your solicitors, because they I think had the references and you could have found this quite easily.
A. Yes. I searched because there was -- got the papers of course, quite late so I had to do the report in, I would say, quite short time in an overabundant programme.
Q. Sorry, you had to the report in quite a short time, and what?
A. Well, so I based myself on the abstract, and on the abstract I think the things for me are quite clear.

Q. Doctor, I'll just ask you this: did you think before you decided to include in your report and indeed in your evidence before the Panel, where you criticise as effectively misleading one of the senior endocrinologists in the country, did you think it would be sensible to read through the report that he was quoting?

A. I think if I had the article it would have been much better. I'm sure about that.

Q. Good. You can have it now. Let me pass it out. (Handed).

I have a copy for the Panel as well.

THE CHAIRMAN: Thank you.

This will be C9 then.

MR KARK: Thank you.

This is very dense material and I am not going to trouble this Panel to read through any more despite what I've just said to you, Dr Hertoghe. But could I suggest that you, first of all, look at the front page. We can see that right on the front page we have a heading "Abstract". What you have done, I think you agree, is you've taken the last few lines of the abstract and put it into your report.

A. I agree.

Q. And to try and give a thumbnail picture of what they

were trying to do here, is they were trying to create a scoring system, really, for the classical signs of hypothyroidism. Would that be fair?

A. And they found less symptoms than before.

Q. They found what?

A. Less symptoms than previous studies two years ago.

Q. All right. Could I just ask you, because I don't want to spend very much longer on this topic, to turn to page 776 of the report.

A. Third paragraph?

Q. Absolutely. Third paragraph, you got there before me. Would you like to read that?

A. Yes, I would like to:

"Faced with the variability of the clinical findings in overt and subclinical hypothyroidism, we cannot recommend the use of the new clinical score for the purpose of establishing the diagnosis of hypothyroidism."

This when I read it is meant that it's solely -- it's not enough on its own.

Q. Yes?

A. "Routine thyroid function testing is the best and most reliable way to identify patients with thyroid failure."

Q. Would you accept having read that, that what Professor Weetman was doing, rather than just quoting

the abstract, was he was quoting directly from the body of the report?

A. Yes.

Q. Do you withdraw your criticism?

A. Actually that all depends on how to interpret the words of Dr Weetman. It all turns around this: if he meant that none of the symptoms or signs just listed would be sufficiently sensitive or specific on their own for the diagnosis of hypothyroidism, even when combined together, if he had put that, I would say then he is right in quoting the article, except that he did not take into account also the conclusion.

Q. Well --

A. So he has omitted the conclusion, but took part of the text that was adapted to his report in the sense he wanted to orient it.

Q. Well, Dr Hertoghe --

A. It's a little difficult for me to -- what I was asked is to analyse how reliable was this report.

Q. Yes.

A. And I find that there is a difficulty in interpretation at least.

Q. Can we take it, and it may be because you simply had to do your report quickly, but can we take it that where Professor Weetman has made his various references,

comments about the references, you haven't read them all?

A. I have read the abstract, certainly.

Q. You've read the abstract, but you haven't read all the reports.

A. But I haven't read all the articles, certainly not.

Q. So whenever we see in your report a criticism of Professor Weetman's report relating to research, we ought to be aware that you haven't actually read the research, you have read the abstract.

A. I have read part of the research, I have read the articles of Dr Weetman, I have read various articles, but it's true that when you don't have so much time a way of doing it is of limiting it to the abstract, but that doesn't mean that the information there is not accurate. It could mean that in certain points the information would have to be specified, like here. It's double sense of what he -- that's the impression I have, personally, that is double sense, it's oriented to give another impression than what the article really says.

Q. When we started, doctor, this is exactly why I asked you the question, I asked you whether you understood the expression "diagnostic of", in other words you can draw a diagnosis from particular features. That's exactly the expression Professor Weetman used,

I suggest, "the diagnosis of"?
A. So you mean it's sensitive enough on its own to make the diagnosis of hypothyroidism?
Q. It's not, is it?
A. It's not in the report of Dr Weetman, that's not what I read. For me ...
Q. Let me move on. TSH levels. You have told us, and I'm sure you're right, that TSH levels fluctuate during the day. Yes?
A. Yes.
Q. Is it also fair to say that they fluctuate in a very minor way compared to the fluctuations that occur at night?
A. Yes, certainly.
Q. Right.
A. There's less fluctuations during the day.
Q. Is it still accepted to be the safest and most accurate test of thyroid function that there is?
A. The most I would say it's -- you have to combine testing and --
Q. T4 and T3 as well?
A. On its own it's not sufficient as well. I wouldn't say it's sufficient on its own.
Q. Do you accept that it is a very important test --
A. Yes.

Q. -- of the thyroid function?
A. Yes. Absolutely.
Q. You're not saying that because you do a TSH test during the day you should ignore it?
A. No, absolutely not.
Q. I don't think you said this but I just want to make clear. You're not suggesting that TSH testing should be abandoned in favour of clinical signs, are you?
A. No. I'm not suggesting that.
Q. And there's not a single piece of research anywhere, I expect, that you've ever read that would support that suggestion?
A. What suggestion?
Q. I would suggest there's no piece of research anywhere that would suggest you should abandon TSH testing in favour simply of clinical signs?
A. No. Well, like you talked yourself, Dr Broda Barnes, he did write things in that sense, but I don't suggest that you should abandon TSH.
Q. No, Dr Broda Barnes is not just at one end of the field he's almost out of the field completely, isn't he?
A. And it's also a long time ago.
Q. All right.
A. But that doesn't mean that he doesn't given valuable information.

Q. But no one seriously challenges the importance of doing a blood test, do they?
A. No. I don't think so. Nobody here anyway.
Q. In not just ideal circumstances, but it would be preferable, wouldn't it, always to do a blood test before you do treatment so you know what your baseline is; is that fair?
A. Yes.
Q. And that would simply mean saying to your patient: look, I need to take a blood test before we start anything at all, go away, come back next week and we'll see where we are?
A. Yes.
Q. Once someone is on thyroxine, to whatever extent, their TSH is going to be suppressed, isn't it?
A. Going to be decreased in level, yes. Suppressed means in a certain sense totally --
Q. I don't mean it in that way, it's going to be decreased.
A. Decreased.
Q. If they are on a lot of thyroxine, I was going to say too much, but I'll say a lot of thyroxine, their TSH level is likely to be suppressed so that it becomes unrecordable.
A. Yes.
Q. Now, I didn't say too much because there are certain

circumstances where that's exactly what you want to do. For instance, treating thyroid cancer.

A. Yes.

Q. You would want to put so much T4 into the body that the TSH is going to be completely suppressed, the thyroid's going to stop working, and it's going to shrivel up.

A. And it has the anti-cancer effect.

Q. All right.

A. And the studies show that generally it's well tolerated, this sort of treatment.

Q. But in normal circumstances when treating somebody for hypothyroidism, you would not want to see a TSH which is suppressed in that way, would you?

A. It's not my goal. My goal is the patient feels good, looks good, and has thyroid test within the reference range although more optimal. But you do have a fraction of the patients who apparently have a degree not only of primary hypothyroidism, but also of central hypothyroidism. That means they cannot secrete easily TSH as much as other people like elderly people, and it may be that a fraction of the people will have, with the correct treatment, a suppressed TSH. But that won't be all of the patients.

Q. I understand that. Just to make it clear, when you talk about central hypothyroidism, it's something I think

we've been referring to, but it's the same thing, as secondary hypothyroidism.

A. Yes. Well, secondary and tertiary. Tertiary means that the hypothalamus is weak, and the secondary means that the pituitary gland is weak, so central means the two.

Q. I understand. Obviously if the patient is sick and the pituitary gland sometimes gets a disease, or it may have a tumour, yes? And if you put T4 into the body, there are circumstances where the TSH would become totally suppressed.

A. Yes.

Q. But presuming all else is working, as it were, you do not want to put so much T4 into the system as to totally suppress the TSH, do you?

A. If I can avoid that, it's better.

Q. Why?

A. Because it's still one of the signs during a follow-up of overdosing. You can, when you overdose, have a suppressed TSH. Here with the lab tests I had here, I could not deduce the information because the suppression was most likely due to a morning intake, but generally I prefer to keep it between, let's say, 0.1 and 2, or like I said, even optimally between 0.5 and 2. But some patients, like I said before, also may have some, in the cells, deficiency and they may need some

higher dose, but it is not the majority.

THE CHAIRMAN: Could I just clarify, 0.1, is that 0.1 or 1.0?

A. That's 0.1. Under this reference range you can have increased losses of bone density in post-menopausal women and other eventual pathologies, so I prefer to be above that level.

MR KARK: That is, just dealing with that, a recognised risk. Nobody knows how serious the risk is but it's a recognised risk --

A. Well, it's a moderate risk because it's not accompanied by fracture risk and we see this also in the cancer patients.

Q. There are studies that say it may be accompanied by fracture risk, you heard last --

A. It may be but, again, not every patient is the same so you still have to evaluate the patient. I think what we should avoid is bringing the patient in what we call a hypothyroid situation. If they have the morning blood withdrawal in appropriate conditions and they have a suppressed TSH that is not detectable with a good laboratory, you may have some patients that will do fine with that and okay, but it's still a minority of patients.

Q. Your goal is to keep it within the reference range?

A. Within the reference range or at least above the 0.1.
Q. When you heard the evidence of Dr Skinner -- were you here when he gave evidence?
A. Yes, I was here. I was here the whole week.
Q. When you heard the evidence of Dr Skinner that there was no TSH reading with which he would be uncomfortable, was that slightly --
A. I did not agree. But I think what was meant, if I give you comment -- or not? Okay.
Q. I think with respect you can comment on what he actually said. The Panel will have to work out what he meant by it.

The reality is that if somebody is on long-term thyroxine, their TSH is going to go down. If they're on too much thyroxine for a long time, their TSH is going to be suppressed under negligible.

A. Yes.
Q. Say for two or three months.
A. But some also hypothyroid at 0.2 or 0.3, so you have individual differences.
Q. If someone's on thyroxine for, say, two or three months or longer in fact, their TSH is going to be completely suppressed if they're being overdosed.
A. Yes.
Q. And it doesn't much matter when you take the blood test

for that person, because that TSH isn't going to recover for some time, it might recover a week or ten days later.

