Medicolegal

Dr Gee's cross examination continues

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The trial of Dr Sidney Gee's libel action against the BBC over allegations about his slimming treatment broadcast on *That's Life* adjourned for the Christmas break on 20 December, Dr Gee's 28th day in the witness box. In a lengthy cross examination about Dr Gee's use of thyroid extract in treating obesity Andrew Rankin QC, leading counsel for the BBC, suggested that to give a euthyroid patient just one of Dr Gee's thyroid tablets a day would result in virtual suppression of the thyroid gland over a period of time. This would cause no harm because the gland would remain suppressed and the patient would receive sufficient thyroid hormone from the tablet. But given two tablets, patients would become hyperthyroid within months.

In reply Dr Gee referred to a 1968 article that showed that thyroxine was poorly absorbed. Thyroid extract, he said, had an even worse absorption rate—only 30-40%. Mr Rankin countered that the replacement dose of 300 µg for patients without any thyroid function mentioned in the article was now outdated; 150 µg was now considered to be the correct dose. Dr Gee replied that at the absorption rate specified in the article 150 µg would mean only about 60 µg available to the body. He did not accept Mr Rankin's suggestion that the replacement dose already took into account the rate of absorption.

Counsel quoted from an article on the administration of thyroid hormone to obese patients: "Four out of 21 subjects had to discontinue thyroid because of ill effects. The subjects developed angina on effort in the absence of a prior history of coronary disease." Though he accepted that the dosage of thyroid hormone was 480 mg a day (230 mg a day more than Dr Gee's dose), the article showed that giving thyroid hormone in a particular dose could produce angina.

He quoted further: "Cardiac arrhythmias have been reported during weight loss. Under such circumstances a subject may well be more vulnerable to the sympathomimetic effect of thyroid and develop a variety of cardiac abnormalities." Dr Gee replied that the adverse circumstances in these cases were due to a combination of large doses and potassium depletion. The patients were receiving not thyroid extract but a synthetic mixture, and the daily doses were 360-840 mg. The paper, he pointed out, went on: "With four exceptions, these subjects seemed to tolerate the large amounts of thyroid given without developing the usual manifestations of toxicity. This experience parallels the observations of others who described only minor ill effects in obese subjects or in mildly overweight, healthy volunteers to whom thyroid in equivalent as well as larger doses was given."

Patients' histories

Mr Rankin referred to articles on thyrotoxicosis and heart disease, and one on death from thyrotoxicosis. He suggested that

two of Dr Gee's patients, Mrs Falconer (who died in 1978 from coronary thrombosis at the age of 34) and Mrs Day, had been identified as hyperthyroid. Dr Gee insisted that their readings were completely within normal limits, given the circumstances and the fact that they were receiving exogenous thyroid hormones.

Counsel suggested that a patient like Mrs Day, with a healthy thyroid gland, given 250 mg of thyroid extract a day for three months would have her thyroid gland's activity virtually suppressed. Dr Gee replied that he would expect some degree of suppression but because of individual variation he could only guess how much. It was apparent from such research as had been done, however, that there was no harmful consequence because the gland recovered and kept some of its spontaneous activity.

Asked whether it had occurred to him that if the gland recovered and started secreting hormones he would be producing a hyperthyroid state when he administered further hormones, he replied that it was impossible. Any excess of thyroxine remained in the blood and was converted into reverse triiodothyronine, not active triiodothyronine. Any increase in triiodothyronine was transient, lasting only a few hours. So unless overwhelming doses were given the metabolic consequences were not in any way harmful.

Mr Rankin moved on to question Dr Gee about the postmortem report on Mrs Falconer. Counsel suggested that the reference to "mild right and left ventricular hypertrophy" was suggestive of thyrotoxicosis. Dr Gee said it would be suggestive of very long-standing severe thyrotoxicosis. But a patient with thyrotoxicosis sufficient to produce left ventricular hypertrophy would have an increased pulse pressure.

Questioning Dr Gee about another patient, Mrs Saunders, who became pregnant and whose doctor wrote to Dr Gee inquiring about the drugs she had been given, counsel reminded him of his reply: "The tablets have no effect whatsoever on implantation or the fetus." Counsel quoted from *Drugs and Pregnancy* by Hawkins: "It seems possible that there may be a teratogenic effect associated with the use of amphetamines in early pregnancy and their use as appetite suppressants should be discouraged." Dr Gee replied that one of the authorities cited referred to biliary atresia, but the biliary ducts did not develop until much later in pregnancy than the stage Mrs Saunders would have reached. Of the two articles on congenital heart disease cited, one suggested that it might be associated with high doses of amphetamine, but the other that it was not.

