***Diagnosis and treatment of primary hypothyroidism***

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**Lack of diagnosis & incorrect treatment**

The RCP/BTA guidelines for The Diagnosis and Management of Primary Hypothyroidism are interesting as in the 2006 BTA guidelines (1)on page 68, it states, "Routine thyroid function testing has been available for more than thirty years. Therefore, it may seem surprising that the quality of evidence to support the recommendations in these guidelines is generally poor, etc."

How are the general public to have confidence in the diagnosis and treatment of hypothyroidism if these 'gold standard' tests actually aren't all they are made out to be?

At the 75th Annual Meeting of the American Thyroid Association, Normal levels of TSH were quoted as 0.5-2.5, high normal as 2.5-5, mildly elevated 5-10 and clearly elevated as >10. Why do patients have to suffer for years or even decades in this country for their TSH to be elevated above 10 when they may (if they are lucky) finally receive treatment?

Of course, once the diagnosis is made the main treatment offered is Thyroxine which definately does not suit us all and then once back within the reference range, medication is often kept just within the reference range even though the patient could still be suffereing severe hypothyroid symptoms. What happened to evidence based medicine?

Dr A Toft (2)has stated that "In some patients, a sense of well-being is achieved only when FT4 or TT4 is raised, for example, 30pmol/l or 170nmol, and TSH low or undetectable."

Dr. Toft (3)talking about the addition of T3 to a patient's medication stated that he believed that "It would appear that the treatment of hypothyroidism is about to come full circle."

In 2004 Colin Dayan,(4) Endocrinologist and Consultant Senior Lecturer at the University of Bristol, stated "This house believes that thyroxine is not an adequate form of thyroid hormone replacement in everyone...",and, "Estimated that 5% are psychologically dissatisfied despite TSH levels in the reference range and that variations in 3 deoidinase anzymes, or cell membrane thyroid transporters could be factors.

Finally, Prof A. P. Weetman (5) stated in 1997 in your own journal that "A high thyroid stimulating hormone concentration >2m/Ul was associated with an increased risk of future hypothyroidism", but more importantly he stated, "The simplest explanation is that thyroid disease is so common that many people predisposed to thyroid failure are included in a laboratory's reference population, which raises the question whether thyroxine replacement is adequate in patients with thyroid stimulating hormone levels >2mU/l."

This all sounds very conflicting evidence to the layman. It is about time that evidence based medicine was even handed on not just on the side of the few. Isn't science meant to produce both sides of the argument? It didn't appear so in the document, 'The Diagnosis and Management of Primary Hypothyroidism'.

(1) UK Guidelines for the Use of Thyroid Function Tests (July, 2006). The Association of Clinical Biochemistry, The british Thyroid Association and The British Thyroid Foundation.

(2) Dr. A Toft, CBE, MD, FRCP (Consultant Physician and Endocrinologist at the Royal Infirmary, Edinburth) Toft A Understanding Thyroid Disorders. Family Doctor Publications Limited in assoc with the British Medical Association 2006)

(3) AD Toft, Endocrine Clinic, Royal Infirmary, Edinburgh, UK Endocrine Abstracts 3 S40, T3/T4 combination therapy.

(4) C. M. Dayan Thyroid Hormone Replacement Therapy - This house believes that thyroxine is not an adequate form of thyroid hormone replacement in everyone..." Endocrine Nurses Training Course 9-11Sept 2004 The Society for Endocrinology - Training.

(5) A P Weetman, Frequencey of Hypothyroidism. BMJ 1997:314:1175 (19 April)

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