A. That's not true.

Q. Tell us about that.

A. Like the picture I showed. If you want I can show it again but I know you prefer not.

Q. I'm sure we'll remember it.

A. You had a decrease of the TSH during 13 hours, half the time. Then it went up again. So with a normal thyroxine, even if you have a suppression, let's say, the whole first part of the day it's very likely the TSH will go upwards later on.

Q. What study have you done in relation to people who have been on thyroxine for longer three months that would demonstrate what you've just told us --

A. The study I just showed you.

Q. -- where the TSH is totally suppressed?

A. Well, this is my experience. If I have patients who have been with a suppressed TSH and they stop the treatment, but it's more experience, it's like you say it's not a study, but experience also is part of here the expertise, is that they don't have any problem, they recover. You saw also these patients. The patients here under Dr Skinner, you saw when they stopped the

treatment although they seemed to have a suppressed -- although I think it is for other reasons -- TSH, the TSH came back to normal. Even in patients who would have a suppressed TSH I think it would come back normal.

There are studies rats for instance but that is for other endocrine deficiencies, where they give an overdose in male hormones to rats and the testicles, or the blood levels of the old rats when the treatment was stopped were the same as in young rats, that means that not only was the suppressive treatment did not have any adverse effects but the testicles of those rats produced the amount of young rats while they should have produced the amount of old rats that is much lower, and it's very likely it's the same with the thyroid function.

Q. Very likely? I suggest you're wrong about that. If a person is on thyroxine for three or four months and they're being overdosed with thyroxine, I suggest to you their TSH is going to go down. I'm not saying their gland can't recover but I'm going to suggest their TSH will be completely suppressed.

A. And will remain completely suppressed later on, that's what you mean.

Q. Yes.

A. No, I don't think so. And on which study do you base this information?

Q. Well --
A. You do have a study to read for us, or don't?
Q. Dr Hertoghe, your evidence about why these particular patients, the TSH was suppressed in these particular patients, is based upon your assumption that the blood test was taken shortly after taking thyroxine, is that right?
A. Not even shortly, five hours later you can have still this effect.
Q. So we have to assume that the doctors who ordered these blood tests, presumably including Dr Skinner, I don't know, didn't know that it would be a bad idea to take a blood test when the patient was full of thyroxine.
A. That's what I read, the report exactly, the first impression I get.
Q. So we're assuming that doctors are not used to saying: before I take take a blood test from you must either fast or you not have sugar --
A. I think the fasting is something people say, but for a medication like thyroid hormones I think they didn't do it.
Q. You think they didn't do it right?
A. No, they did not say it.
Q. Simply based on the test --
A. I have no indication that is so, and normally there

should be at least one indication. It should also in the reports of the endocrinologist here, and I see no information, here except for a little sentence of Dr Weetman.

Q. That may be because it was thought to be blindingly obvious that --

A. Maybe, but the type of test, what really is determined for me that the patients took it before is that there was apparently no clinical signs or symptoms of overdosage, neither in Dr Skinner's report, nor in those of the GPs or the endocrinologist who have seen the patients. That seems to me very likely that this is the cause, and I would have preferred having a test additionally two weeks later or one week later, whatever, in the appropriate conditions.

Q. Clinical manifestations of hypothyroidism. I just want to deal with some of those that you have raised. Let's just deal with headaches. You have quoted and printed part of this book in your various slides.

A. Yes.

Q. If we have a look at page 17, just dealing for the moment --

A. Which?

Q. This is of the slides, and it's of the first bit of the slides.

THE CHAIRMAN: D14.
MR KARK: Thank you.
THE CHAIRMAN: Remind of me of the page.
MR KARK: Starting at page 17. I think you repeat it where we can see that under "Symptoms of hypothyroidism", you have --
A. Headaches.
Q. Have you just photocopied this or is this a print that you've done?
A. This is a Powerpoint.
Q. Yes, that's not a photocopy of it that you've scanned.
A. No, it was later.
Q. Have a look at that, this one we're looking at there --
A. What I said was it was an adaptation. So you have here headaches but what I added is especially diffuse and migraines. This is a sort of lecture to inform what sort is typical.
Q. Right. You have put that down as a symptom of hypothyroidism?
A. Yes.
Q. And then you're quite right, you have indeed photocopied part of the book. If we go to page 51, you've put next to it "New edition" on the left?
A. This is not the real new edition, this is the edition previous, but the page number is the one for your book.

Because I looked up in your book, you had a confirmation, but I didn't have the possibility to photocopy.

Q. So you did have available to you the new book when you wrote this?

A. Yes, and that's why I add that now in the new book it was caused as a symptom of tumours, and not in the first.

Q. Page 52, another bit of Powerpoint.

A. It's the same actually.

Q. You've actually written in a special little section there, "Headaches equals symptom of hypothyroidism".

A. Yes. These are the articles here.

Q. Do you remember you expressed surprise that neither Mr Lynn nor Professor Weetman knew that given that it was in the book that Professor Weetman had actually contributed to?

A. Yes.

Q. Can I show you the page from the book that you saw.

A. The difference with the new edition is that they have added it's in case of tumours.

Q. It's in case of what?

A. Is that the point you wanted to make?

Q. Just wait until the Panel have it, would you?

THE CHAIRMAN: This is C10.

MR KARK: If we look at the top of the page, first of all, clinical manifestations of hypothyroidism.

Symptoms: fatigue, lethargy, sleepiness, mental impairment, depression, cold intolerance, hoarseness, dry skin, decreased perspiration, weight gain, decreased appetite, constipation, menstrual disturbances, arthralgia, and paraesthesia.

Signs: slow movements, slow speech, hoarseness, bradycardia, dry skin, not pitting oedema, hyporeflexia, delayed relaxation of reflexes. So headaches don't make a mention in there.

A. Yes, but you already have given a certain way the answer.

Q. What's that?

A. You have listed very few symptoms here. So here are some of the most characteristic symptoms of low thyroid function, but that does not mean that these are the only ones.

Q. And we don't see anything about side vision hallucinations there, do we, either?

A. Yes.

Q. Let's have a look at the bottom of the page, which is the bit you photocopied from the old book and apparently had available to you:

"Clinical manifestations of specific causes of

hypothyroidism.

"Finding: headache, cause of hypothyroidism, pituitary or hypothalamic tumour."

A. That is true.

Q. Could you just help us: you've been very critical of the experts called by the GMC, about them not quite giving the full picture. Why did you not choose to photocopy that or make it clear when you gave your evidence?

A. What do you mean exactly? Because in the first -- what I photocopied it's clearly inside. Clinical manifestations.

You have in a previous edition, which is probably just before, it's written in the whole column. But what is the textbook actually? A textbook contains -- tries to summarise the most important findings. It does not mean that if the symptom is not here, it's not a symptom of hypothyroidism. In probably other textbooks you might find it. That is why I added, when I talk about the headaches references, where you see clearly an association with headaches in hypothyroidism and that means also that when you give thyroid hormones, those headaches may go away.

Q. Let's just go back to page 17.

A. Of my report?

Q. Of your slides. You presented this to the Panel,

symptoms of hypothyroidism. The right-hand side of the page:

"Headaches (especially diffuse).

"Migraine."

You printed your reference there as being from Werner & Ingbar, from this very book. Now, that is totally misleading, isn't it?

A. No, because at the moment I showed the slide I clearly said it was an adaptation. An adaptation of the text. So I took many of the symptoms -- for instance you will never see morning depression because the morning depression is not described in the textbook called the thyroid. So that's why I specifically added this information.

Q. Do you think, I just want to have your evidence clear, that you made it clear that in the bible, as it were, as I think it might be described, the bible of thyroid problems, Werner & Ingbar, you think you made it clear, do you, that in fact when you refer to headaches, those were actually headaches as a result of the pituitary --

A. In your book for the moment. But in the other book it was not specified. It was specific causes of hypothyroidism.

Q. So when you say in the other book, which other book?

A. Well, it's the Werner & Ingbar previous edition.

Q. Let's have a look at that then. Page 51. Do you see page 51 is where you've actually copied the old book.
A. Yes.
Q. Symptoms and signs associated with specific causes of hypothyroidism. Diffuse or nodular goitre, and then symptoms and signs of pituitary or hypothalamic tumour. Headaches, visual impairment.
A. Yes.
Q. You think that was clear, do you?
A. No.
Q. Right. Let's move to?
A. That's why I quoted other studies that were much more clear.
Q. Is this the Maryland study?
A. Which study are you talking about?
Q. The study you told us there was a study that did show headaches were a common symptom of hypothyroidism. Is that the Maryland document you gave us?
A. The list where you had headaches is symptom of hypothyroidism, and those are in the article you received yesterday, I believe, about headaches.
Q. This is from Maryland, was it Maryland University?
A. I think it was the other article from the Headache Association or something like that.
Q. You see, looking through -- you get all sorts of reports

don't you?
A. Yes.
Q. And you can have patient surveys, you can have articles in magazines, then you can have research, you can have peer reviewed research.
A. Yes.
Q. You can have articles and peer reviewed articles, and there's a gradation of scale there?
A. A gradation absolutely.
Q. Headaches do not appear, I suggest, as a classic manifestation of hypothyroidism in recognised peer reviewed research.
A. I don't think so.
Q. All right.
A. I think the studies you have here from Cephalgia is a peer reviewed journal. Journal of Clinical Endocrinology and Metabolism is the number 1.
Q. Okay. Of course if --
A. You don't have everything in a textbook in the right place.
Q. I understand. If Werner & Ingbar is right that specifically headaches are linked to pituitary and hypothalamic tumours --
A. Yes, also that's not the only cause.
Q. I understand that. Just pause for a moment, and I'll

ask the question.

If that's right and headaches are linked to those particular problems, when you are looking at a patient that you think might be hypothyroid and they're complaining of headaches, what red flag ought to go up in your brain?

A. Well, actually for me it's not the classical symptom of hypothyroidism. I know that if they have headaches and I can discriminate with typical signs of hypothyroidism like the typical dry skin, the elbow keratosis, the swollen eyelids and also the panoply of other signs and symptoms, then I know there's a high chance that their headache will disappear, and with thyroid therapy, that doesn't mean it's the absolute therapy for it and I would not give it for headaches alone.