Mr Rankin cited an article that reported an excess of oral clefts in the offspring of mothers prescribed amphetamine in the first 56 days from the last menstrual period. Dr Gee replied that there was no evidence that taking it in very early pregnancy would lead to abnormality of the palate, whose development took place later. Under his treatment the longest period for which a patient could inadvertently take amphetamine would be two weeks before she missed a period. Mrs Saunders's last period had been on 26 February, and therefore she had become pregnant about 10 March. Twenty one tablets had been sent to her by post on 15 March, but it would be unjustified to assume that she had kept taking them once she had missed a period at the end of March. The mothers of the babies born with oral clefts had first been exposed to amphetamines on days 43 and 50. In Mrs Saunders's case day 43 would have been 10 April. Dr Gee emphasised that he did not give his treatment to

women known to be pregnant. He said that the average woman knew that if she became pregnant she should not take drugs.

Referring to the stillbirth of Mrs Downs's baby (15 December 1984, p 1683), Dr Gee asked whether the defendants were contending that the stillbirth was a result of his treatment. Mr Rankin made it clear that this was not part of his clients' case.

During Mr Rankin's absence with flu Dr Gee was cross examined by Charles Gray QC, leading counsel for Dr Blackwood and Dr Mitchell, the two doctors Dr Gee is suing for libel over their part in the *That's Life* broadcast. Mr Gray asked Dr Gee if, when Mrs Day consulted him, he had found out that she had been diagnosed as prediabetic and had been treated with oral hypoglycaemics. Dr Gee replied that a glucose tolerance test carried out in 1969 in the eighth month of pregnancy had shown a level of 115 mg/100 ml, which was not diabetic or prediabetic. Mrs Day, he added, had been put on phenformin not as an oral hypoglycaemic but to control her weight.

Counsel suggested that Mrs Day had become thyrotoxic on Dr Gee's treatment. He put it to Dr Gee that her pulse rates showed a declining curve from around 28 April, just before she stopped taking thyroid extract. Dr Gee pointed out that her pulse rate had gone up again on 10 June. Mr Gray suggested that the symptoms Mrs Day had complained of to Dr Blackwood were symptoms of thyrotoxicosis. Dr Gee admitted that some of them could be symptoms of thyrotoxicosis but said that they could all have a cause other than thyrotoxicosis. Most of the symptoms she allegedly complained of—for instance, palpitations, heat intolerance, and irritability—could be caused by nervous tension or an anxiety state. Hair dropping out was a symptom of hypothyroidism, and shortness of breath was not a symptom of thyrotoxicosis except where it was sufficiently long term to produce heart damage.

Dr Gee agreed that severe and prolonged thyrotoxicosis led to increased force and output of the heart and excitation of the cardiovascular system, and that the extra demand on the heart could lead to the growth of extra tissue. He also agreed that prolonged hyperthyroidism could give rise to atrial fibrillation. He agreed that with this condition a clot could form on the wall of the atrium which could break off and be expelled through the arterial system and cause death by cutting off the blood supply to the part that was blocked. But Mrs Falconer died from a large coronary thrombosis, which the literature showed was very rare with thyrotoxicosis.

Results of laboratory tests

Resuming his cross examination, Mr Rankin quoted from a letter sent to Mrs Falconer's husband by the hospital after her death: "Features of her medical history and examinations suggested thyroid overactivity, and as you are aware she had for some time been taking tablets for slimming. In fact some of these were thyroid tablets." Dr Gee insisted that Mrs Falconer's thyroid tests had shown no evidence of any thyroid abnormality. Mr Rankin pointed out that the free thyroxine index was 200, outside the normal laboratory range. Dr Gee said the free thyroxine index was an outmoded method of calculation that was no longer used. The tri-iodothyronine referred to in the laboratory result was not a test of thyroid function, or was so only indirectly. The blood test had shown that there was no thyroid overactivity. Dr Gee referred to an article that he said showed that the free thyroxine index could be grossly raised in women, like Mrs Falconer, who were taking the pill.

