

thrombolytic drug after a myocardial infarction although he was without pain or shock. He writes elegantly and persuasively, pointing out that, since absolute mortality is reduced by 2% and the risk of severe bleeding is 1.1%, it is quite extraordinary that we should think that thrombolysis is mandatory. He goes on to say that beta-blockers should not be used routinely after myocardial infarction because, although they prevent four deaths in the first 2 years, they take the edge off life and can be seriously damaging. He quotes practolol as an example.

To take the last point first, I think 4% in 2 years is better than nothing, we don't use practolol, and I don't know of any other beta-blocker that has caused unpredictable damage. Furthermore, none of us give beta-blockers to everyone; a patient after a small first inferior infarction without evidence of arrhythmias or reversible ischaemia may remain unblocked, and one with a non-Q infarction but no failure is probably better served by diltiazem. The beta-blockers have most of their benefit within the first 6 months, so if they are taking the edge off life they don't have to go on for ever.

The figures for thrombolysis are true but incomplete. They apply to short-term studies. Thrombolytics open up arteries earlier; logically they should only then be used when this is likely to produce a significant benefit. But benefit is surely not only in reduced hospital mortality<sup>1</sup>? Opening an artery earlier than the patient can is intended to save muscle. This in turn should prevent, postpone or reduce muscle damage<sup>2</sup> with its corollary of heart failure and the miserable quality of life that may go with it. These people too can be counted on the winning side, although I'm not aware of any trials long enough to demonstrate this benefit, and the improvement in infarct size and ejection fraction, though not early mortality, may be restricted to those given a thrombolytic drug within 2 hours of symptoms developing<sup>3</sup>.

I hope many people have read Dr McGouran's article. Apart from the artist's bias it points out only too well how drug company profits and p values can distract us from thinking; in particular thinking about what is best for each individual patient.

## References

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- 3 Chareonthaitawee P, Gibbons RJ, Roberts RS, Christian TF *et al* (for the CORE investigators). The impact of time to thrombolytic treatment on outcome in patients with acute myocardial infarction. *Heart* 2000;84:142-8.

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## In response

Editor – Dr Best claims 'artistic bias' in my article but I was very careful to stick to the facts, questioning only their application in clinical practice. Unlike him I did not claim a long-term benefit from thrombolysis in improved myocardial function because, as he admits in the same sentence, there is no evidence supporting this.

He points out that we don't use practolol today and of course I am aware of this but practolol was one of the beta blockers used in the early studies of death in the two years after infarction and the recommendation then was that these drugs should be used routinely in infarct patients. He doesn't dispute that the trials showed that 4 patients in 100 would benefit and 96 would not. Surely therefore the advice that every patient should take a beta blocker could only have been justified if we had been certain that they were without significant side effects and the subsequent tragic history of practolol confirms that we can't know this.

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## Prevention and treatment of osteoporosis

Editor – I have found the RCP osteoporosis guidelines (*JRCPL* November/December 2000, pp518-21) a useful practical guide in a difficult area. However, I wonder if I could clarify a number of points.

The grading of interventions is obviously important at a local level when priorities

need to be set. In this regard there seems to be some discrepancy between the published tables of anti-fracture efficacy and those mentioned in the editorial, in that non-randomized studies appear to have slipped from grade A to grade B. In addition I am not aware of any major peer-reviewed studies showing that risedronate has an effect on hip fractures alone.

In the large scale bisphosphonate and raloxifene studies most patients received supplements of calcium or calcium and vitamin D – would the writing group suggest routine supplementation, or should vitamin D levels be checked first?

Lastly, the vexed question of screening for myeloma. The writing group suggest myeloma screen 'when indicated'. We have spent some time discussing at what ESR level etc we would do this and would be grateful for clearer guidance.

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## In response

We are grateful to Dr Lloyd for drawing attention to the ambiguous setting of Table 1 in the Update document, in which it might appear that well-designed controlled studies without randomisation are included in the Grade A evidence base. This is, of course, not the case and only randomised controlled trials or meta-analyses of these are considered a sufficient evidence for Grade A recommendation.

Dr Lloyd also questions the evidence for the effect of risedronate on hip fracture. At the time of publication of the Update, the clinical trial data had been presented at several international meetings although publication was only available in abstract form. A full peer-reviewed paper has subsequently been published (*New England Journal of Medicine* 2001;344: 333-401).

