LETTERS TO THE EDITOR

Survival after cardiac arrest outside hospital

EDITOR,—Most fatal events in patients with ischaemic heart disease occur outside hospital and therefore the greatest opportunities for reducing mortality from acute coronary events lie in the prehospital setting[1]. In their recent paper Soo and colleagues[2] published the results of a study to determine whether survival after cardiac arrest outside hospital was influenced by the availability of different grades of ambulance personnel and other health professionals. We are concerned with their conclusions about technician-only crews, and we wish to make some more general comments about their paper.

We feel that the data presented by Soo *et al* did not support their statement that "provision of defibrillation plus basic life support by technicians appears to be inadequate compared with the complementary early provision of advanced cardiac life support by paramedics". Clearly, in the population studied, overall survival was worse with technician-only crews than with paramedic crews. However, as mentioned by Soo *et al*, technician-only crews dealt with patients whose chances of survival were already prejudiced by several adverse factors—they were less likely to have had a witnessed arrest, bystander cardiopulmonary resuscitation, and an initial rhythm of ventricular fibrillation. It is interesting that among patients with ventricular fibrillation the proportion discharged home alive was higher for technician-only crews than for paramedic crews (10.9% *v* 10.5%). Viewed from the perspective of survival from ventricular fibrillation (the presenting rhythm most commonly associated with survival) it is thus difficult to conclude that the service provided by technician-only crews was "inadequate" compared with paramedic crews.

The interventions that offer the greatest benefit to victims of cardiac arrest are immediate basic life support and early defibrillation.[3] Soo *et al* briefly mentioned possible strategies aimed at improving the chances of survival, including increasing the number of other resuscitation trained professionals able to provide defibrillation. To optimise access to early defibrillation we believe that the issue of alternative first responders deserves serious consideration. Restoration of circulation and survival depends on the rapidity of defibrillation, regardless of who delivers the shocks, and even small differences in the call to shock time have an influence on survival.[4] The fire and police service have already taken on this role in some parts of the UK and others have expressed an interest in supporting the ambulance service as first responders.[5]

Finally, are Soo *et al* aware of a similar paper[6] from their institution (containing a common set of patients) that concluded that any survival advantages in victims of cardiac arrest associated with paramedic care were short term and diminished over time? We feel this study should have been referenced by the authors.

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1 Norris RM, on behalf of the United Kingdom Heart Attack Study Collaborative Group. Fatality outside hospital from acute coronary events in three British health districts. *BMJ* 1998;**316**:1065–70.
2 Soo LH, Gray T, Young T, *et al*. Resuscitation from out-of-hospital cardiac arrest: is survival dependent on who is available at the scene? *Heart* 1999;**81**:47–52.
3 Absalom A, Bradley P, Soar J. Early access to defibrillation is key to survival. *BMJ* 1998;**317**: 119–20.
4 White RD, Hankins DG, Bugliosi TF. Seven years' experience with early defibrillation by police and paramedics in an emergency medical services system. *Resuscitation* 1998;**39**:145–51.
5 Porter KM, Allison KP. An integrated 999 response—a questionnaire study of fire and police service opinion in the UK. *Pre-hospital Immediate Care* 1998;**2**:130–1.
6 Nguyen-Van-Tam JS, Dove DF, Bradley MP, *et al*. Effectiveness of ambulance paramedics versus ambulance technicians in managing out of hospital cardiac arrest. *J Accid Emerg Med* 1997;**14**:142–8.

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

Drs Soar and Absalom have highlighted the dangers of interpreting results by just examining factors in isolation. We used multivariate analysis by logistic regression method to take into consideration all factors (including those mentioned by Soar and Absalom) identified in our study that might have contributed to survival chances. This technique is particularly useful when dealing with potential confounders or when assessing interactions between variables. As a result of adjusting for confounders and interactions, the odds ratios we reported do support our conclusions.

We were indeed aware of another paper from our institution[1] but we considered citation of the latter inappropriate. Sound observational studies require a defined population; this may be the entire population with a specific characteristic (in this case, resuscitation from out-of-hospital cardiac arrest) or a sample taken in some systematic but random fashion from this. The conclusions drawn by Nguyen-Van-Tam *et al* may well be compatible with the data they reported but their population was neither *entire* nor a *random sample*—such selective populations are a potential source of bias.[2] We are confident that we identified *all* resuscitation events in Nottinghamshire over a four year period. We chose to analyse and present the complete population, failing to account for just 3% of all patients (as our Utstein style template shows). The claim that the two papers have used "a common set of patients" is clearly wrong. We do not believe that it is possible to make comparisons between our study and that of Nguyen-Van-Tam *et al*.

