Guidelines for specialist training in cardiology

Sir,-I strongly endorse the view of your editorial 
that the publication of guidelines for the training of cardiologists should be welcomed. These guidelines suggest that competence at temporary cardiac pacing should be established during the first 5 years of higher professional training and that a minimum of 25 procedures be performed. This recognises the fact that temporary cardiac pacing can be technically difficult and that serious complications sometimes arise.

In practice, however, temporary pacing is a procedure that is learned by senior house officers (SHOs) undergoing general professional training. In a recent survey, 81% had learned temporary cardiac pacing at SHO level and teaching was provided primarily by medical registrars and fellow SHOs. A median of two procedures had been performed under supervision before the SHO was left to perform temporary cardiac pacing unsupervised.

Problems and complications with temporary cardiac pacing are frequent. This partly reflects the inexperience of junior medical staff who largely provide this service. The primary aim in providing guidelines for specialist training in cardiology must be to provide a better-cardiological service, through raising the standards of individual trainees. The problems with temporary cardiac pacing will not be overcome by any approach.

Training in temporary cardiac pacing must form part of general professional training and the British Cardiac Society should press the Royal College of Physicians to establish guidelines. The “see one, do one, teach one” approach to invasive procedures is no longer acceptable. Formal teaching could be provided within tutorials, by using training videos or mannequins, and a minimum number of procedures performed under supervision should be specified. Without this approach, the complications of temporary transvenous cardiac pacing will remain unacceptably high.

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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).


This letter was shown to the authors, one of whom replies as follows:

Sir,-I read with interest the suggestion from Dr Partridge that there should have been a greater emphasis on the physics and technical aspects of angiocardiography in the guidelines for training in cardiology. It has been pointed out that physics revision would be addressed during courses on radiation protection. It is accepted that cardiologists in training should understand the technical aspects of any equipment over which they have control, particularly equipment that is expensive and potentially hazardous. A contribution on this topic would be welcome if the guidelines are revised.

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Warm blood cardioplegia

Sir,-We read with interest the commentary by Youhana on warm blood cardioplegia and would like to clarify some of the issues that were raised.

Youhana implies that continuous normothermic blood cardioplegia (CNBC) has unanimously been shown to be superior to standard hypothermic techniques. Though Lichtenstein et al showed that mortality was reduced when CNBC was used in patients with long cross clamp times and after recent myocardial infarction, others found no difference in mortality between warm or cold cardioplegic techniques in patients undergoing urgent or emergency revascularisation. Furthermore, many of these studies are flawed by the use of retrospective controls to represent the hypothermic groups. The largest randomised study to date compared warm cardioplegic techniques in 1732 patients and showed no significant difference in mortality or the incidence of non-fatal Q wave infarction between the groups. Therefore, we suggest that currently there is no convincing evidence that overall clinical outcome is improved by the use of CNBC.

The commentary fails to address the important issue of adequate delivery of cardioplegia when warm blood cardioplegia is used in patients undergoing myocardial protection. Evidence from experimental models suggests that efficient delivery of cardioplegia may be far more important than the temperature of the solution used. In pigs antegrade warm blood cardioplegia resulted in reduced regional and global left ventricular function and increased necrosis compared with retrograde after left anterior descending artery occlusion and reperfusion. Though surgeons using cold blood cardioplegia can take comfort in the knowledge that they do not compromise myocardial protection by using a technique employing intermittent periods of ischaemia, those who advocate warm blood cardioplegic techniques must beware of inadequate delivery of cardioplegia in the face of coronary vascular disease.
It is also important to distinguish between the effects of the "warm heart"—that is, warm blood cardioplegia—and the "warm body" perfusion of normothermic cardiopulmonary bypass—because many reported studies have combined both techniques. The commentary suggests that "there is less postoperative bleeding in patients who have NCBC". This may not be a consequence of the use of NCBC in itself, but rather of the associated use of normothermic cardiopulmonary bypass. The effects of systemic hypothermia on platelet function and clotting factors are well established, and a more likely explanation of improved coagulation after normothermic perfusion than the effects of acid shifts on protamine activity.