Q. Is there a serious danger that by giving that person, complaining of headaches, thyroxine, which may well get rid of their headache, you are going to conceal pituitary or a hypothalamic tumour?

A. No.

Q. Why not?

A. Because there are other signs of deficiency when you have a pituitary tumour. You have a decrease in secretion of all the other hormones and the TSH, FSH, LH, SATH, growth hormone are all very low.

Q. Then you should look at those, shouldn't you?
A. Yes, you should look. When you see these other signs you should look, but there's also signs like visual impairment, it's also more lateral, so there's a lot of possibilities, there should be other signs, but just headaches on their own is probably not due to pituitary tumour, but if they keep on being there, although a treatment has been installed, you should do a scan or a magnetic resonance imaging.
Q. You say even if treatment has been installed, if you have a headache, walk through the door, your patient is hypothyroid, she's got a headache, right? And you have a look in this book -- I appreciate that the book isn't everything -- and you understand that headaches may be linked to pituitary and hypothalamic tumours, you would want it make checks, wouldn't you?
A. Certainly I will, but not in all circumstances because you have common headaches. And when it comes from a pituitary tumour you really have some typical signs, like it aggravates with time so you have a medical history suggestive of it, but I agree with you completely that you should not wait too long to do additional tests.
Q. Let me turn to another symptom that Dr Skinner apparently found and which caused an eyebrow, to be

raised, as it were, by Professor Weetman and I think by Mr Lynn. This is side vision hallucinations. You deal with this at paragraph 43 of your report.

How many patients have you seen, do you think, over the last couple of years who you think are hypothyroid?

A. Just a moment. What page are you referring to?

Q. Page 16 of your report.

THE CHAIRMAN: Do you mind just trying to remember the Ds?

MR KARK: I'm sorry, D13.

Page 16, paragraph 43. This is your report.

A. Yes.

Q. I asked you over the last couple of years how many patients do you think you have seen who appear to you to be hypothyroid?

A. So to be hypothyroid?

Q. Yes. To be suffering from hypothyroidism.

A. I think a lot, but they also come with -- it's a special group of patients. I think Dr Skinner has the same. It's people who have already often done their self-diagnosis, they are more likely to be, so the group is bigger.

Q. Pause there a moment before we come back to your report and I just want to ask about that. You've seen a bit about Dr Skinner's practice. He's a secondary referral specialist.

A. That is what I understood, yes.
Q. Are you in the same position?
A. No, in Belgium we don't have this splitting. People can come directly to my consultation, so we don't have a split.
Q. They don't need to be referred to you?
A. No, it's not an obligation.
Q. But if you were here, you would be in the same position as Dr Skinner.
A. It might be.
Q. Because people would be coming to you because they think they have a particular condition and you're the specialist in their condition.
A. I must say that one of my collaborators had this problem in Belgium and they decided that this sort of work is equal to that of the general practitioner. So it might be a different position.
Q. All right. What you say in your report at paragraph 43 is this:

"I did not know that the complaint of visual side perceptions was a typical hypothyroid complaint, but ignorance does not mean it doesn't exist."

Can I just ask you this: if you had come across a number of patients who made a similar complaint to you, presumably you would have put it in your report?

A. I would, yes.
Q. And we can take it therefore that you have not had similar experience to Dr Skinner?
A. But I didn't ask the question because when patients come to my place, they have already so many questions to answer that we stick on to those questions.
Q. Right. Well, perhaps a patient who comes to you might not mention a symptom of seeing things out of the corner of their eye, but some would, wouldn't they?
A. It probably would, but the fact -- I'm confused because I didn't expect to see this, but when I saw that their visual field effects in 73 per cent of patients was primarily hypothyroid following one study I think I'm going to search for.
Q. Yes.
A. So I'm open, that doesn't mean that I believe that there are hallucinations, but there seem to be anyway visual field effects, and I was so curious that I wanted to see if it was really possible if hypothyroidism, and I found several causes.
Q. But nowadays with modern TSH testing, how often do you actually come across visual field problems?
A. I wasn't aware of the problem, so I never checked for that.
Q. Because visual field defects I suggest are a sign of

gross hypothyroidism and you simply don't get to that position these days with TSH testing, do you?

A. Probably not. But again I wouldn't make exceptions.

Q. I'm sure you can make exceptions.

A. We have a lot of people that are diagnosed on other grounds on hypothyroid that have headaches, and the reason is they have a compression. There's a mild degree of myxoedema that compresses the nerves and gives headaches.

So why not visual side effects in the same frequency?

Q. Yes.

A. So I'm open, you know. When you're remaining open, it makes that sometimes we learn more.

Q. What you say in your report at the bottom of page 16 is:

"As you can see, there seems to me enough material to take the complaints of visual side perception seriously as a possible hypothyroid complaint."

It's not one which is recorded anywhere in the literature, is it?

A. Well, here it is recorded in one reference so it's maybe interesting to go for it. But I still --

Q. Sorry, which reference?

A. The first references. Normally we have copies of the abstract. I have it here anyway.

Q. Do you have it?
A. Yes. I have it here. We can make a photocopy. You have it, I think.
Q. We do have a huge file but I'm afraid --
A. The big file where you were --
Q. Do you want a copy of it?
A. You have this. It's maybe just -- I think you have the full article where we talked about headaches. 51, I think. And late on ...
Q. Can you tell us where in your report is the reference?
A. 55. Page 55 in the slides.
Q. That's visual hallucinations and psychosis occur more often in hypothyroidism.
A. Above.
Q. Oh, visual side perceptions as a hypothyroid complaint. That is referring to visual field defects again, isn't it?
A. Yes.
Q. Yes.
A. It could be different but I don't know. Defects is a very general term.
Q. I thought you had accepted that nowadays, hopefully, you only get visual field defects in severely hypothyroid patients.
A. I'm not sure about that because the skull is

a non-expandable bone structure and whatever oedema you have, there can be too much, so in severe hypothyroidism it is certainly much more frequent. I think it must be more frequent. But you might have people who have in certain tissues more sensitivity to a lack of thyroid hormones and more myxoedema around there , and could have visual side effects so I stay open.

Q. It's not something you've ever seen?

A. I cannot reject -- I never checked for the -- I'm certainly going to check in the future.

Q. Finally, this, I think, on the causes of hypothyroidism. You mentioned at page 4 of D13 --

A. I don't have the number, is that the slides?

Q. Your report. Page 4. I'm just trying to find it. I'm trying to find the point at which you discuss aging as a --

A. Aging of the thyroid?

Q. Aging of the human body as one of the causes of hypothyroidism.

A. Well, that was in the slides.

Q. Ah, sorry.

A. The bigger one.

THE CHAIRMAN: D14.

A. Page 20 and 21?

MR KARK: Okay. Can you just help us with this: are you suggesting that signs of aging should be treated as a symptom of hypothyroidism?

A. No. I've written an article in the annals of the New York Academy of Science. It's a very extensive article, I think it's more than 1,100 references, that aging could be caused by multiple endocrine deficiencies.

One of those is the thyroid function. You see here in these slides a lot is aging, but you have this for most of the other endocrine systems, there's an aging on all sides but the body stays at the same size and volume and it apparently needs more or less the same levels than you have in young adulthood. So this is one of the aspects of aging.

We age also with a lower production of thyroid hormones and lower levels in our cells.

Q. Are you suggesting, I don't know the answer to this, that the normal changes which occur in the body as a result of aging require treatment with thyroxine?

A. For the thyroid axis? Yes, if the thyroid axis has too low levels, you will have all those possible pathologies and the people feel not so good, so they need treatment. So sooner or later, if you live long enough, the likelihood is very high that you might need thyroxine or

other thyroid preparations.

Q. Can we turn to the disadvantages, if any, of using thyroxine. I think you accept that if a patient is overtreated with thyroxine, they may suffer bone density reduction.

A. Yes.

Q. Do you accept also that if a patient is overtreated with thyroxine they may suffer episodes of atrial fibrillation?

A. I think it is possible, yes. But a real overtreatment, not biochemical, but complete overtreatment, yes, and hypothyroidism also.

Q. What do you mean not biochemical but a complete overtreatment, when is an overtreatment?

A. You need to have clinical signs also. To be sure that you have clinical signs it's a whole picture. And I can tell you when the thyroid hormones are too high, when the blood tests are really too high, done in appropriate conditions, you usually do have clinical signs.

Q. Dr Hertoghe, isn't that slightly circular? If a person is suffering from heart palpitations, that may be one of the signs of hypothyroidism?

A. Yes, and the typical sign is tachycardia, but regular tachycardia, that's not typical for atrial fibrillation. But you may have atrial fibrillation if you really

overdose.

Q. You don't have to have a whole list of other signs before you conclude this patient is being overdosed. If a patient is receiving thyroxine, their TSH level has disappeared down so that it's negligible, and then they suffer from heart palpitations, you, I expect, would very quickly want to reduce the dose.

A. Yes.

Q. You would recognise that immediately, wouldn't you?

A. Yes. When a person is clinically overdosed, which happens from time to time, you can always not do things 100 per cent correctly. One of the cardinal signs -- if I hesitate, I listen to the heart, it pounds very hard and quick, but regular.

If it is really seriously overdosed, the risk of atrial fibrillation I think is certainly increased, but you do have to have a 24-hour excess, because what happens with the treatment, when they take their treatment there's higher peak value and higher dose during the day than -- lower values at night, so they might be only temporary, part of the day.

Q. Sure. So you might have to do --

A. So it's less dangerous. That's why I think, my interpretation of all those studies, the studies that show increased risk of atrial fibrillation is generally

always in people who are not being treated but have spontaneously too high hormones.

Q. But if you do think a patient is being potentially overtreated, what's the basic test you want to do to find out what's happening with their blood?

A. Well, if it's during a treatment and it's just a slight overdose, of course we just reduce the dose, we see it comes into correction. But generally the patient has always come also with a blood test two or three weeks before, so we have at the same time the blood levels that will at that time in appropriate conditions, generally also be overdosed.

Q. The medical way, the scientific way of establishing if a patient is being overdosed with thyroxine is to take a blood test, isn't it?