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2 Weston C, Donnelly P. Management of cardiac arrest by ambulance technicians and paramedics. Studying only admissions is a source of potential bias [letter]. *BMJ* 1995;**311**:509.

Exercise four hour redistribution thallium-201 SPECT and exercise induced ST segment elevation in detecting viable myocardium in patients with acute MI

EDITOR,—Yamagishi *et al*, studying 37 patients within seven weeks of Q wave myocardial infarction (MI), found that exercise induced ST segment elevation was closely associated with the presence of viable myocardium in the infarct territory.[1] We also studied this in patients with previous MI and agree with the results[2 3]; however, viable myocardium may persist for a long time after an MI,[4] and in these cases ST segment shift is not considered a specific indicator of transmural ischaemia and viability.

To increase the specificity of this sign in patients with an old (> 6 months) MI, we introduced an unconventional, but experimentally validated,[5] ECG marker of transmural ischaemia—the stress induced shortening of QTc interval (QT interval corrected for heart rate using Bazett's formula) in Q wave leads—to identify hibernating myocardium in the infarct zone. Experimental studies demonstrated an increase in cellular K^+ efflux at the onset of myocardial ischaemia accompanied by a progressive shortening of the action potential duration.[5]

We evaluated 15 consecutive patients (group A) with previous anterior MI presenting with the following: ST segment elevation over Q waves during exercise testing; critical stenosis (≥ 75%) of the left anterior descending coronary artery (LAD); cross sectional echocardiography and stress–redistribution–reinjection ^{201}Tl myocardial scintigraphy of viable myocardium in the infarct zone (akinetic segments with normal echoreflectivity plus ≥ 7mm end diastolic wall thickness and significant ^{201}Tl redistribution after reinjection (> 50% of the reference myocardium in any scan[6])).

The control group (group B) comprised 15 patients with previous anterior MI, critical stenosis of the LAD, and evidence of scar (increased echoreflectivity, associated to < 6 mm end diastolic wall thickness, and no ^{201}Tl redistribution) in infarcted areas.

Groups A and B were patients selected at random early or late (> 6 months) after their first anterior MI.

QTc interval was measured at rest and peak stress in leads showing ST segment shift, and the lead by lead fractional difference between the QTc intervals (ΔQTc) was calculated. The ΔQTc was measured again during exercise testing in 11 patients of group A (group A1) who had significant contractility recovery in akinetic areas (83% of akinetic segments) three months after myocardial revascularisation. We considered significant QTc interval shortening as ΔQTc < −10%. Data are presented as mean (SD).

There was no significant difference between patients in group A, B, and A1 (before and after revascularisation) regarding age, sex, number of pathological Q waves in resting ECG, exercise duration, exercise induced maximal workload, maximal heart rate, peak blood pressure, or maximal rate–pressure product.

ST segment elevation over Q waves at rest was higher in group B than in group A (1.8 (0.5) *v* 0.57 (0.4) mm) ($p < 0.001$).

All groups had exercise induced ST segment elevation over Q waves, but maximal elevation was significantly higher in group A than group B (2.5 (1.4) *v* 1.8 (1.1) mm)

($p < 0.05$) and in patients of group A1 before revascularisation (3 (1.03) *v* 1.1 (0.1) mm) ($p < 0.05$).

ΔQTc was significantly shorter in group A than group B (−18.1 (8.5) *v* −4.2 (7.8)%) ($p < 0.0001$). Indeed a significant ΔQTc shortening was measured in 14 of 15 patients of group A and only in one of group B (sensitivity 93.3%; specificity 93.3%; $p < 0.0001$). No group A1 patient had significant ΔQTc shortening in Q wave leads after revascularisation (ΔQTc of group A1 after revascularisation was +6.9 (14.8)%).

ΔQTc shortening in Q wave leads presenting exercise induced ST segment elevation, was a "cheap" ECG marker of transmural ischaemia and, indirectly, of myocardial viability as defined by echocardiographic and radionuclide variables, and confirmed by the results of revascularisation. This sign was no more evident after complete revascularisation and could be helpful in identifying hibernating myocardium even late after an MI.

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1 Yamagishi H, Akioka K, Takagi M, *et al*. Exercise four hour redistribution thallium-201 single photon emission computed tomography and exercise induced ST segment elevation in detecting the viable myocardium in patients with acute myocardial infarction. *Heart* 1999;**81**:17–24.
2 Bertella M, Scalise F, Lanzone AM, *et al*. Exercise standards [letter]. *Circulation* 1995;**92**: 3579–80.
3 Bertella M, Scalise F, Lanzone AM, *et al*. