

DOCUMENT 3**Document of Record concerning UK Guidelines for thyroid function test
October 2005.**

In October 2005 the Association for Clinical Biochemistry, British Thyroid Association, and British Thyroid Foundation kindly prepared the document named as above and invited comment and input through the medium of e-mail with assurance that due consideration would be given to any such advice.

Some eight weeks have elapsed and it is unclear how such input has been integrated or its authorship or indeed when will be the next consultation paper. These are not criticisms but we feel that there is need to enshrine a Document of Record as a future point of reference for both the profession and patients. The latter in particular may find themselves in a difficult position if the Department of Health and the medical profession adopt or even advocate these Guidelines. This is important; there is little doubt that as time goes by Guidelines soon become Rules and the profession will believe – and not without justification in the present environment – that report of non adherence to Guidelines to the General Medical Council can result in adverse outcome or civil litigation. We require clear directives from the General Medical Council and Defence Societies that sensible decision-making outside Guidelines does not engender disciplinary action for the Practitioner.

A second concern is that once Guidelines have transmuted into Rules, the precise authorship of these Guidelines and, importantly, any caveats or arguments against precepts contained within the Guidelines slip into obscurity; it is then assumed that Guidelines represented a majority view of the wise and any deviation from that view bespeaks a certain ‘charlatanry’ or ‘marginality’ in the medical practice of that colleague.

This document focuses specifically on hypothyroidism. This Working Group believes that too many patients have suffered at the hands of a ‘non-evidence-based mantra’ which (ironically) has been paraded as ‘evidence based’ where there is no evidence for example to support the core precept of Guidelines that thyroid chemistry is an independent and reliable marker of illness or health. This is purposefully stated in the simplest terms to avoid any possible confusion on this fundamental misconception which will result in continuing ill health for many patients and will continue so to do if these Guidelines are enshrined in stone for the future diagnosis and management of hypothyroidism.

We respectfully submit that we do not accept the following precepts contained in the Guidelines.

**1.EXCLUSION OF HYPOTHYROIDISM ON THE BASIS OF THYROID
CHEMISTRY**

There is no evidence that free thyroxine or thyroid stimulating hormone levels within 95% reference intervals exclude a diagnosis of hypothyroidism. If there is contrary evidence to this view then it should be presented and further time allowed for analysis

and discussion of such evidence; if there is no such evidence, then this must be unequivocally stated towards redirection of medical practice vis-a-vis management of hypothyroidism.

2.THERAPEUTIC BENEFIT IN RELATION TO DIAGNOSIS BY CLINICAL OR THYROID HORMONE LEVELS.

There is no evidence on a crucial therapeutic issue namely that the outcome of thyroid replacement is better or worse if the diagnostic criterion has been based on clinical features of thyroid chemistry. Pending formal clinical trial, we argue that patients will fare better if diagnosis is based on clinical features.

3.INTERPRETATION OF THYROID HORMONE LEVELS AND REFERENCE INTERVALS.

There is little discussion in the Guidelines concerning possible technical and pharmacological shortfalls in a non critical interpretation of free thyroxine, thyroid stimulating hormone and 95% reference intervals as pivotal criteria in the diagnosis of hypothyroidism; various arguments on these issues have been presented in the following references (1, 2, 3, 4).

4. THYROID HORMONE REPLACEMENT AS MONITORED BY THYROID HORMONE LEVELS.

Levels of thyroid replacement or choice of thyroid preparation should be monitored by clinical considerations rather than thyroid chemistry which is virtually an axiomatic proposition. In the present environment, chronic hypothyroid ill health is too often accepted from an unfounded anxiety over perceived pathogenicity of raised FT4 and/or low TSH levels outwith 95% reference intervals; we have provided evidence in publication that clinical outcome is not related to thyroid chemistry but more closely to thyroid dosage level which was based on clinical evaluation of the patient (!)

5. RELATIVE THERAPEUTIC EFFICACY OF AVAILABLE THYROID PREPARATIONS

There is no evidence teaching advantage of thyroxine versus triiodothyronine versus Armour Thyroid excepting observation of practitioners who have used all three preparations over a number of years. It is thus unreasonable that there is repetitive suggestion from a number of colleagues in the field that Armour Thyroid must prove its mettle when it was actually first at the post by a long way. We ask for a measure of

equability in the evaluation of medicinal products; there is urgent need for a comparative evaluation.

6. LONG TERM HEALTH DETRIMENT FROM ABNORMAL THYROID HORMONE LEVELS

Long term adverse outcome from abnormal thyroid chemistry has been exaggerated from non cognisance of the clinical status of patients in long-term studies of this issue. It is realised of course that patients with long term evidence of thyrotoxicity may well develop pathological sequelae but there is no secure evidence that suppressed TSH in clinically euthyroid patients carries such detriment. If there is such evidence, it should be stated but, if not, we feel the document should make unequivocal statement to the contrary. It is considered highly improbable that continuance of hypothyroidism with its manifest pathological sequelae - including the oft-ignored long term complications of increased cholesterol level and atheromic deposition - is a safe alternative to clinical euthyroidism and optimal health.

CONCLUSION

We submit that the diagnosis of hypothyroidism and evaluation of replacement dosage levels should not be pivotally dependent on thyroid chemistry but on clinical evaluation of the patient with sensible cognisance of thyroid hormone levels as an adjunct if required in patients where there might be dubiety or uncertainty on the evidentiality of clinical features. There is no evidence from clinical trial to support the relative therapeutic benefit of either of the three available thyroid preparations in single or combinative use. There is urgent need to subject unresolved issues as highlighted above in 1 – 6 to the scrutiny of formal clinical trial.

REFERENCES

1. Clinical response to thyroxine sodium in clinically hypothyroid but biochemically euthyroid patients'. G.R.B. Skinner MD DSc FRCpath FRCOG., D. Holmes., A. Ahmed PhD., J.A. Davies., BSc and Benitez MSc. Journal of Nutritional & Environmental Medicine (2000) 10, 115 – 124
2. Thyroxine should be tried in clinically hypothyroid but biochemically euthyroid patients. G R B Skinner, R Thomas, M Taylor, M Sellarajah,S Bolt, S Krett, A Wright. Letter to the Editor. BMJ. 14th June 1997.
3. Diagnosis and Management of Hypothyroidism. Gordon R B Skinner. Louise Lorne Publication. Birmingham, UK. 2003.
4. Communication to Dr G H Beastall in response to proposed 'UK Guidelines' 22.12.2005 (Enclosed)

