Sir,
We welcome the comments from Morgan and Mant on our paper on the use of warfarin in atrial fibrillation (March Journal, p.153). We would like to address the points they raise.

The calculations for the number of patients who would need to be treated to prevent one stroke were derived initially from the paper by Cairns and were also confirmed by Nolan and Bloomfield. We checked these data and they were correct. We think that Morgan and Mant have produced calculations derived from the data in the meta-analysis but our data do not represent an error.

We accept that on Table 1 the ‘relative risk of warfarin (%)’ is better expressed as ‘risk reduction with warfarin (%)’.

We regret that in Table 2, data for two of the studies were inadvertently transposed. The Landefield paper referenced in Table 3 contains the correct data but these are associated with a different publication.

We suggest in the paper that it may not be safe to extrapolate from the small residual group of patients who are in the trials to the mass of patients in the care of general practitioners in the United Kingdom. The challenge is not so much the quality of management as to know when physical, psychological and social factors justify stopping or starting warfarin. We were surprised that Morgan and Mant should raise the issue of specialist liaison nurses for whom they offer no evidence. Such nurses are unlikely to be able to balance the risks of starting or stopping warfarin better than clinical generalists.

There were two main points raised in our review article. The first was the discovery that high exclusion rates (up to 97%) of eligible patients in the main trials raises serious scientific questions about the generalizability of the findings. Secondly, the complication rates have not yet been satisfactorily evaluated in terms of patient safety and the impact on primary health care teams.

We would welcome further comments from practising general practitioners.

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References

Acute myocardial infarction
Sir,
I am rightly reproved by Michael Moher (letter, August Journal, p.444) for not mentioning the importance of aspirin in acute myocardial infarction in my editorial on the general practitioner’s role in the early management of acute myocardial infarction (April Journal, p.171).

Aspirin is an antiplatelet drug that helps to prevent a coronary thrombus from extending or from reforming after it has been lysed. Because aspirin has a different mode of action from that of thrombolytic drugs, its benefits are additional to those of thrombolytic therapy, which was the main topic of my article. Aspirin also differs from thrombolytics in that the timing of administration is not so critical. From knowledge of its action, we would not expect the benefit of aspirin to be much increased by earlier administration, and this seems to be the case. Aspirin should certainly be given to the patient with suspected acute myocardial infarction, but when it is given is not critically important.

By contrast, the timing of thrombolytic therapy is of the utmost importance; restoring coronary flow within an hour or two of occlusion results in myocardial salvage, smaller infarcts, and substantial immediate and long-term benefits. The additional benefit of giving thrombolytic therapy before admission to hospital may exceed the absolute benefit of giving thrombolytic therapy in hospital four to five hours after onset, or of giving aspirin at any time. In terms of the potential number of lives saved, giving thrombolytic therapy is as urgent as the treatment of cardiac arrest; ideally, either treatment should be carried out by the first qualified person on the scene.

The general practitioner called to see a patient soon after acute myocardial infarction has the chance to reverse the underlying pathophysiology with thrombolytic therapy and aspirin, a golden opportunity indeed.

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Management of angina
Sir,
We thank McKinley and Khunti (letter, June Journal, p.328) for their interest in our paper, but fear that they have misunderstood our main findings. We are puzzled that they reiterate the basics of exercise testing and its predictive value even though our paper categorically states that exercise test results from tertiary centre patients cannot be extrapolated to angina patients in the general population, and explicitly acknowledges the lack of data for community-based angina patients.

The central finding of our study was that the vast majority of patients presenting with angina are reportedly not referred for a specialist cardiac assessment. Observational data from prospective studies show that at least 10% of new angina patients experience a myocardial infarction or die as a result of coronary heart disease within one year of presentation, and that the risk of angina patients developing unstable angina or myocardial infarction or of dying within two years of presentation is threefold higher than that of the general population. We have further demonstrated that about 30% of patients with typical angina have marked ischaemia (>3 mm ST segment depression) at a low workload on exercise testing at the time of initial presentation to their general practitioner, and a similar proportion have coronary artery disease for which revascularization with coronary angioplasty or coronary bypass grafting is considered appropriate by consultant cardiologists.

Thus, while we are pleased that McKinley and Khunti repeat our concluding remarks underscoring the urgent need for randomized clinical trials to optimize selection of community-based angina patients for coronary revascularization, we believe that until such evidence is available, a policy of ‘no action’ by general practitioners would be a retrograde step. It is difficult to imagine how the short-term prognosis of new angina patients in the community can be improved without increasing the rate of specialist cardiology referral.

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