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 (event 9-5% vs 62-3%) (P < 0-001). 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.

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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 monitoring and not exercise testing or any other investigation. In her letter Solimene confirms the figure noted by us; 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 ambulatory monitoring (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 as an 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 after substernal 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 or whether anything further happened to them.

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Issues in cardiac pacing: can agism be justified?

SIR,—The continuing debate surrounding the cost effectiveness of rate adaptive pacing in the elderly remains a handicap to the current research effort because of 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 specifically included elderly subjects, have confirmed the clinical impression that the elderly stand to gain as much from physiological pacing as younger patients.2,3 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 £264 557 to the regional pacing budget (an increase of 57%).4 It is quoted of figures it is little wonder that there is some reluctance to implement the BPEG guidelines in the elderly.4 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 how many of these elderly 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 electrophysiological pacing in the elderly, we need reliable information on the costs of implementing these research findings.

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4 Avery PG, Banning AF, Lederle FA, McCormick L, Buchalter MB. Age should not be a contraindication for physiological pacing (abst). Br Heart J 1994;71(suppl III):13B.

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

SIR.—A major limitation of the Peet index, even in its modified form1 is that it does not take into account the adverse prognostic significance of the association between left ventricular infarction and ST segment depression. In thrombolytic trials such patients continue to have a high mortality despite treatment—a not only because ST segment depression is an independent predictor of poor prognosis but also because it sometimes signifies structural damage caused by previous myocardial infarction.2 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.3 These patients should, therefore,
be targeted for interventions such as treatment with angiotensin converting enzyme inhibitors after myocardial infarction, perhaps even irrespective of criteria generally implemented in other post-myocardial infarction subgroups.

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

St. —Dr Timmis discussed the limitation of early biochemical diagnosis of acute myocardial infarction in guiding thrombolytic therapy.1

The mortality of infarct patients in Newham General Hospital who present without ST elevation is only a third of that with ST elevation, none the less about one in 20 of such patients died. In addition, in infarct patients who present with predominant ST depression one year mortality is high (31%).2 De Wood et al’s angiographic study of myocardial infarction was performed up to 24 hours after acute myocardial infarction3 and the patency rate caused by spontaneous coronary re-canalisation would be expected to be higher than in the first 12 hours, the time window when thrombolytic therapy is believed to be effective.4 Even so, 26% of these patients had occluded coronary arteries and might have benefited from revascularisation treatment. The result of the ISIS-2 trial suggests that patients without ST elevation (except bundle branch block) would not benefit from thrombolytic therapy.5 The inclusion criteria of ISIS-2 raise the possibility that an appreciable number of these patients may not have had a myocardial infarction at all. No definitive data are currently available to define treatment in patients with early biochemical confirmation of acute myocardial infarction, though the LATE study6 included patients with old or equivocal electrocardiograms. Early biochemical diagnosis may be useful in guiding this treatment.

Furthermore the use of rapid assays may offer advantages in terms of efficacy. In patients with atypical chest pain may be discharged earlier after negative results. None the less, to exclude acute myocardial infarction, myocardoglobin should be measured 4–6 hours after the onset of chest pain and creatine kinase MB 6–8 hours after the onset of chest pain.7

Rapid biochemical diagnosis of acute myocardial infarction may be useful in guiding treatment and the more efficient management in coronary care units of patients who present with chest pain.

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4 LATE Study Group. Late assessment of thrombolytic efficacy (LATE) study with alteplase 6–24 hours after onset of acute myocardial infarction. Lancet 1993;342:759–66.


7 Lee HS, Cross SJ, Garthwaite P, Jennings K. Rapid exclusion of acute myocardial infarction in patients without ST elevation using serial cardiac enzyme analysis with a non diagnostic electrocardiogram (ECG) on admission to hospital, but a confirmed diagnosis of infarction on discharge. The GISSI study enrolled 451 patients with ST elevation on the ECG; the mortality in this whole group was 18.4%, and did not differ significantly whether streptokinase or placebo was used.2 ISIS-2 enrolled 1137 such patients, and again the mortality rate of 18.6% was not improved by streptokinase.3 The ASSET study distinguished only between normal and abnormal ECGs without distinguishing specific ECG abnormalities.4 The EGG study recruited 2534 patients who were admitted with chest pain, and of these 65% were not enrolled with ST segment elevation only.1 In the LATE study, 93% of patients had a discharge diagnosis of definite or possible infarction. In the group of 2544 patients without ST elevation on the ECG, the 35 day mortality was 7.5% in the placebo group and 6.4% in the group treated with alteplase.
Detection of left ventricular dysfunction after myocardial infarction: comparison of clinical, echocardiographic, and neurohormonal methods.
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