A. That's included but it's not sufficient.

Q. I understand that. I understand that you say that you need to have clinical signs as well.

A. As well.

Q. It's a basic test.

A. Yes.

Q. Can we deal with the idea of back stacking and failing to convert T4 to T3. I'm going to touch on this very briefly.

If you have a patient who isn't responding to your

treatment, even quite high treatments of thyroxine, what are the possibilities?

A. Well, the first thing I would think that there might be other endocrine deficiencies. For instance, a woman who is in the post menopause might need female hormones.

My experience is you need to treat all the deficiencies in order for a person to feel optimum. In these cases here I think, for instance the post-menopausal woman might benefit from a safe treatment with female hormones, she would have better improvement.

Q. So what would you do to find out if that was the right thing to do?

A. I would do a blood test and a clinical examination in the same sense, and questionnaires over the typical symptoms of low female hormones.

Q. You would be looking for alternative problems to the thyroid, wouldn't you?

A. Yes.

Q. Because one possibility, apparently, is that they're failing to convert but there are a myriad of other possibilities?

A. Yes, but generally we already know if they are poor converters because either they have a low T3 because (inaudible) the T3 and a higher T4 in the blood, that is

easier, borderline low T3 and more acceptable level of T4, or they have deficiencies like iron deficiencies that are severe enough to impair the conversion.

Q. I think you just said it, but the test that you would do, you might do a number of tests, but one test you would inevitably have to do would be find out what their T3 is?

A. Yes.

Q. Because if you just keep loading them up with T4 or even add T3 to the system, you might be overdosing them with T3?

A. Yes, and maybe check the iron so that you see the cause of eventually a poor conversion.

Q. You don't just assume, do you, that the problem is still hypothyroidism but you are going to pour more T4 and T3 into them?

A. It's still hypothyroidism, but the better method that I propose is to check also other reasons why the conversion would not be good enough, but it's true that if you keep on giving thyroxine, you end up by having at the end sufficient T3, but then with a sort of abnormally high T4 and a more or less acceptable T3.

I showed you the slide where you needed with the actual treatment with thyroxine to have a sufficient T3 level to have it borderline high or above the upper

reference range.

Q. Isn't one of the things that you would have to consider, that you had got your diagnosis of hypothyroidism wrong if the patient isn't getting any better, no matter how much T4 --

A. That's why it's called a therapy trial. If the therapy trial doesn't work, you might have to look elsewhere. But there are different ways, so you can increase the dose but I think the best is to have a little larger screening of other deficiencies that may cause those symptoms?

THE CHAIRMAN: If you're finished with back stacking, shall we break for 20 minutes?

MR KARK: Could I ask what time we're going to aim to continue until this afternoon.

THE CHAIRMAN: 5 o'clock.

MR KARK: I hope I'm going to finish this afternoon.

THE CHAIRMAN: So we will meet again at 3.20. Thank you.

(3.00 pm)

(A short break)

(3.20 pm)

MR JENKINS: Can I raise one matter. I did ask you last night whether you wanted the extra slides, and I had completely forgotten this morning, perhaps because I didn't think we were wanting to see more slides. But

there are quite a lot of slides, about half an inch worth, that were copied yesterday, which should have been distributed first thing this morning. Sorry about that.

I think you have seen the relevant slides, but it is appropriate that you should have copies of them as well. I know they're available.

THE CHAIRMAN: That is fine. Is this what Mr Kark had last night?

MR JENKINS: I don't know that Mr Kark had them.

MR KARK: Having asked for them, I left without taking them with me. If we could all have them.

THE CHAIRMAN: D18.

MR JENKINS: Thank you very much.

MR KARK: I was going to turn to page 9 of your report and paragraph 17. D13. You write this:

"The US National Academy of Clinical Biochemistry, world's best recognised institute for making up laboratory guidelines, indicates that thyroid treatment should consist of maintaining the TSH level in the lower half of the reference range, that is, between 0.5 and 2."

Then you say:

"This guideline is in contradiction with Dr Weetman's view that the serum TSH should be in normal

reference range (0.4 to 0.5 or 5.5). It is also in contradiction with his 2001 editorial where he says, 'The observation also suggests to me a rationale for maintaining a TSH below 2 milliunits per litre in patienting receiving thyroxine'."

Again, I don't want to spend too much time on this, but first of all, do you agree that the aim in treatment should consist of maintaining the TSH level in the lower half of the reference range?

A. Yes, I think that's a good guideline.

Q. Right. So you want to keep it between 0.5 and 2, yes? You would have to take fairly regular blood tests to see what was happening, yes?

A. Yes.

Q. What would "fairly regular" be, quarterly, every three months?

A. In the beginning it's more often, and we are, let's say in Belgium, more limited, but I would do it every three to six months, and occasionally when a patient is on a very long-term treatment, sometimes there are patients where -- I think optimally it's every six months, later on.

Q. But at the beginning you would want to do it rather more often than that?

A. Well, thyroid treatment starts slowly, so you may not

make the patient return back before more or less two months. Otherwise there's not sufficient results. Actually, at that moment the patient is still slightly underdosed normally and by clinical observation we can know we can increase, and that is -- for instance, in Belgium we are limited. I think it is safe to eventually not ask a test if there are test restrictions. I don't know if there are test restrictions here in this, I believe so because there are not many tests that are passed. So I can do the following follow-up consultation a check, and generally I do it at every follow-up consultation.

Q. A blood test?

A. Yes. But if the dose is already quite low, it may not always be helpful to do a blood test at --

Q. If you're having to change your dosage or introduce T3 into the system, you would want to do a blood test?

A. Yes. As additional help, but also you need a clinical examination. It's always a combination.

Q. You say this:

"This guideline is in contradiction with Dr Weetman's view that the serum TSH should be in the normal reference range."

But that's his view in relation to before you start treatment, it's not in relation to once treatment has

started. You're comparing with respect two different things?

A. Yes, later on he himself wrote that his optimal values during treatment was up to 2.5.

Q. Yes, exactly. You deal with that at paragraph 22 of the same report. So Professor Weetman is aiming at almost the same, I think, he accepts 2.5 as the upper limit, doesn't he, in treatment?

A. Well, when I read the report, apparently it's what he does but that's not his guideline, I think the guideline of the British Thyroid Association --

Q. Is simply to stay within the reference levels.

A. Yes.

Q. I understand.

A. Which is apparently not sufficient, because if you stay within the reference range during day time, probably at night you are not any more in the reference range, it might be higher.

Q. But you agree that the goal of treatment should consist of remaining in the lower half of the reference range --

A. Yes.

Q. -- between 0.5 and 2.

A. Optimally. There are always patient variations. There are patients you cannot lower the TSH under the 2 or they might feel some heart palpitations. There is

absolute clinical evaluation necessary to spot the differences. Always have exceptions.

Q. Can we then turn to the upper limit of the reference range, because this is where there's some controversy. First of all, your evidence yesterday, and I have taken this, if anyone has yesterday's transcript, from page 93. I'm presuming that what we receive on e-mail is the same page number as it will be when it's printed out. That isn't always the case.

Let me just repeat your words to you:

"I was quite surprised when one of the experts said that three out of four studies I have checked in healthy and non-healthy persons, that reference ranges were the same. I've seen so much the report of that expert, I have very great doubts that whatever he says is correct. I'm a little ashamed to be able to say that, but I was so surprised either the level is not good or there is a problem there. There is a scientific evidence problem."

Now, Professor Weetman, I think what you're referring to is that Professor Weetman was talking about three out of four --

A. Yes.

Q. -- studies.

A. I think also in the words I said, I said I have to check

that information.
Q. Okay.
A. I think it's also in the -- it must be in the transcript because of my remembering, I also have to check.
Q. All right.
The study that I think Professor Weetman was referring to --
A. The three studies.
Q. Well, yes. They're actually encapsulated, I think, in one report. It was four studies and it's headed "Is there a need to redefine an upper normal limit of TSH"?
A. That's a very important article from Wartofsky or something.
Q. This one actually isn't Wartofsky, this is from the European Journal of Endocrinology, 2006, and it's actually appendix 2 of Professor Weetman's report, which I think the Panel were given. The essence of it is this -- I am going to refer to the abstract.
Do you have Professor Weetman's report there just so you can follow what I'm saying?
A. Yes, I do have.
Q. If you go to the back of it, you should find various appendices.
A. I haven't receive it, but I have read the article of Wartofsky, it's a lot of references I found in this

article. So I know what it's about.

Q. Have a look at appendix 2 just so that you can follow with me.

A. I don't have it.

MR KARK: Can my solicitor assist?

This is, as you say, of some importance. Do you remember this article?

A. Is there a need, is that?

Q. Yes:

"Is there a need to redefine the upper normal limit of TSH?"

A. Wasn't that an answer to the article of Wartofsky or something like that.

Q. It may be but I think it's one of the most recent articles on this issue that we've looked at. We can see that those who contributed to this article came from -- we can see in italics at the top. It's a very wide range of, perhaps you'd accept, impressive contributors?

A. At least the last one we're seeing is very well-known.

Q. Right:

"Abstract. Mild forms of hypothyroidism, subclinical hypothyroidism have recently been discussed as being a risk factor for the development of overt thyroid dysfunction" --

A. Excuse me, where are you reading exactly?

Q. Under the Abstract.

A. Okay.

Q. Just read it through:

"The diagnosis critically depends on the definition of the upper normal limit of serum TSH. Cut-off levels of 4 to 5 TSH have been conventionally used to diagnose an elevated TSH serum concentration. Recent data from large population studies have suggested a much lower TSH cut-off with an upper limit of 2 to 2.5 but application of strict criteria for inclusion of subjects from the general population studies aiming at assessing TSH reference levels ie no personal or family history of thyroid disease, no thyroid antibodies and a normal thyroid on ultra sonography did not result in an unequivocal upper limit of normal TSH of 2 to 2.5.

"When summarising the available evidence for lowered upper TSH cut-off values and their potential therapeutic implications there is presently insufficient justification to lower the upper normal limit of TSH and for practical purposes it is still recommended to maintain the TSH reference interval of 0.4 to 4.

"Classifying subjects, the TSH value between 4 and 4 milliunits as abnormal as well as intervening with thyroxine treatment in such subjects is probably doing more harm than good."

