Benign prostatic hyperplasia

Sir,

I read with interest the article on benign prostatic hyperplasia (BPH) (vol 63(8), 2002, p. 460). While the article served as a helpful overview of BPH management, some of the assertions made are misleading and open to challenge in light of recent evidence.

First, the authors suggest alfuzosin may reduce the incidence of acute urinary retention (AUR), citing a small, 6-month study (Hartung, 2001). It is surprising that the Medical Therapy of Prostatic Symptoms (MTOPS) trial was not mentioned. MTOPS studied 3047 men over an average of 4.5 years, and was a larger and longer trial with AUR as a predefined endpoint. It confirmed that treating BPH with doxazosin and finasteride in combination was more effective that either drug alone in preventing AUR or the need for BPH surgery. Finasteride alone was shown to be significantly better than doxazosin. This trial vindicates the current situation whereby finasteride is the only currently licensed drug for the reduction of AUR risk.

Second, in addressing the question of combination therapy, the authors cite two relatively short studies (the 12-month VA study and the 6-month ALFIN trial) where 'no additional benefit was seen with finasteride plus an alpha blocker compared to alpha blockers alone'. Findings from these trials have been superseded by results from MTOPS. This showed that doxazosin and finasteride act synergistically in combination in reducing symptoms in BPH. The MTOPS findings also showed that finasteride monotherapy was as effective as doxazosin alone in improving symptom score.

Last, the article described the side effects of finasteride (in the text) and of alpha blockers (in *Table 5*). Both drug classes are extremely well tolerated but only alpha blockers are associated with dizziness, headache and hypotension.

In the management of BPH, clinicians have sound therapeutic options, and in most cases, drug therapy for BPH can (and should) be confidently prescribed. For this to happen effectively, we must be armed with the latest and most robust evidence to support our choice.

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Hartung R (2001) Do alpha-blockers prevent the occurrence of acute urinary retention? *Eur Urol* **39**(Suppl 6): 13–18

Thyrotoxicosis-induced hyperferritinaemia

Sir,

A 57-year-old gentleman presented with a 3-month history of weight loss (total 14 kg) and anxiety. There was no significant past medical history or thyroid disorder. Physical examination was normal, except for mild hand tremor and a small diffuse goitre. Thyroid function tests showed free thyroxine = 60.7 pmol/litre (normal range (NR): 10.0–20.0 pmol/litre) and thyroid-stimulating hormone <0.005 mIU/litre (NR: 0.45–4.50 mIU/litre). Serum ferritin was elevated at 993 mg/litre (NR: 20.0–320.0 mg/litre). Serum iron and transferrin were within normal limits. His full blood count and liver enzymes were normal. He was commenced on carbimazole, which rendered him euthyroid within 2 months. His serum ferritin level came down gradually in line with the amelioration of his hyperthyroidism, and normalized over the year following improvement of his thyroid status.

The effect of hyperthyroidism on haematological indices, in particular serum ferritin level, is not widely recognized. Serum ferritin concentration is usually related to the quantity of body iron stores. In iron overload state, e.g. haemochromatosis, serum ferritin level is typically very high (>1000 mg/litre), but serum ferritin level can be elevated out of proportion to iron stores in various clinical disorders.

Serum ferritin levels have been reported to be elevated in patients with hyperthyroidism (Delfino, 1993). Serum ferritin levels decreased after thyroid function returned to normal (Takamatsu et al, 1985). The mechanism of thyroid hormone-induced hyperferritinaemia is not well understood. Studies in rats reported that triiodothyronine (T_3) increased expression of ferritin H mRNA as a consequence of increased transcription rate of ferritin H gene (Iwasa et al, 1990). Leedman et al (1996) suggested that the effect of T_3 on ferritin expression in the liver may be caused by T_3-induced modulation of iron regulatory proteins binding to the ferritin mRNA iron-responsive element.

This case illustrates that the high serum ferritin levels decreased to normal after the patient was rendered euthyroid. With the increasingly frequent use of ferritin in clinical practice, recognition of this phenomenon of hyperthyroidism-induced hyperferritinaemia is vital.

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Delfino M (1993) Serum ferritin in hyperthyroidism. *Ann Intern Med* **119:** 249
Iwasa Y, Aida K, Yokomori N et al (1990) Transcriptional regulation of ferritin heavy chain messenger RNA expression by thyroid hormone. *Biochem Biophys Res Commun* **167:** 1279–85
Leedman PJ, Stein AR, Chin WW et al (1996) Thyroid hormone modulates the interaction between iron regulatory proteins and the ferritin mRNA iron-responsive element. *J Biol Chem* **271:** 12017–23
Takamatsu J, Majima M, Miki K et al (1985) Serum ferritin as a marker of thyroid hormone action on peripheral tissues. *J Clin Endocrinol Metab* **61:** 672–6

Correction: alcohol misuse

In the correspondence from Touquet and Huntley (Vol 64(1), 2003, p.49) the heading of the letter was shortened in such a way that it misrepresented the letter. The full title to the letter was originally: 'Prevention of the development of alcohol dependence by the early detection of alcohol misuse in accident and emergency departments'. We would like to apologize for any confusion or embarrassment caused by this error.