LETTERS TO THE EDITOR

● *The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.*

● *All letters must be typed with double spacing and signed by all authors.*

● *No letter should be more than 600 words.*

● *In general, no letter should contain more than six references (also typed with double spacing).*

Asymptomatic ischaemia during daily life in stable coronary artery disease: relevant or redundant

SIR,—In their interesting review on the prognostic implications of silent myocardial ischaemia¹ Mulcahy *et al*, referring to our paper on silent ischaemia after myocardial infarction,² wrote: "Solimene *et al* performed ambulatory ST segment monitoring in 40 patients eight weeks after a first myocardial infarction and followed them for two years. Six patients had asymptomatic ischaemia during ambulatory monitoring. No events occurred in them: there was one cardiac death in a patient without ischaemia." There was some misinterpretation of our data. In fact, our investigation showed that 11 (27.5%) out of 40 patients had silent ischaemia after infarction: five only on exercise testing, five on exercise testing and Holter monitoring, and one on Holter monitoring. Of those 11 patients, four (36%) had a non-fatal cardiac event whereas only one (3.6%) of 29 patients without silent ischaemia had a cardiac event (fatal reinfarction) during this two year follow up. Kaplan-Meier analysis showed that during this period patients without silent ischaemia were much less likely to experience a cardiac event (96.5%) than patients with ischaemia (62.3%) ($P < 0.007$). We concluded that silent myocardial ischaemia after myocardial infarction is of considerable prognostic significance—a somewhat different conclusion from that reached by Mulcahy *et al*.

MARIA CECÍLIA SOLIMENE
Instituto do Coração,
Av. Enéas de Carvalho Aguiar 44, 2o A,
050403-000 São Paulo, Brazil

1 Mulcahy D, Purcell H, Patel D, Fox K. Asymptomatic ischaemia during daily life in stable coronary disease: relevant or redundant? *Br Heart J* 1994;72:5-8.

2 Solimene MC, Ramires JAF, Gruppi CJ, *et al*. Prognostic significance of silent myocardial ischaemia after a first uncomplicated myocardial infarction. *Int J Cardiol* 1993;38:41-7.

This letter was shown to the authors, who reply as follows:

SIR—I thank Dr Solimene for her letter. Our review was about the prognostic significance of transient myocardial ischaemia detected on ambulatory ST segment moni-

toring and not exercise testing or any other investigation. In her letter Solimene confirms the figure we quoted of six patients with transient ischaemia on ambulatory monitoring after myocardial infarction.

In their study of 40 patients Solimene *et al* related silent ischaemia after myocardial infarction detected by any investigation (that is, exercise testing, $n = 10$; ambulatory monitoring, $n = 6$; one or the other, $n = 11$) to events, and not to a straight assessment of ambulatory ischaemia versus outcome. Only one "hard" coronary event (acute myocardial infarction or sudden coronary death) was reported by Solimene *et al* (cardiac death), and this occurred in a patient who did not have transient ischaemia on ST segment monitoring. We reported this in our review which focused on the relation between transient ischaemia and subsequent death or non-fatal myocardial infarction. Recurrence of angina (referred to as a non-fatal cardiac event by Solimene *et al*) was reported to occur in four patients with silent ischaemia on either exercise testing or ambulatory monitoring—Solimene *et al* do not state which. To reply to Solimene's letter in the context of our review, and to establish whether "soft" end points occurred in those with transient ischaemia during daily life, we would need to know how many of these four recurrences of angina occurred in those with only a positive exercise test, and whether anything further happened to them.

DAVID MULCAHY
Department of Health and Human Services,
Public Health Service,
National Institutes of Health,
National Heart, Lung, and Blood Institute,
Bethesda, Maryland 20892,
USA

Issues in cardiac pacing: can agism be justified?