First of all, do you accept that this is a very respectable piece of research?

A. I think it's very respectable, yes. But it's one of the articles that answer to the (inaudible) launched by Wartofsky; they must be listed here somewhere.

Q. I just want to concentrate on this for the moment. There was one study, wasn't there, which was the study in Pomerania, if we will go to page 635, the top right-hand corner, where they reported -- if you look at the left-hand side of 635, I'm sorry this is so dense, but this is I think the only reference that I'm going to make to it.

Do you see about five lines down:

"Indeed the study of health in Pomerania Ship 1, an area in Germany with previous iodine deficiency reported a TSH reference interval of 0.25 to 2.12 in a reference population of 1,488 subjects."

Down to the next paragraph:

"Thus the only study so far providing a direct data to support an upper normal TSH limit of 2 to 2.5 is the Ship 1 study."

Then it deals further, I think, with the iodine deficiency as there had been in the population?

A. So it's a study, if I may comment, that may confirm, because if it was a previous iodine deficiency, they

have given iodine probably in the drinking water or other means, in salt, so it corrected the iodine deficiency of that region. So I would say it's an optimal population.

The other articles that I see, the Danish study and other German study, you did not have such a correction. Well, Germany is iodine deficient country. Denmark I thought also. So I would say close analysis would say it's probably the most important study of those three that I see here.

Q. Right. Which one is the most important study, the Pomeranian --

A. My impression is this one, because, if I'm right, it's a corrected iodine deficiency region, so you really have a more optimal population because iodine deficiency can cause increases in TSH and low thyroid function.

Q. That's not, with respect, what this report found, is it? If you go to the second paragraph again, I don't think we have to go into the detail quite so much:

"Thus the only study so far providing direct data to support an upper normal TSH limit of 2 to 2.5 is the Ship 1. However, despite their proper exclusion criteria, the Ship 1 study probably still included people with autonomous functioning areas in the thyroid as judged from the fall in median values with age

probably reflecting the previously moderately severe iodine deficiency in the Pomeranian population."

So the problem with that study was still an uncorrected iodine deficiency, wasn't it?

A. Yes, but if you then correct iodine, the TSH would be even lower in that region.

Q. Where do you say the TSH value should be?

A. Optimally it should be in the majority of people between 0.5 and 2, but there's a range of people between 2 and 4 that are good at that level. They have no clinical hypothyroid symptoms, I wouldn't treat those patients.

There's part of the population between 0.1 and 0.5 that may have hypothyroid symptoms because they have a sort of central hypothyroidism. So I would eventually also treat those patients, so it's not that simple.

I need to have this confirmation by the clinical exam to know if I can treat. There's a grey zone where some people might be treated because they have a lot of complaints and a lot of signs of deficiency and some people shouldn't.

Q. The American Thyroid Association guidelines, which were revised in 2002, I think note that the optimal TSH range has been narrowed to 0.3 to 3.

A. Yes.

Q. That is the standard presently in the US, is it?

A. Yes. Well, for the endocrinologists who are part of that organisation, so they have lowered the reference range.
Q. If you --
A. But I would still think that there could be a person at 3 or 3.5 who might not need thyroid hormones because they're clinically not hypothyroid. There will be probably some exceptions but still.
Q. What is the range in Belgium?
A. The ranges in Belgium is between 0.4 and 3.5 or 4.0.
Q. Right. If you have somebody outside the range in Belgium of, say, 4.5, but without symptoms, there is an argument, isn't there, for treating that person because of the danger that in the long-term hypothyroidism will develop? Yes?
A. There might be a danger, but I need still -- I would say, normally most of those patients do have clear symptoms of low thyroid function. But if they absolutely have no sign of thyroid deficiency, I would recheck later on the value to see if we do have a pathological value or not. I wouldn't rely on one test when I find no clinical signs of deficiency.
Q. This, if I may say, is Professor Weetman's approach, isn't it?
A. Voila.

Q. If on the other hand they are beyond the reference range, say 6 or 7, but showing no clinical signs, would you treat them or would you wait for clinical signs to develop?

A. That's a more difficult question. I would do more testing, but I never encountered this situation.

All patients who are above 4 I saw clinical signs of deficiency. Physical signs like low Achilles reflex, that is the reflex of the heel when you hammer, put a hammer on it, it goes slowly, it's very typical. I almost never saw a patient with a TSH higher than 6 or 5 that did not have a slow reflex. So there are typical signs.

So it's a situation that is theoretical, but I have never encountered it. So I would certainly do much more screening, try to do more testing, examine maybe two months later the patient. Because I think doctors should not work dogmatically, here under this limit people should be treated. We should by examining the patient, doing the test, have a sort of interpretation. Why would doctors be necessary otherwise if we don't have this sort of possibility of interpretation?

Q. Well, what you're looking for, when you are doing a blood test of this nature, is you're trying to find out if there's any sickness in the thyroid, aren't you?

A. Yes. Absolutely.
Q. I see shaking heads opposite me, but that is your view, that you are trying to find sickness in the thyroid?
A. Yes.
Q. Right.
A. Additional information.
Q. If you find signs of what you say are signs of hypothyroidism, some of these classic signs that we've seen here, and you treat that patient with thyroxine, it's likely they're going to have to stay on a level of thyroxine for a long time if not forever.
A. Well, normally it's a sign that they have a definite deficiency, that they need to be on it. But if it is not the correct treatment or for one of the other reasons the patient wishes to stop, his thyroid function comes in the 3 to 6 weeks later completely back to the results of before.
Q. That is what I was going to ask you, if you stop your treatment and you've been treating a patient for some time but you find that when you stop your treatment actually the patient's T4 levels and TSH levels are exactly the same as they were before, within the reference range, in fact --
A. Yes, after 3 or 4 weeks.
Q. The answer to that might be thought to be: there was no

thyroid disease in the first place. That would be right, wouldn't it?

A. It could be right. Not in all circumstances.

Q. But if you're treating thyroid disease, and you're treating it with thyroxine, and then you withdraw your medication, you would expect the thyroid to have got worse in the meantime?

A. Yes, but it's only for two or three weeks.

Q. With these patients that we're going to turn to in a moment, those that came off thyroxine, that's exactly what happened, isn't it?

A. Yes.

Q. They reverted.

A. That is classical.

Q. To exactly where they had been before. Yes?

A. Yes.

Q. With any disease you would expect there to be a gradual deterioration, would you not?

A. With age, yes.

Q. It's not just age, we all --

A. With any disease the fact that you give a medication doesn't mean that you're suppressing the thyroid gland so that it will atrophy. Because what we're giving, we're giving a fraction of what they need.

Not all thyroxine is absorbed, it's between 45 and

73 per cent. So as we give just a fraction, we're not suppressing totally the function of thyroid gland. When we stop the small 20 or 30 per cent lower activity that it had, lower secretion, will come back as normal before.

But there's always a certain inhibition of your thyroid gland when you give a hormone, but it's a temporary, a transitory emission(?). If you stop they go back as they were before, even if they take 20, 25 or 30 years the medication, which is not normal because normal, after 20 or 30 years, your thyroid hormones should be lower, because levels lower with age. So there seems even to be a certain preservation.

Q. Have you actually ever seen a thyroid gland that's been treated with some form of thyroxine for 20 years?

A. Yes, of course. I have patients from my father and other -- yes, and sometimes the patients have stopped after 20 years their treatment, and then they come back after a year because the physician said, their general practitioner said it was not necessary, they come back after a year, and they say: I feel so awful, I should never have followed the advice.

And then they come back but their levels are not that dramatically reduced, it's just back to what it was before. I get the files from, for instance, the

patients of my father, and I have several, I would say, cases a year.

Q. Can I turn, please, to the area of your report where you deal with this, and I'm looking at paragraph 47 of D13, your report. I am going to move on to the patients.

You disagree with Professor Weetman and I don't want to, frankly, get into the battle between you on this issue.

But you say this:

"Professor Weetman's explanation may lead to misinterpretation for two reasons."

And I'm reading about five lines down:

"First, it may make people believe that 'normal' means 'healthy', which is not the case. In fact the reference ranges are not healthy ranges. Mark the limits between healthy and sick values. They are merely statistical ranges."

Then go further down the page please to second:

"It may make people believe that healthy results may lie outside the reference range, while in general it is the other way around. Values outside the reference range are generally all pathological, while some values within the reference range maybe pathological too."

A. I would take away, except the "all", it "generally", most of the time.

Q. Because I was going to suggest you take away "all", I might even suggest you take away "generally". There are occasions when people are outside reference range and you can see it no doubt as any walk through the door. There are many, many people I suggest who will be outside the reference range walking and talking quite normally.

A. Not so easily for the thyroid gland because for thyroid gland you really see the signs quite quickly. The reference ranges for the thyroid hormones are very large so they include a lot of people, healthy and unhealthy people, but outside it is often unhealthy.

Now, if we would follow the guidelines to decrease the TSH up to 2.5.2 which is suggestible --

Q. Which is at the bottom of the page.

A. -- I think then we will have a lot of people that are healthy that would be outside of the reference range.

So that is the problem, why it is not so simple. It is not a question of putting a reference range and then we finally have a narrower reference range where everybody inside is healthy, everyone outside is not healthy.

There's on either part of the reference range a grey zone where the doctor in full competency and full professional work and in full knowledge of the situation

should decide whether or not it is time to treat and then do a therapy trial and see how it works out. If the patient doesn't need it, he will be quite quickly overdosed. With thyroid hormones they will feel the symptoms, no dangerous symptoms, heart palpitations but not really dangerous, nervousness, and then we know it's too much, we have to decrease.

So we need to have a grey spend zone, we need actually to have two reference ranges. One very strict where most of the people are probably hypothyroid, and one where you have to look at the clinical situation to see if it's necessary or not to treat.

Q. Dealing with these particular patients, you appreciate, do you not, that, whether in England or America, with these reference ranges, all of which were below 2.5, the TSH levels, all of them for each of these four patients, was below 2.5 --

A. Yes.

Q. -- you would be very hard pushed to find an endocrinologist who would treat them with thyroxine?

A. Yes, in England.

Q. And America. If they were applying their guidelines.

A. Yes. An endocrinologist, diabetologist.

Q. As opposed to somebody, a member of your organisation, the hormone society?

A. And to doctors who would treat also the borderline deficiencies, because all is, what is the question here is: can we treat or not borderline deficiencies or intimate degrees of deficiencies. It is not so strict that a person is either hypo, either not. There is a progressive decrease in the function of the thyroid gland, and somewhere the symptoms get to be too apparent that there's treatment necessary for the health and quality of life of the patient.