We agree that in recent randomised controlled trials calcium and vitamin D supplementation has been given either to all patients or to those with evidence of vitamin D deficiency and hence the efficacy of these agents has been established only in vitamin D replete individuals. In clinical practice, it seems reasonable to recommend calcium and vitamin D supplementation in those at risk from deficiency (eg in individuals with little sunlight exposure

or low dietary calcium intake) but routine assessment of vitamin D status in otherwise healthy postmenopausal women is unlikely to be cost-effective.

Finally, the decision about whether or not to screen for myeloma is a difficult one and cannot be based on any one parameter. In general it should be considered in individuals presenting with spinal osteoporosis

in whom there are no obvious risk factors or other underlying causes. However, if clinical suspicion exists on any basis (including an elevated ESR) screening should be performed.

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## Clinical & Scientific letters

Letters not directly related to articles published in *Clinical Medicine* and presenting unpublished original data should be submitted for publication in this section. Clinical and scientific letters should not exceed 500 words and may include one table and up to five references.

### Temporary cardiac pacing and the physicians of tomorrow

In district general hospitals (DGHs), temporary pacing is usually provided by general medical firms and it continues to be a core component of general internal medicine (GIM) training. Limited instruction and supervision<sup>1</sup> have contributed to a high complication rate<sup>2-4</sup> and the declining number of temporary pacings<sup>5</sup> plus shorter working hours of doctors in training have further compromised exposure. Are specialist registrars (SpRs) currently in training likely to have gained sufficient experience to provide a pacing service when they become consultants?

All SpRs in one region were identified and those training in cardiology and in specialties unlikely to provide GIM were excluded. A questionnaire was sent to the remainder, of whom 80/102 replied (78%). The median experience at SpR level was 3.3 years and all but two were expecting to provide an acute medical service when they became consultants. In the preceding year the median number of pacings performed was one and 35 (44%) had not paced for over 12 months. Forty-nine (61%) felt that their training had been inadequate and only 24 (30%) felt that upon completion of training they would be competent to pace. Thirty-seven (46%) thought that general physicians in DGHs should pace, 35 (44%) thought that only cardiologists should be

involved and 8 (10%) believed that both could be involved, depending on experience.

Forty-nine SpRs were within two years of completing their training and their responses were analysed separately. Two of them had never performed a pacing and both were expecting to provide an acute medical service. Thirty-seven (76%) did not feel capable of providing a pacing service, 51% believed that pacing should be provided purely by cardiologists.

The current practice of temporary cardiac pacing is unacceptable. It should no longer be provided by all general physicians and should not be a core component of GIM training. Those who wish to provide a pacing service require guidance on how many procedures they have to perform to achieve and maintain competence. All units and individuals who pace should audit the practice and complications of their service in conjunction with their cardiac referral service. Solutions will vary according to local facilities and expertise. For units that lack expertise, atropine and external pacing could facilitate rapid transit to more experienced centres.

### References

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### Bile acid malabsorption and post-infective diarrhoea

A recent article on bile acid malabsorption and persistent diarrhoea (*JRCPL* September/October 2000, pp448-51) highlights an important cause of diarrhoea. I would like to draw the attention of the authors to our study (*JRCPL* January/February 1997, pp53-6) which was the first to observe an association of bile acid malabsorption and post-infective diarrhoea. This study was also conducted in a district general hospital setting.

We found that a subgroup of patients, 16 out of 29 with idiopathic bile acid malabsorption after excluding the known causes of bile acid malabsorption, dated their diarrhoea back to an episode of infective gastroenteritis. The time period ranged from about 3 months to 18 years. Interestingly the response to cholestyramine was seen in all but one patient. In their study, Bardhan *et al* rightly emphasise the significance of bile acid malabsorption in so-called diarrhoea predominant irritable bowel syndrome. However, they have not looked at the association of gastroenteritis and bile acid malabsorption as we have published before. In our practice now, enquiring about an episode of gastroenteritis just prior to the onset of diarrhoea is an important part of history taking in patients referred for investigation of chronic diarrhoea. This I feel is quite useful as a trial of cholestyramine where <sup>75</sup>ScHCAAT scan is not available and can be useful both diagnostically as well as therapeutically.

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