Letters to the Editor

Carcinoid tumours

Sir,

The recent article by Janmohamed and Bloom provides an excellent overview of current methods of investigating and treating carcinoid syndrome (CS).[1] Octreotide is effective and has revolutionised the management of CS but a small number of patients do not respond to this treatment and in others refractory disease may develop. In these cases some alternative treatment is required. Meta-iodobenzylguanidine (MIBG) has been used in the investigation and treatment of phaeochromocytoma and although mentioned only briefly in their review, has been shown to be therapeutic in CS.[2-5] Taal and colleagues demonstrated a 60% improvement in symptoms with both MIBG and ^{131}I-MIBG, although a longer-term benefit was noted with the latter formulation.[2] Similar improvement and disease stabilisation was demonstrated by Bestagno and colleagues in four of six cases treated with ^{131}I-MIBG.[3] In a further study comparing 'salvage' therapy in CS, greater clinical efficacy was demonstrated in the subjects treated with ^{131}I-MIBG than with chemotherapy.[4]

In view of the distressing symptoms associated with refractory CS, we would suggest that ^{131}I-MIBG should be considered as a useful treatment option for this condition.

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1 Janmohamed W, Bloom S. Carcinoid tumours. *Postgrad Med J* 1997; **73:** 207-14.
2 Taal BG, Hoefnagel CA, Valdes Olmos RA, Boot H, Beynen JH. Palliative effect of MIBG in metastatic carcinoid tumours. *J Clin Oncol* 1996; **14:** 1829-38.
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4 Zilembo N, Buzzoni R, Bajetta E, *et al.* Salvage treatment after r-interferon 2a in advanced neuroendocrine tumours. *Acta Oncol* 1993; **32:** 245-50.
5 Watanabe N, Seto H, Ishiki M, *et al.* I-123 MIBG imaging of metastatic carcinoid tumour of the rectum. *Clin Nucl Med* 1995; **20:** 357-60.

Management of congestive heart failure

Sir,

Guidelines, such as those proposed by McAlister and Teo for heart failure management,[1] are useful as a basis for good practice, but their limitations lie in their failure to cater for patients who prove unable to attain optimum drug doses due to limitations imposed by drug side-effects. Such is the lot of patients who already have pre-treatment borderline hypotension due to severe left ventricular systolic failure, rendering them incapable of tolerating optimum doses of angiotensin-converting enzyme (ACE) inhibitors. Due to consequent hypokalaemia resulting from the imbalance between the potassium-conserving activity of the ACE inhibitor and the kaliuretic action of concomitant loop diuretic therapy, these patients are often coprescribed potassium supplements, but a more advantageous strategy would be the coprescription of spironolactone instead, due to its ability to potentiate loop diuretics when coprescribed with ACE inhibitors,[2-4] ultimately leading to a reduction in diuretic requirements. Other aspects of coprescription of ACE inhibitors and spironolactone currently under scrutiny include a possible survival benefit in heart failure patients,[5] and the use of long-term oxygen therapy. Oxygen therapy may be useful not only for patients with cor pulmonale as the sole diagnosis,[6] but also for those who, as a result of the shared risk of chronic bronchitis and coronary heart disease in cigarette smokers, have cor pulmonale coexisting with left ventricular systolic failure, the latter diagnosis being often overlooked in the presence of chronic obstructive airways disease. The corollary to this is to mistake the coexistence of cor pulmonale and left ventricular systolic failure for the evolution of the latter into biventricular failure. Whichever way one looks at it, opportunities remain largely unexplored for maximising diagnostic as well as therapeutic opportunities in congestive cardiac failure.

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1 McAlister FA, Teo KK. The management of congestive heart failure. *Postgrad Med J* 1997; **73:** 194-200.
2 Dahlstrom U, Karlsson E. Captopril and spironolactone therapy for refractory congestive heart failure. *Am J Cardiol* 1993; **71:** 29A-33A.
3 van Vliet AA, Donker Ab JM, Nauta JJP, Verheugt FWA. Spironolactone in congestive heart failure refractory to high-dose loop diuretic and low-dose angiotensin-converting enzyme inhibitor. *Am J Cardiol* 1993; **71:** 21A-28A.
4 Ikram H, Webster MWI, Nicholls MG, *et al.* Combined spironolactone and angiotensin converting enzyme inhibitor therapy for refractory heart failure. *Aust N Z J Med* 1986; **16:** 61-3.
5 The RALES investigators. Effectiveness of spironolactone added to angiotensin-converting enzyme inhibitor and a loop diuretic for severe heart failure (The Randomised Aldactone Evaluation Study (RALES). *Am J Cardiol* 1996; **78:** 902-7.
6 Medical Research Council Working Party. Long term domiciliary oxygen therapy in chronic hypoxic cor pulmonale complicating chronic bronchitis and emphysema. *Lancet* 1981; **1:** 681-6.

This letter was shown to the authors who responded as follows:

Sir,

We agree with many of the comments by Jolobe. Our paper was an attempt to provide a summary of contemporary knowledge, derived mostly from major clinical trials and epidemiological data, which can be applied in the management of congestive heart failure, a condition that is becoming increasingly common as the population in our countries ages. Clinicians can only use guidelines as a distillation of the knowledge available with which to guide practice but must make unique judgements in providing the individual patient with effective and safe therapy. We too have faced the frail, hypotensive patient with a severely impaired left ventricular function who cannot tolerate even a small dose of an ACE inhibitor and in whom we have to consider the use of other vasodilators. In hypokalaemic patients requiring large doses of diuretics, the combination of potassium-sparing diuretics such as spironalactone with an ACE inhibitor can be useful and can replace the large doses of otherwise necessary potassium supplements, but in this case it is only sensible that serum potassium levels be monitored regularly to avoid the danger of hyperkalaemia.

We are aware of the need for additional therapy in special groups of patients as mentioned by Jolobe, but we decided to refrain from expressing opions about therapies which appear physiologically sound but on which there are no definitive trial data. We also agree that the diagnostic and therapeutic options are far from complete, and there are many more research opportunities to be explored.

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