Q. Can we turn please to Patient A. In terms of your report, I think it starts at page 26. So far as this lady is concerned, she had TSH levels which you would accept, I think, were well below the reference range for treatment in England, America, Belgium, or anywhere else you would like to mention?

A. Completely.

Q. Sorry?

A. Yes, completely agree.

Q. And we know that she had previously received advice that she was clinically euthyroid before she was referred to Dr Skinner. She went to see Dr Skinner on 16th January 2003 and her last TSH level was back in May 2002. On the basis of what you have told this Panel this afternoon, you obviously would have taken a blood test, yes?

A. Yes. Sure.
Q. We know that Dr Skinner did take a blood test. You, it would seem, probably wouldn't have started her on thyroxine until you had received the blood test back, but that may be a minor difference.
A. In most cases. There are patients who are at your consultation, they are clinically so hypothyroid and they beg you for treatment that I give once upon a time a treatment. I say: do first your blood test but it's almost sure that you need this therapeutic trial and we go very, very slowly so in the meantime you're relieved. But these are exceptions.

We don't have to forget that here are only four cases, so generally the cases that arrive to the General Medical Council I suppose it's so, are cases that are, let's say, the most atypical, the most questionable, and this is one of the cases where you could, because it's so clear, look, bradycardia, 56 per minute, cracked heels, yellow tint. It's typical, I have other textbooks where you can find the yellowish keratin as a typical sign of hypothyroidism: and how did she improve?
Q. What question are you answering at the moment?
A. Well, I would say that in this case I would have waited for the thyroid test.

Q. Thank you, so the answer is "I would have waited".

Now, when he did get the blood test back, we see that the TSH was almost exactly what it had been before, 1.4, and the T4 was 12.

2. Help us: what would you have been looking for in the blood test?

A. Well, like I've written here, the T4 is low and she has clinical syndrome of deficiency with constipation, very typical, and bradycardia, hoarse voice in the morning and external curve of the eyebrow, the sign of Dr Eugene Hertoghe. So I would have started a therapeutic trial.

Q. What would you have been looking for in the blood test?

A. I would look under the reference values of T4 and above the reference values of TSH and if not, to see if they're in a zone where there is a risk are more diseases, and one of the cases is the T4 in this case.

Q. One of the things you would think about is: I know she has some signs and symptoms and hypothyroidism, but these TSH levels are within the reference range, this T4 level is within the reference range, I ought to consider my alternative diagnosis.

A. Also, but usually when I do a blood test I have other results I can look at those alternative endocrine or nutritional diagnoses. But here, there is -- you know, cracked heels the only other pathology where you see

that is vitamin A deficiency, and here there is -- should not be a vitamin A deficiency because there's a lot of keratin, that's provitamin A, that's why there's a yellow tint of the skin.

So the pathology, the disease that causes the insufficient conversion of the provitamin A that accumulates like a yellowish pigment into vitamin A is generally low thyroid function, and so there are so many signs in physical examination, that here my first diagnosis of those rather typical symptoms of low thyroid function and signs would be to give the thyroid hormone. That would be my diagnosis of thyroid deficiency and do a therapeutic trial.

Q. When the --

A. But it won't be the only. I would still look with more attention to other deficiencies.

Q. What would you have done? Taken another blood test, what?

A. What I usually do, I do a whole screening of most of the hormones I give and vitamins.

Q. So you would have been looking for something else just in case?

A. Just in case, yes.

Q. Would you simply, when the patient phones up to say: I don't like this very much, it's making me feel

rotten and tense and I'm getting ahead headaches, would you have simply have switched on to grain Armour or would you have reconsidered the diagnosis?

A. Here I have difficulties, I don't have enough information to answer you. It's a small dose where the symptoms came, so it's not sure it was caused by the medication. It's a very small dose. I would have stuck probably with the same medication and tried to see the patient back, or probably it was on the phone, I think, that there's a switch that was made. I'm not sure about that.

Q. Yes.

A. But what I usually do is I ask all the questions, all the symptoms related to too low or too much thyroid hormones. Some patients do have at that moment -- say, "Yes, I don't sleep well, I'm trembling, I'm too warm", so the dose is too high. I would then even stop the medication. But if, when I'm asking on the phone, they have a lot of symptoms typical of low thyroid function I say, "Maybe something's wrong with this thyroxine."

You know, sometimes people have allergies to the fillings of the medication and just by changing medication, you don't get the problem. So I might then indeed go to another medication. It depends on the answers I get to my questions on the phone that are

clinical signs and symptoms that I ask.

Q. So having got those blood tests back, which show that the patient's TSH was 1.4, the T4 was 12.2, then the patient complains that the medication isn't working for her, you would simply have switched to Grain Armour, would you?

A. Yes, but I would have been comforted in this approach by finding a lot of signs of deficiency that remained. The bradycardia, that means slow pulse, the constipation and all those things. So I need to have this other additional information before making a decision.

Q. Can I ask this, on a completely different topic for a moment. How many doctors are there in Belgium doing what you do?

A. About 30 doctors.

Q. Treating hormones, hormone deficiency?

A. Yes.

Q. What do they describe themselves as? What sort of specialism is this?

A. It fits more in the speciality of anti-aging medicine. But it's just --

Q. Are they mostly members of the Anti-aging Society or whatever it's called?

A. Yes.

Q. I asked you about endocrinologists in England and

America doing what Dr Skinner did. You apply the same TSH tests in Belgium, don't you? And an endocrinologist in Belgium --

A. Yes.

Q. -- is most unlikely to have done what Dr Skinner did, isn't he?

A. But I add the T3 and I add the thyroid antibodies in initial assessment.

Q. Let's have a look at Patient B, please. This patient was very depressed. She had TSH scores in August of 2002 of 2.3 and T4 16.7, so it's a pretty healthy T4 score, isn't it?

A. It's a pity we don't have the T3 here.

Q. Yes, indeed it is. 14th January 2003, TSH almost exactly the same, 2.4, and a T4 of 16.8. So there doesn't seem to have been any problem. Between those two dates, the four or five months between August and January, no worsening of any disease, if there was any disease, was there?

A. No, so that's why a therapeutic trial is useful.

Q. And it's relevant that she was very depressed, isn't it?

A. Yes.

Q. Right.

A. And there were two endocrine causes that were suspected: hypothyroidism and menopause. This patient, I believe,

should also have been treated with female hormones to get out of the depression.

Q. Would you have done some tests before that or would you have just launched that as a trial?

A. Well, she had a test, but I would examine her also for that and ask to pass a gynaecological control. In case we treat, she must have been checked before for cancer.

Q. The only test we know that Dr Skinner had was the one from January, two months earlier, which showed her TSH to be 2.4 and her T4 to be 16.8.

A. But we had an FSH that was 52.9 and that is indicative of lack of female hormone. That's above on the top of the page.

Q. Is it page 3?

A. Page 29 on top, under depression, Beck's depression inventory, 52.9. That's indicative of menopause.

THE CHAIRMAN: Sorry, we are on page 29 and it's up near the top --

A. Medical history.

MR KARK: In your report.

A. Yes. My report, yes.

Q. I still can't find it, where is it?

A. 29 above.

Q. Oh, FSH 52.9, I'm sorry. What's the date of that?

A. I don't know. It was in the files of the general

practitioner. It was probably a year before, not necessarily very close to it.

Q. All right. We don't know from his notes how much thyroxine Dr Skinner gave, but we've heard from him and you've heard that he instituted an incremental dose. Again, looking at the TSH score, I suggest no endocrinologist in England, Belgium, or America would have been treating this woman with thyroxine at those levels, would they?

A. No diabetologist.

Q. We know that nine months later --

A. I cannot say no diabetologist because we have in our group also endocrinologists and diabetologists and they do treat like this, but it's a very small fraction compared to the majority.

Q. We know that nine months after treatment, this lady's TSH was below 0.1 and her T4 was at 39. Now, you explain that, as I understand it, in your report, by your conviction that that must have been that whoever took the blood test didn't really know what they were doing and hadn't warned the patient not to take the tablets?

A. I'm almost completely sure of that.

Q. Why?

A. If the information I have here, of course, is correct.

If there is a bradycardia of 52 per minute it's -- so it's even slower than it was before, it's almost sure that she was not even sufficiently treated, but it's the peak value after intake of the medication.

Q. Sorry, you're saying that she wasn't sufficiently treated?

A. She wasn't sufficiently treated, apparently based on the clinical examination. I never saw sort of an overdose of thyroid hormones with a patient with a bradycardia of 52 per minute, or she was taking a medication that was blocking the heart. It's really very low, you know, 52 per minute. But of course it's based on the information I have here.

Q. Can we just have a look at Dr Skinner's note of the second consultation. We have a typed version. The easiest way to find it is right at the beginning of tab 4 of the patient notes.

A. Which page number?

Q. If you find the cardboard tab 4, just after that you see a typewritten document headed with the patient's name.

A. I see here more information also with low body temperature, but it's peripheral.

Q. Would you have a look, please? It's written as 20th June 2004, second consultation. I think it actually should be the 21st.

"Taking reduced dose of 200 micrograms per day on account --"

We heard there was a slight change to that, "free of palpitations", it might be either "on account of" or "free of palpitations" for one month.

Now, 200 micrograms, is that a pretty strong dose of thyroxine?

A. Well, it's in the guidelines of the BNF, 100 to 200-micrograms, and we saw that a patient who is overt hypothyroid with 125 keeps having half of the time a too high TSH. So I think it's an acceptable dose if it's tolerated, if it doesn't cause any sign of overdoses. But again here, 56 per minute with an enlarged tongue. "Less swelling of feet", that means that they are still swollen. "Hair less dry" means actually that there's still some persisting signs of thyroid deficiency and that she might benefit from more, on the condition she doesn't have any signs appearing of overdoses.