Youhana cites Wong et al who studied the effects of "normothermic" cardiopulmonary bypass and who found that changes in neuro-psychological function were no worse than after hypothermic bypass. However, this study included 38 patients only and those in the "normothermic" group were in fact subjected to a degree of mild hypothermia (mean temperature 34-7°C). A much larger study by Martin et al in a subset of 150 patients randomised into either a normothermic or a hypothermic group found no statistical difference in postoperative neuropsychological dysfunction. Nevertheless, in the larger part of this study in which over 1000 patients were randomised, the incidence of stroke was found to be significantly higher after normothermic perfusion (warm 3-1%, cold 1-0%). This study is limited, however, by the inconsistencies in the use of cardioplegic techniques between the two groups. Retrograde warm blood cardioplegia was used in the warm group, whereas the hypothermic group received cold antegrade crystalloid cardioplegia, thus introducing another confounding variable. Becker et al argue that the potential for flushing debris from the native coronary arteries and into the aorta by retrograde cardioplegia, may result in embolic events after removal of the cross clamp. Furthermore, the relative hyperglycaemia associated with blood cardioplegia, along with the systemic vascular resistance11 and the more frequent use of vasocostrictors12 during normothermic perfusion, may all contribute to the increased risk of cerebral damage to the warm, metabolically active brain.

In summary, the implications of normothermia during open heart procedures have been confused by inconsistent terminology, differences in the number of variables altered between groups, and a host of non-randomised studies of limited value. The relative influences of normothermic myocardial protection, and normothermic systemic perfusion after open heart surgery still needs to be evaluated by randomised controlled trials in which the only variable is either cardioplegia temperature or systemic perfusion temperature. Until this happens, the "normothermic" myocardium must be viewed with cautious enthusiasm.

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5 Kavanagh B, Mazer D, Panos A, Lichtenstein S. Effect of prophylactic perioperative management of platelet function and clotting factors on incidence of low cardiac output syndrome, reflecting immediate postoperative cardiac function and the need for inotropic support was significantly reduced in the warm group, indicating superior myocardial protection in the warm group.
6 The issue of adequate cardioplegic delivery when warm blood cardioplegia is used is clearly stated in the commentary as being one of the problems related to this technique and not the result of the use of a hypothermic cardioplegic solution. This is especially important in severe three vessel disease and hypertrophied myocardium.
7 Birdi et al quote Martin and associates study as showing significantly higher rates of stroke in the normothermic perfusion groups. I would like to emphasise two important points about this study. First, many studies about coronary artery surgery report that predictors of perioperative stroke include age, left ventricular function, and extent of peripheral vascular disease, particularly carotid artery disease. Martin and associates did not include peripheral vascular disease in their study, despite its central importance to the rate of functional outcome, and Martin et al's study is limited by inconsistent use of cardioplegic techniques, as stated by Birdi et al, and the fact that the temperature in the warm group was not normothermic—in some it was as low as 35°C.
8 Birdi et al state that vasocostrictors, which are more likely to be given during warm perfusion, are a possible cause of the increased rate of neurological complications in the group of patients, patients may be affected, unrelated because the catecholamines are most commonly used in this setting act on a receptors in the artery wall. Cerebral and coronary arteries have few a receptors, and thus are spared the vasocostrictive effect of these drugs.
9 I agree with Birdi et al that the terms "warm blood cardioplegia" and "warm body perfusion" have become confused. This is not because the terms are synonymous. The two, and the two terms have more or less synonymous. I also agree that many factors contribute to the reduced blood loss in CNBC patients. One, mentioned by Birdi et al, is the use of on cardiopulmonary bypass machine. Another is the ablation of rebound heparin activity that occurs in hypothermic vasocostricted patients after warming up. In addition, changes are acid-base balance affect the activity of protamine.
10 I agree that further, prospective, randomised controlled trials are needed before this technique becomes widely practised.

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ACE inhibitors after myocardial infarction: patient selection or treatment for all?