SIR,—The continuing debate surrounding the cost effectiveness of rate adaptive pacing in the elderly remains handicapped by a lack of reliable data.¹ The antagonists would point to the absence of hard clinical evidence to support the use of sophisticated pacemaker technology in the elderly. Recent trials, however, which have specifically included elderly subjects, have confirmed the clinical impression that the elderly stand to gain as much from physiological pacing as younger patients.²⁻⁴ However, the biggest stick with which to beat the enthusiasts is that of cost. In a retrospective analysis, de Belder *et al* estimated that implantation of dual chamber pacemakers in all suitable patients (that is, those with advanced atrio-ventricular block and sinus rhythm) aged over 75 years would have added an extra £264 357 to the regional pacing budget (an increase of 57%).⁵ Given these figures it is little wonder that there is some reluctance to implement the BPEG guidelines in the elderly.⁶ It is important to realise, however, that these figures were based on the assumption that all electrophysiologically suitable patients aged over 75 would have been given DDD pacemakers.

Patients aged over 75 years may constitute a selected group in whom the presence of advanced conduction disease may be a marker of an advanced aging process. Limiting, non-cardiac disease or cognitive impairment—for example, previous stroke—is not uncommon in this

group and such patients would not normally be considered for a dual chamber system. We do not know what proportion of these patients are offered VVI systems on the grounds of limiting, non-cardiac disease or cognitive impairment. Nevertheless, it is clear that available estimates of the financial impact of the BPEG guidelines are likely to be exaggerated and serve only to foster inappropriate implantation policies.

In addition to further clinical trials, which are likely to confirm the overall benefits of physiological pacing in the elderly, we need reliable information on the costs of implementing these research findings.

M R HARGREAVES
O ORMEROD
Cardiac Department,
John Radcliffe Hospital,
Headington,
Oxford OX3 9DU

- 1 Payne GE, Skehan JD. Issues in cardiac pacing: can agism be justified? *Br Heart J* 1994;72:102-3.
- 2 Channon KM, Hargreaves MR, Cripps TR, Gardner M, Ormerod OJM. DDD vs VVI pacing in patients aged over 75 years with complete heart block: a double blind crossover comparison. *Q J Med* 1994;87:245-51.
- 3 Jordaens L, de Backer G, Clement DL. Physiologic pacing in the elderly. Effects on exercise capacity and exercise-induced arrhythmias. *Jap Heart J* 1988;29:35-44.
- 4 Avery PG, Banning AP, Lawson T, McGurk L, Buchalter MB. Age should not be a contraindication for physiological pacing [abstr]. *Br Heart J* 1994;71(suppl):P71.
- 5 de Belder MA, Linker NJ, Jones S, Camm AJ, Ward DE. Cost implications of the British Pacing and Electrophysiology Group's recommendations for pacing. *Br Heart J* 1992;305:861-5.
- 6 British Pacing and Electrophysiology Group. Recommendations for pacemaker prescription for symptomatic bradycardia. *Br Heart J* 1991;66:185-91.

The British Pacing and Electrophysiology Group Guidelines on pacemaker group prescription, published in 1991, have generated much controversy. It is clear that universal implementation of the BPEG guidelines will be very expensive. We need to know whether this expense is justified in terms of longevity, quality of life, reduction in strokes and effect on heart failure. We cannot know until we have the results of planned trials. Let us have a moratorium until then.—EDITOR

Detection of left ventricular dysfunction after myocardial infarction: comparison of clinical, echocardiographic, and neurohormonal methods.

SIR,—A major limitation of the Peel index, even in its modified form¹ is that it does not take into account the adverse prognostic significance of the association of myocardial infarction and ST segment depression. In thrombolytic trials such patients continue to have a high mortality despite treatment²⁻³—not only because ST segment depression is an independent predictor of poor prognosis⁴ but also because it sometimes signifies structural damage caused by previous myocardial infarction.⁵ Furthermore, even when patients with ST segment depression prove to have smaller infarcts than their counterparts with ST segment elevation, they still have more severe impairment of left ventricular systolic function.⁵ These patients should, therefore,