Q. Your take on this, is it, is that she's not getting enough thyroxine?

A. Apparently there might be still not enough at that moment.

Q. This is all on the premise that her problem was hypothyroid in the first place?

A. Well, when I look here, energy and libido could be

caused -- if they are better but not still perfect, that could be caused by other things. But "hair less dry" points more in the direction of thyroid deficiency. "Memory still poor" could be caused by lack of female hormones. "Less swelling of feet", so swelling of feet is more linked to low thyroid function especially if it's in the morning. "Tongue enlarged" is more a sign of thyroid deficiency and "indented tongue" is a sign of another problem, not enough water, and things like this. But pulse 56 per minute is almost typical.

Q. But you're completely ignoring the blood test, aren't you?

A. No, I explained to you that I will re-do a new blood test and I will not accept to have a suppressed TSH at that moment. I would have re-done the blood test.

Q. So you would have done a new blood test, is that right?

A. Yes. First I would have asked to the patient, "Did you take the thyroid medication before or not?"

Q. Right.

A. What I think would be -- seeing the response, I think, yes, I have taken it, so do it again.

Q. Right. Let's just imagine for a moment you get your blood test back and it's right, it is below 0.1 TSH and her T4 is 39. Would you still be dosing her up with more thyroxine?

A. I would look for other causes.
Q. You would look for other causes?
A. Yes, for those symptoms.
Q. Because if those readings are right, this woman is in danger, if not already, of being thyrotoxic?
A. Almost impossible with a pulse of 56 per minute. If this information is correct, almost impossible. And "hair less dry", you get wet hair. You get not the dry skin, you get a wet skin. When you touch the skin of a patient who is slightly overdosed it's already a little wet all the time because they perspire too much.
Q. Would the palpitations have caused you any concern at all?
A. Yes, of course, if it's true that it's palpitations. It seems to be that yourself you say it was free of palpitations. If the palpitations would be the first sign, I would reduce the dose.
Q. With the blood tests that you had, we can see at page 8 of tab 4, and the mention of palpitations, you wouldn't have dreamt, I suggest, of adding T3 to this pot, would you?
A. I would have asked for T3 also, so I would have had more information with this blood test. We first have re-done the blood test with T3 and T4 and the TSH before

changing the dose. But this is only in the hypothesis, which I'm almost sure that it's not the correct hypothesis --

Q. No.

A. -- that she did her blood test in adequate conditions. I'm almost sure it's not true because you wouldn't have such a clinical symptom. And I never saw it, when they have such a fellow(?) they never have this, like, low pulse rate unless they have taken high medications.

Q. Can I ask you the question again. With that blood test, the patient had been under treatment for nine months with thyroxine. With that blood test of less than 0.1, T4 of 39, palpitations, you would not have dreamt of giving this woman Tertroxin, would you, without another blood test?

A. And I would have lowered the dose in meanwhile.

Q. Sorry, what's the answer?

A. I would not have increased the dose, I would have lowered the dose and done another blood test if there were palpitations.

Q. Is the answer -- I'm sorry to push you on this -- "I would not have given her Tertroxin without a further blood test"?

A. You cannot stop suddenly a thyroid medication.

Q. I'll ask you once more. Do you accept that you would

not have given this woman Tertroxin, T3, without a further blood test?

A. Yes.

Q. Thank you.

A. But again, I repeat, it's well specified in the hypothesis, the test was done in appropriate conditions.

Q. And if it wasn't done in appropriate conditions, you would demand a further blood test?

A. Yes.

Q. You wouldn't just crack on regardless, would you?

A. Not regardless, but you do have to take into account that in certain situations -- you know, every situation is different. If the person is suffering deeply from hypothyroidism, there's no palpitations, I might have switched over in the meanwhile because I was almost sure that the pathological blood test was -- abnormal blood test was due to the wrong condition. So it's not black and white. But generally it is in the sense that you suggest that I would not have changed medication before having more information if she had palpitations and if the blood test was done in appropriate conditions.

Q. Let's go to the third consultation with Dr Skinner on 18th March 2004. Do you see that towards the bottom of that typewritten page?

A. Yes.

Q. She had now been on Tertroxin for a couple of months and again there's a reference to three-quarters of an hour beating heart. I think that must mean a thudding heart or heart palpitations. What would you have done? She wouldn't be in this position in the first place, would she, because you wouldn't have given her Tertroxin? But what would you have done in March?

A. Three-quarters of an hour heart beating during alcohol, I heard that she had such an episode a long time before also. When you have too much thyroid hormone your heart is pounding all the time: at night, during the day, each moment you sit, each moment you lie down you hear it harder. So it's a very clear symptom. It's not three-quarters of an hour, that's not a symptom of overdosage of thyroxine hormones. It's tachycardia all the time.

Q. So are you saying, effectively, you would have dismissed this three-quarters of an hour thudding heart as something quite irrelevant to the T4 and the T3 you were giving her?

A. If it happens once and I knew that it happened before I still would investigate further what would be the cause.

Q. Right.

A. Too much coffee intake, alcohol intake, others.

If I have a satisfying answer, I won't find -- search later, but I will search until I have the answer.

Q. Would you have been curious as to what her TSH and her T4 and her T3 were doing?

A. All the time. Each time they come at a consultation, yes.

Q. And it's easy to find out because you do a blood test, don't you?

A. Yes, and in a couple of days you have the answer so I would do it before I see the patient.

Q. Without doing a blood test, you wouldn't be writing off to anybody, I suggest, suggesting that this patient was still hypothyroid?

A. Well, what I see on the next page is pulse of 52 per minute.

Q. Yes.

A. I don't have much more information. I do have information that she's feeling better and stopped antidepressants but can be emotional and shaky, having hassle from Job Centre. In people in chronic stress situations I do watch out with increasing the dose. But still 52 per minute is very, very slow; it's almost pathognomonic of low thyroid function. This is the major hormone that determines heart rate. That's why we have to be careful with it.

Q. Low diet function. Is that right?
A. What?
Q. What did you just say, "low diet function"?
A. Low thyroid function.
Q. I'm sorry.
A. 52 per minute. And don't forget that thyroid hormones improve heart arrhythmia. There are references on that. The typical symptom, when you have a heart rate problem caused by low thyroid function, is irregular heart, extra systoles, and that is improved with thyroid hormone. So if the palpitations were slow palpitations, it means not a quick rhythm, it's typical for low thyroid function.
Q. But given that your last TSH was below 0.1, again this is the second consultation, you have seen her since that date, you would not, I suggest, have given her more thyroxine or Tertroxin without blood --
A. But all these things are theoretical.
Q. Is that right?
A. It's so clear for me that the chance is very high that it's caused by taking the thyroid hormones before. I would really ask for another blood test. If it happens twice like this and the patient really says to me, "This time I did it correctly", I would watch out.
Q. You would have taken blood, wouldn't you?

A. I would, yes.
Q. Right. Before allowing this patient to continue with that medication, you would want to find out what was happening, wouldn't you?
A. If I'm sure, yes, I would. I would say, "Wait for the blood test", it doesn't take much time to have results, and then we decide.
Q. In fact, July of 2004, she gets another prescription for 150 micrograms of thyroxine for three months. We have that at page 28 of the bundle. Again, no blood test and on it went. I ought to ask you about that. Can I take it from your previous answers you wouldn't have been giving her that prescription either, would you, without a further blood test?
A. I'm looking at the dates of the blood test. So what I only have here is a blood test in September and of November, after the one in December.
Q. Yes, exactly.
A. It's difficult to tell if it's accurate or not because I do have patients I see every nine months, they have one blood test every nine months. So it's a little difficult, but I would have done a test. If I'm completely sure that the patient had taken the thyroid hormones before the blood test and that falsified, and she's clinically clearly not overdosed, has no

palpitations, I might have not asked again the test.
Q. But if you --
A. I have not golden rules, but generally I try to be quite strict. I don't have so much the problems because most of the patients know very well what to do and we give a paper with the information on on how to do the blood test.
Q. I don't want you to be here forever, as it were, but the question was: in July 2004 you wouldn't have given this patient more thyroxine without a blood test, would you?
A. In a consultation?
Q. In fact there was no consultation, it was just a prescription, I think. Page 28.
A. It's difficult to tell because I probably would have had her on the phone if there was no consultation and if she really marks that she had a lot of -- I would ask all the list of symptoms of low thyroid function. I would have increased the dose, it could have been possible. But of course the patient is well-informed to the signs she has, she has a too high dose and is informed to telephone back two or three weeks later -- so there's a safety zone. I cannot answer you 100 per cent, but what I can say to you is that generally it will be done that the safety is guaranteed of the patient.
Q. Dr Hertoghe, are you really acting as an independent

expert here?

A. I am. I'm talking about if I was the person. What you want to have as information is: is there black and white? And I'm explaining that in consultation we have to adapt the treatment to the patient. For some patients it is a stress to have a blood test and there's so many signs at clinical examination, or, when we interview the patient, that there's hypothyroidism, that a slight increase in dose may be acceptable.

And this is the thing I do, so if you ask my advice, I don't care about Dr Skinner, I just saw him twice, once at the congress, and here. What I care about is good medicine and I'm explaining to you that good medicine is not the dogmatic medicine, it's the medicine adapted to the patient.

Q. Good medicine would have required you to find out what was happening with this patient's blood and not continue his prescriptions time and time again? That would have been good medicine.

A. Certainly not with blood tests. You do have to have a blood test one(?) time, I'm sure about that.

Q. Can we just look at what happens when this patient comes off thyroxine. We know she was reducing it in August of 2004 and that she was off thyroxine, we're told, on 14th October 2004. Yes?

A. Yes.
Q. If you just accept that from me, I can give you the page reference if you want.
A. I think it's correct, yes.
Q. By 11th November 2004 her TSH is 2.4 and her T4 was 13.5. By 9th February 2005 her TSH was 2.8 and her T4 was 16.6. So she was back to the levels, very close to the levels, I suggest, which she had been at before she ever took thyroxine two years before, two and a half years before?
A. Yes.
Q. Does that tell you anything about whether there was anything wrong with this woman's thyroid?
A. But back to the same levels, not necessarily better. You said yourself 2.8, which is still in this grey zone where some people with complaints should be treated, others that do not have complaints should not be treated.
Q. Let me move to patient C.
THE CHAIRMAN: Mr Kark, just to say that the Panel Secretary would like to stop at 4.45 so that she can try the video link.
MR KARK: Right.
THE CHAIRMAN: Sorry.
MR KARK: I was hoping to finish, but it doesn't look as

though I will, I'm afraid.