Re-examined by his own leading counsel, Michael Beloff QC, Dr Gee said that Mrs Day's 1969 glucose tolerance test had shown that she was not diabetic. Looking at her hospital notes, it appeared that in 1970, when phenformin was prescribed, a random blood sugar test had shown a level of 70 mg/100 ml.

At the time phenformin was used mainly for weight reduction in obese non-diabetics. If it was being used as an oral hypoglycaemic it would have had to be continued, and there was nothing in Mrs Day's notes to suggest that she received phenformin over the years 1971 to 1975. Dr Gee said that the reading of 115 mg/100 ml at one and a half hours on Mrs Day's glucose tolerance test in 1969 would have been higher than 160 mg/100 ml if she had been diabetic. He

added that all Mrs Day's symptoms could have a cause other than thyrotoxicosis. The only diagnosis that fitted all the symptoms was an anxiety state.

He cited a paper from *Acta Endocrinologica* that gave a mean free thyroxine level of 53 ± 8.6 and a mean thyroxine level of 288 ± 53 for thyrotoxic patients. He pointed out that Mrs Day's corresponding values were 28 and 156 respectively, and she had been having exogenous thyroid. He also cited articles showing that antibodies were apt to develop with the administration of thyroid extract, which could increase the free thyroxine levels, and that false positive increases in the free thyroxine index occurred commonly in psychiatric patients.

Looking at the graph of Mrs Day's pulse rates, which had been shown to him by Mr Gray, and continuing the graph with later recorded pulse rates up to January 1984, Dr Gee said they showed a labile pulse rate subject to variation without any obvious cause, and therefore the variation must have an emotional basis. It was impossible that even an overdose of thyroid hormones (which his treatment was not) could produce such effects over a year later.

Nor was there any evidence either in Dr Blackwood's notes or in his report back to the general practitioner that there was anything wrong with her heart. The echocardiogram and 24 hour cardiograms were normal. Reading through her notes from November 1983 until August 1984, culminating in Dr Blackwood's comment, "I think Mrs Day is slipping into quite a marked psychiatric state. She seems to have feelings of claustrophobia, depression, anxiety and probably agoraphobia," Dr Gee concluded that Mrs Day was a psychiatric case.

Mr Beloff said that the BBC was alleging that Dr Gee's treatment caused the death of Mrs White (on 4 July 1983). Dr Gee replied that he did not accept that in any measure. He reiterated his evidence that her last visit had been on 30 June. He had a cheque signed by her and dated that date, which was for the consultation and treatment. Asked to what he could attribute the intermittent palpitations of which Mrs White had complained to her general practitioner, Dr Gee replied that they could have been due to a disturbance in the conducting mechanism of the heart, a pre-excitability. This could be caused by smoking, stress or alcohol.

The trial was scheduled to resume on 14 January.

This article follows on from the one published on 15 December 1984 (p 1683).

Correction

Research in general practice: pursuit of knowledge or defence of wisdom?

We regret that an error occurred in this article by Professor J G R Howie (22-29 December, p 1770). The lecture on which Professor Howie's paper was based was wrongly described. The footnote should have read: "Based on the sixth Wedgwood lecture delivered at the North Staffordshire Medical Institute, Stoke on Trent, in March 1984."

The symptoms of a 66 year old man with lifelong narcolepsy of moderate severity with hypnagogic hallucinations and cataplexy are largely controlled with 12.5 mg of dexamphetamine daily. Recently he added clomipramine 10 mg daily to the existing medication, and this has helped. It would have been of more help if he could take 20 mg daily but clomipramine, even at the dose of 10 mg daily, causes erectile failure. Is there any other preparation with the same effect as clomipramine that is less likely to cause erectile failure?

Erectile failure is largely a result of the anticholinergic effect of clomipramine, a property shared by the other tertiary amino tricyclic antidepressants such as imipramine, trimipramine, and amitriptyline. Changing to one of these alternative tricyclic drugs would probably be unhelpful. Some of the newer antidepressants such as mianserin, a tetracyclic compound, have little or no anticholinergic activity and may be less likely to cause ejaculatory failure. On the other hand, the evidence that mianserin is effective in narcolepsy is less convincing than for the tricyclic drugs, but it would be worth trying, starting with a single night time dose of 30 mg.—A RICHENS, professor of pharmacology, Cardiff.