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BRITISH MEDICAL JOURNAL

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Because we receive many more letters than we have room to publish we may shorten those that we do publish to allow readers as wide a selection as possible. In particular, when we receive several letters on the same topic we reserve the right to abridge individual letters. Our usual policy is to reserve our correspondence columns for letters commenting on issues discussed recently (within six weeks) in the BMJ.

Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue. We may also forward letters that we decide not to publish to the authors of the paper on which they comment.

Letters should not exceed 400 words and should be typed double spaced and signed by all authors, who should include their main degree.

Are biochemical tests of thyroid function of any value in monitoring patients receiving thyroxine treatment?

SIR—The authors of your For Debate article (27 September, p 808) have so badly misunderstood the purpose of monitoring these patients that we suspect this could be a deliberate ploy to provoke “debate.” If we have fallen into the trap then so be it.

The dose of thyroxine required by most adult hypothyroid patients to render them euthyroid is known—2·2 µg/kg body weight.¹ The major reason for requesting thyroid function tests in these patients, not mentioned by the authors, is to check compliance.² Failure to comply, as with any self administered therapy, is common. Their assumption that the clinical assessment is correct is flawed because thyroid function tests typically move ahead of the clinical findings,³ allowing the laboratory to detect the non-complier so that early advice can be given before the patient becomes symptomatic—surely a valuable role? Also, thyroid dysfunction may be very difficult to detect clinically. For example, babies with congenital hypothyroidism detected by screening programmes are often without clinical signs, pregnancy and hyperthyroidism have many similar features, and masked thyroid disease in the elderly is well recognised. In these patients the sequelae of a misjudgment or non-compliance with thyroxine therapy are particularly dire, and judgment of its adequacy is usually possible only on the basis of results of thyroid function tests.

The design of this study can also be criticised as the time of sample collection was controlled and probably close to the peak concentration of thyroid hormones that occurs after a dose of thyroxine³ (assuming their patients took a single morning dose, a detail not mentioned). Their results are therefore likely to be peak thyroid function test values. “Reference ranges” for patients on replacement need qualification because the

intraindividual variations of circulating thyroid hormones in euthyroid individuals are wide,⁴ but the intraindividual variation seen in their patients on thyroxine was not investigated; thus how their results translate to real life, where sample collection and dosage times are uncontrolled, is not clear. In our view adequacy of replacement and compliance is best judged by cumulative results on an individual by recognising the level of free (or total) thyroxine that normalises free (or total) triiodothyronine and keeps thyroid stimulating hormone values within the normal range or below.⁵ Once the dose bringing this about has been identified monitoring by a thyroxine estimation only is usually adequate.

In the present state of knowledge the real area for academic debate is the long term effect on the heart⁶ and skeleton.⁷ Thyroid function tests are the best tools available for monitoring patients with hypothyroidism, and so they should be used.

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5 Mardell RJ, Gamlen TR, Winton MRJ. High sensitivity assay of thyroid stimulating hormone in patients receiving thyroxine for primary hypothyroidism and thyroid carcinoma. *Br Med J* 1985;290:355-6.

6 Jennings PE, O'Malley BP, Griffin KE, Northover B, Rosenthal FD. Relevance of increased serum thyroxine concentrations associated with normal serum triiodothyronine values in hypothyroid patients receiving thyroxine: a case for “tissue thyrotoxicosis.” *Br Med J* 1984;289:1645-7.

7 Perry HM. Thyroid replacement and osteoporosis. *Arch Intern Med* 1986;148:41.

SIR—It is unfortunate that Dr W D Fraser and his colleagues have overstated their case that thyroid function tests are of little, if any, value in monitoring patients receiving thyroxine replacement therapy.

By showing a pronounced discrepancy between clinical assessment based on the Wayne index (which was not intended for patients on thyroxine therapy) and the results of thyroid function tests, they conclude that the tests must be at fault. As an analogy to this flawed logic, imagine if diabetes mellitus were still diagnosed by tasting the urine for sweetness and that this procedure was used as the gold standard against which a new test, plasma glucose concentrations, was judged.

More seriously, we would accept that it may be difficult to know the importance to attach to abnormal thyroid function test results in patients taking thyroxine. For example, not all would accept our view that undetectable serum thyroid stimulating hormone measured by sensitive assay is evidence of overtreatment.¹ Equally, few would disagree that a raised thyroid stimulating hormone value is a sign of undertreatment or poor compliance which would not necessarily be detected clinically.

The place of thyroid function tests is to supplement clinical judgment, and, although they do not carry the same diagnostic weight in patients taking