THE CHAIRMAN: Press on and you might.

MR KARK: Turn to Patient C, please. Obviously we need to turn up the right tab, which is tab 6. She had her first consultation with Dr Skinner on 6th March 2004.

A. Which page is that?

Q. It's right at the beginning behind tab 6.

A. Okay.

Q. So just cast your mind over that. First consultation with Dr Skinner, we don't know from the notes what the prescription was. We know that he took a blood test and he started this lady on apparently a low dose of thyroxine. We know that her TSH, as reflected by that blood test, was 2.2, and her T4 was 11.6. TSH 2.2.

Again, same question, I'm afraid. No endocrinologist, I suggest, in this country, in America, or in your country, is likely to have treated this woman with thyroxine?

A. Almost, no.

Q. Second consultation with Dr Skinner. She was by then taking thyroxine and Tertroxin. I'm sorry, she was taking thyroxine and then there was a prescription for Tertroxin. We can see that that consultation was 8th May. The patient had put her own dose up to 200 micrograms. She was feeling slightly better but

marginally tired. Is that the maximum dose according to the BNF, 200?

A. Don't you have that in your book there? The doses that the BNF stated were between 100 and 200 as the regular dose, but I don't think there's a maximum.

For instance, Professor Heineman was one of the great thyroidologists, Professor Heineman from the Netherlands takes himself 300 micrograms. He's one of the big authorities.

Q. Yes, 100 to 200 daily, normal dose according to the BNF. We know that she was then put on to 150 micrograms of thyroxine and 20 of Tertroxin. That happened in May. There was no blood test at that time, but there had been a blood test back in March 2004. The third consultation with Dr Skinner was on 7th August and her prescription was repeated at 150 micrograms and 20 micrograms plus B12, and blood seems to have been taken.

The TSH, as reflected by that blood test in August of 2004, and we have the lab report at page 10 if anyone wants to have a look at it, was down to -- well, it was unrecordable. Less than 0.1, we heard, meant unrecordable. Her T4 was well over the reference range at 25.5. Her T3 was 8.9. How would you describe those levels if they're accurate?

A. Hyperthyroidism.

Q. Hyperthyroidism?
A. Excess of thyroid hormones.
Q. You are presuming, for the purposes of your report, that this was another bad blood test?
A. Based on the fact that there was no thyrotoxicity, if the report of Dr Skinner is correct and based on the physical signs of 64 per minute of blood pulse.
You don't have that with these lab tests. I don't see that, when I have patients like that and they have done adequately the blood test, I don't have this sort of hypo or euthyroid situation.
Q. But presuming for a moment that Dr Skinner has some experience in taking blood tests with patients of his who are on thyroxine, and this was one of his blood tests so he ought perhaps at least to be an expert in this area, you would expect this blood test to be accurate, wouldn't you?
A. If it is, yes.
Q. If it's accurate, you described it --
A. I don't have this information and I doubt that Dr Skinner has proposed to his patients, and it's really serious what I say, it's really honest.
Q. You would regard this blood test as revealing this patient to be hyperthyroid?
A. If it was in accurate conditions this would be

hyperthyroid because I'm almost sure that we would see clinical signs of excess thyroid hormones also. Both hormones are higher and the TSH is suppressed, so this is hyperthyroidism.

Q. Would you warn the patient of what was happening, given that this was a patient who had in the past put her own dose up?

A. Yes.

Q. Would you make it --

A. I would say you have to stop for one or two days and then go back at a lower dose. That would be my reaction in the hypothesis, because it's not confirmed by the clinical situation, that the blood test was done accurately.

Q. But you would want to make it clear to the patient, wouldn't you, that from the blood test she was hyperthyroid and it would appear she may be taking too much?

A. Yes.

Q. And that she ought to cut down?

A. Yes.

Q. You wouldn't write to that patient, "Here are your request thyroid chemistry and cortisol, which indicate that the levels are a little on the high side, but if you're not feeling any adverse effects then you should

stay on the same dose until I see you next"?

A. No, I wouldn't write it with this, but the fact that the symptoms were too low, I would probably have phoned the patient at that moment and asked the patient if she was not feeling symptoms of overdose. I would go more to roughly around the list before writing that.

Q. There was another blood test. You see, you've rather presumed for the purposes of your report that the August blood test taken by Dr Skinner was duff, as it were. You understand what I mean? It wasn't good because she had just recently taken a pill. But there was another blood test in September, 24th September 2004, and that TSH -- this is in the patient notes, tab 5, page 1, this TSH, I've recorded it as 0.01. Let me just check.

A. 0.01, no? I think it is so,.

Q. 24th September 2004. Yes, 0.01, thank you. That again is just negligible, isn't it?

A. So what I said here is that there's still a hypothesis here that she did the blood test in adequate conditions because you see that the T4 is lower, but the first choice would still be she took it before the blood test.

Q. So that is your first choice. So August, the blood test is wrong, September the blood test is wrong. But actually they went and did another blood test in October and we have that at 5/1A. The TSH was still 0.01

so August and September and October, you got a negligible TSH reading, for each of those --

A. It's unacceptable to have three times in a row a low TSH. But what I feel here, when I listen to the endocrinologist and I see the reports of Dr Skinner and see what the GPs do, they never think about that. I never heard it mentioned. It's basic guideline not to take the thyroid hormones before the blood test so I presume they just take the blood test when the patient comes by, whatever the patient has done, but they don't pay attention to that.

Q. Is that on the basis that, like the endocrinologists in this country you don't think very much of, you don't think very much of the GPs either?

A. I would say when I hear that and that nobody pays attention, I think there's a little problem at least in the (inaudible word).

Q. And you're saying --

A. Because in my experience when a patient is hypothyroid or euthyroid and she has this sort of list, it's almost always that she took the blood test before -- the thyroid hormones before medication.

Q. So you are prepared to say that in relation to the August blood tests, the September blood tests, and the October blood tests, we can set them to one side

because they all must have been taken wrong?
A. I cannot evaluate correctly based on these lab tests.
Q. Shall we imagine for a moment that they're right, that they actually do reflect the values.
A. If they're right, the first time it is overdosed, the second time it depends, and the third time you said it was also 0.01. Did you have a thyroid hormone or not?
Q. 5/1A, and the T4 is 15. Now, this is after a note that the patient had decided to stop treatment, so let me just draw your attention to this. Tab 5, page 1A, this is 15th October 2004. So does that look to you, given your experience, that the patient actually has stopped taking her T4, so her T4 has come down but the pituitary hasn't quite yet started producing the right amount of TSH?
A. My impression is that she was still taking the medication.
Q. Why has her T4 come down so much?
A. Well, this sort of blood test has two possibilities: either she took the thyroid medication before, then she has a suppressing of TSH. Either she didn't take it before.
Q. Then why is the TSH still suppressed?
A. So there's a small little fraction, like I said, of my patients that have with treatment -- are clinically

normal and have a suppressed TSH because their pituitary gland fails, not only the thyroid gland fails. So when they get the thyroid hormones, they have -- but it's a small fraction, it's not all the patients.

Q. I think we can exclude this patient because we know her TSH in March 2004 was 2.2 and her TSH, in fact the following TSH in February 2005 had recovered to 2.4.

A. Yes.

Q. So she's not going to be one of those that her pituitary is so sick she's not producing TSH, is she? So what's the alternative?

A. There are certain patients that, when you give thyroid treatment, undergo a greater decline than others. That's why, when we see a study, it's at average. But there are people who, with the same dose of thyroxine, will respond much more or much easier with the suppression; others that will even have just a slight inhibition, but not very notable. So there are different changes. It's not so easy. So what I'm trying to do is to be here a honest person, to give you honest answers, and I'm showing that medicine is not that easy. You need to take the whole clinical picture in order to be able to analyse it correctly.

Q. If these blood tests are right, this woman had been overdosed, she had become hyperthyroid, hadn't she?

A. Yes.
Q. And when she comes off it, her TSH recovers?
A. But without clinical hyperthyroid symptoms, it's very unlikely. But if that was right, she was overdosed, but in my patients I always see signs. I don't know why English patients would be different.
MR KARK: I'm about to turn to Patient D. I think that is a convenient time.
MR JENKINS: Before that happens, I wonder if Mr Kark is going to ask any questions about Armour that this patient said she was taking at the end of August and that the GP record on page 1, tab 5, suggests she was on.
MR KARK: Well, not at the moment. I might ask that on Monday. But if that is a convenient moment, I don't want to rush through just to get it finished.
THE CHAIRMAN: Thank you, Mr Kark. Thank you, Dr Hertoghe. I think this is an appropriate moment to stop. I would just like to make clear that I'm uneasy about the video link and I think it would be preferable if you were here if that was possible. I do understand the difficulties so I shall leave it in your hands. We will meet here at 9.30 on Monday morning and will be in touch one way or the other.
MR KARK: I was just wondering, given the problems with

the video link, if it would be sensible to make it 10.00 to give everybody an opportunity to test it on Monday morning.

THE CHAIRMAN: You are right, 10 o'clock on Monday morning.

A. It might be a little problem for me to be here on Monday morning, maybe not. Is it possible eventually to come a later day, the Tuesday or the Wednesday, or not?

THE CHAIRMAN: I think, if you didn't come and do the video link, and if that doesn't work, then we will obviously have to do other arrangements anyway, so that would be Tuesday or Wednesday.

A. Okay.

MR JENKINS: We are not due to be here on Wednesday.

THE CHAIRMAN: It will be Tuesday. We will make every effort to ensure the video link does work. We are likely to know very soon whether it's likely to work or not.

MR JENKINS: Can we have permission to speak to Dr Hertoghe about travel arrangements and other arrangements to ensure that you're able to see him on Monday?

THE CHAIRMAN: Dr Hertoghe, you know you're still under oath. Yes, of course one must give you leave to discuss the travel arrangements. Thank you.

(4.45 pm)

(The hearing adjourned until 10.00 am

on Monday 16th July 2007)