The renal effects of dopamine and dobutamine in stable chronic heart failure

Sir,

Among the observations made by Good and colleagues¹ was the generalization that angiotensin converting enzyme inhibitors usually blunt the diuretic response to frusemide. Whilst it is true that the beneficial effects of these drugs on cardiac performance² could be offset by renal deterioration, and, hence, impaired diuresis, due to drug-mediated hypotension³ or co-prescription of excessive doses of frusemide,⁴ there must be some patients in whom careful drug titration⁵ strikes the right balance between the beneficial effects on natriuresis⁶ and the adverse effects on glomerular filtration rate. The consequence could be a stabilization in diuretic requirements or even a diuretic sparing effect, as shown by some of the patients reported by Dzau *et al.*⁷ and by Odemuyiwa *et al.*⁸ These beneficial effects may be obscured by the dosing schedules employed in double-blind trials of angiotensin converting enzyme inhibitors.

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rarely with predominant mineralocorticoid deficiency preceding critical loss of glucocorticoid reserve in evolving adrenal failure. They may be clinically Addisonian, but with serum cortisol concentrations lying within the normal range. There has been some dispute about what constitutes a normal response to Synacthen,² although we have attempted to define the correct range in a prospective series of 50 patients admitted acutely to hospital.³ In this study we suggested a normal basal cortisol concentration of 250 nmol/l, and a 30 minute peak of > 600 nmol/l. The case reported by Butcher *et al.* fulfilled these criteria – even though the peak serum cortisol response was assessed at 60, rather than at 30 minutes. Nevertheless it seems that the patient's glucocorticoid reserve was essentially normal at presentation, while his aldosterone concentration was low. This has been previously reported⁴ and we also have managed a similar case.

The patient was a 25 year old motor mechanic who had been previously well. He presented as an emergency with a 9 day history of weakness and intermittent collapse. On examination lying/sitting blood pressures were 130/80 and 110/90 mmHg, respectively. Serum sodium was 108 mmol/l, potassium 6.8 mmol/l and urea 11.9 mmol/l. Even though he was not pigmented, Addisonian crisis was suspected and a short Synacthen test was carried out, subsequently showing a basal 9 a.m. cortisol of 110 nmol/l and peak 30 minute cortisol of 720 nmol/l.

He was rehydrated and started on hydrocortisone and fludrocortisone with good effect. In view of the normal response to Synacthen, corticosteroids were stopped for 24 hours and the short Synacthen test was repeated (5 days following the initial test). On this occasion the basal 9 a.m. cortisol was 150 nmol/l and peak 30 minute cortisol 170 nmol/l. Plasma ACTH on an admission sample was 220 ng/l (upper limit of normal 120). Plasma renin concentration (overnight recumbent) was grossly elevated at 510 ng/ml/hour (1–4.5) with a relatively low serum aldosterone concentration of 0.83 nmol/l (0–5.5 nmol/l). Adrenal antibodies were positive. He remains well on full corticosteroid replacement therapy since that time.

These cases emphasize the need to interpret the results of the short Synacthen test in the light of the clinical presentation. It remains, however, a simple and extremely robust test, and generally has high sensitivity.

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Addisonian crisis presenting with a normal short tetracosactrin stimulation test

Sir,

The case reported by Butcher *et al.*¹ draws attention to the fact that patients with Addison's disease may present