Hormones and the heart — is sleep apnoea the link?: Reply

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Sir,

We welcome the discussion on the effects of androgen therapy in patients with heart failure. We have recently completed a pilot study on the effects of testosterone therapy in men with stable congestive heart failure. This study showed an increase in functional capacity and a reduction in the NYHA class in patients treated with intra-muscular testosterone compared to placebo. These patients were not screened for obstructive sleep apnoea (OSA) prior to randomization, although sections of the questionnaires used did address quality of sleep and levels of daytime fatigue. Daytime somnolence was not identified as an adverse effect of treatment. Our impression from the literature was that treatment for OSA (with continuous positive airways pressure ventilation (CPAP)) was only indicated for patients with this feature, since only patients with daytime somnolence derive symptomatic benefit. The haemodynamic effects of CPAP in patients with stable congestive heart failure and OSA are certainly impressive although there are currently no outcome studies with clinical endpoints. In our pilot study, if testosterone therapy had precipitated or worsened OSA we would have expected a deterioration in function, whereas the reverse was seen. Furthermore, there was no deterioration in ejection fraction or of any haemodynamic variable tested. In addition testosterone therapy in healthy and morbid populations reduces obesity, a benefit that theoretically would reduce the severity of upper airway obstruction. However, the comments made by Dr Morton do have implications for safety of our patients with symptomatic OSA. In our future studies of testosterone on patients we will enquire about symptomatic OSA using screening questionnaires.

Finally, spironolactone is a weak androgen receptor blocker. The acute haemodynamic effects of testosterone are not mediated at the androgen receptor level since observed effects occur too rapidly to be mediated by nuclear transcription and are not inhibited by complete blockage of the androgen receptor by flutamide. In vitro cell culture and myograph work performed by our group shows that testosterone causes acute vascular dilatation mediated by inhibition of calcium channels in the vascular smooth muscle membrane.

References


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