Sir,—The question of selection criteria for angiotensin converting enzyme blockade after myocardial infarction (MI) posed by Lindsay et al.,1 needs to be set in the wider context of stratification of risk after MI according to therapeutic outcome.2 The latter strategy fully justifies the expense of radionuclide measurement of left ventricular ejection fraction (LVEF) because an LVEF < 40% has emerged as a modifiable risk factor for recurrence of MI (including MI related mortality),3,4 significantly surpassing even exercise induced segment depression5 in its prognostic accuracy. By analogy with the therapeutic outcome of coronary artery bypass and other poorly controlled chronic stable angina,6 the use of coronary angiography should also now supplant the exercise test in the risk stratification of patients with refractory angina after MI because, in the study cited above,6 reduction in mortality risk was more significantly correlated with the anatomical distribution of coronary atherosclerosis than with the electrocardiographic stigmata of the exercise test.

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1 Lindsay HSJ, Zaman AG, Cowan JC. ACE inhibitors after myocardial infarction: patient selection or treatment for all? Br Heart J 1995;73:397-400.
4 Micklely H, Fless P, Nielsen JR, Berning J, Moller M. Transient myocardial ischaemia after a first acute myocardial infarction and its relation to clinical characteristics, red blood cell exercise testing and cardiac events at one year follow up. Am J Cardiol 1993;71:139-44.
5 Myers MG, Bagirie RS, Charlat ML, Mogan CD. Serial exercise tests used in prediction of outcome after myocardial infarction in elderly people? Lancet 1993;342:609-72.

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

SIR,—Dr Jolobe argues for the widespread use of radionuclide ventriculography (RNVG) for risk stratification after myocardial infarction. The prognostic significance of a low ejection fraction has long been recognised but its value has been limited by our inability to modify this risk. The results of the SAVE study1 have changed this by showing that ACE inhibition can modify prognosis in patients with a low ejection fraction. In SAVE ventricular function was assessed relatively late at a median of 11 days post-infarction. However, data from GISSI-3 (Gruppo Italiano per lo Studio della Streptochinasi nell’Infarto Miocardico) show that much of the benefit of selected ACE inhibition occurs in the first few days after infarction,2 suggesting that treatment needs to be started in the first days after infarction. Indeed this is one the main points in favour of Dr Coats’ argument for an unselected approach to ACE inhibitor therapy after infarction.3

Selecting treatment on the basis of ejection fraction in the first days after infarction poses several problems. In the SAVE investigation, ejection fraction was estimated by RNVG. Many hospitals in the United Kingdom would have difficulty in routinely assessing left ventricular function by this method within the first 72 hours of infarction. Proponents of echocardiography would argue that it is an adequate substitute for RNVG with the advantage that it is readily performed at the bedside, that it is widely available, and that it is comparatively cheap. However, it is questionable whether echocardiographic assessment of ejection fraction is comparable to RNVG4 and this makes it difficult to choose a cut off value below which ACE inhibition should be started.

Moreover, the role of ejection fraction in selecting treatment in the first days after infarction is uncertain. There are few published reports on changes in ejection fraction in the early days after infarction but it seems that there is some recovery in ejection fraction within the first week.5,6 This makes it difficult to interpret the significance of the ejection fraction measured within the first few days. We have found echocardiographic ejection fraction assessed on day 3 to be a poor predictor of composite cardiovascular event rates.5 Hence, it is unsurprising and uninformative that in a trial that involved only 112 patients White et al found no significant effect of fibrinolytic therapy on this composite outcome.7 The use of the composite measure seems particularly inappropriate since deaths, which are the one component of it that might reasonably be expected from other settings to be reduced by fibrinolytic therapy, made only a small contribution (four deaths, as against 84 other events).

It has been suggested that composite outcome measures might, by increasing the number of events in a study of a given size, allow the reliable assessment of treatment without increasing the sample size.8 But this is not the case when the treatment under investigation does not actually influence some common component of a composite measure (or, worse still, produces effects in an opposite direction to those of other components: as with the proposed combination of death and haemorrhagic stroke for fibrinolytic therapy). Hence, although the trial by White et al was restricted to patients with ST depression, its composite analysis does not provide useful evidence about the efficacy of