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EDITORIAL

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Testosterone and the primary care physician

Once the diagnosis and the decision to treat has been made, the benefits are in many cases startling

Outside a relatively small group of clinicians – urologists, endocrinologists, family physicians and those with a particular interest in the subject – the clinical significance of the fall in testosterone in the older man is not widely understood, despite a large bibliography over the last decade. It is well recognised that as men age, there is a decline in serum testosterone levels, mean values at age 75 being about two-thirds of those at age 25, with more than 20% of men older than 60 years with a subnormal testosterone level (1). However, the importance of late onset hypogonadism, or 'the andropause' as it is popularly but inaccurately called, is either ignored, denied by many or even scorned, with one endocrinologist being so scornful as to call the andropause or so-called male menopause a 'myth to excuse the lazy and unfit' (2).

A report by the American Institute of Medicine in 2003 in reviewing what had been written until then on the risks and benefits in replacing testosterone in older men, pointed out gaps in our knowledge and the need for further research (3). Miner and Seftel in their paper in this journal have now surveyed some key studies written since the 2003 report, and having updated it, have in their paper focussed on those areas of particular interest to primary care physicians or family doctors, stating categorically that family doctors can easily identify and treat men with hypogonadism.

The symptoms of testosterone deficiency are multiple and are individually often confused with those of depression, but typical symptoms are: decreased sex drive and sometimes erectile problems, unusual fatigue, sometimes with profuse nocturnal sweating and flushing and mood changes for the worse. Although these symptoms may arouse a high degree of suspicion, a diagnosis should not be made on these alone but should be supported biochemically. An accurate diagnosis is important as a low testosterone is associated with prevalence of coronary artery disease, a greater

incidence of type 2 diabetes compared with eugonadal men and an incidence of hypogonadism in a quarter of men with erectile dysfunction. Bone mineral density is reduced in men with a low serum testosterone and overall, a low testosterone is positively related to the onset of the metabolic syndrome. The authors justly point out that the high prevalence of the metabolic syndrome and diabetes is becoming a major economic significance because of their costs.

Once the diagnosis and the decision to treat has been made, the benefits are in many cases startling. Mood improves, sexual desire increases and function improves, fatigue lessens, sweating and flushing diminish, cognitive function improves, HDL cholesterol levels improve as do hypertension and hyperlipidaemia and the general quality of life improves considerably. Erectile dysfunction is considerably helped in a proportion of men who have used a phosphodiesterase-5-inhibitor alone with little success, possibly by decreasing systemic vascular resistance.

However, the majority of family doctors are wary about initiating replacement testosterone therapy because they know insufficient about problems which may be associated with treatment. In particular, there is considerable anxiety about provoking carcinoma of the prostate, as the relationship between exacerbating an existing carcinoma and provoking a new one is not fully understood.

A high proportion of apparently healthy men have been found at postmortem to have had foci of neoplastic cells in the prostate which have not grown or affected the patient during life. The great question has been, can testosterone therapy set off growth of these quiescent seedlings? As Miner and Seftel say (4), the anxiety arose from animal studies and anecdotes, which cannot be extrapolated to men. Although it has long been recognised that an already proven prostate carcinoma is androgen dependent, which has led to attempts at androgen ablation with anti-androgens or castration, so far, there is no evidence that replacement – not supplementary – androgen therapy instigates carcinoma. A meta-analysis of 19 randomised studies evaluating 651 men concluded that the risk of prostate cancer did not increase significantly during testosterone replacement therapy (5).

Family doctors will be reassured with their conclusion that no new prostate safety issues have been reported since 2003, despite the cautious caveat that even so, the evidence for an association between testosterone replacement therapy is still inconclusive.

In a recent editorial in this journal, Jackson has pointed out the negative worries for primary care physicians – aggravating existing prostate cancer, but not causing it, reducing sperm count and an increase in haematocrit (6). With careful monitoring of the PSA, the main worry can be diminished and Miner and Seftel finish their review with a helpful list of recommendations for monitoring prostate health before and during testosterone replacement.

Once family doctors have realised that their anxieties can be resolved, there is no reason why men with late onset hypogonadism cannot be treated perfectly safely in the clinician's clinic outside the hospital, without referral to an endocrinologist or urologist, unless the occasional support is required. A protocol should be prepared and adhered to and there will then be little or no worry attached to hormone replacement in the male, and a great deal of satisfaction will be obtained both by the clinician and the patient.

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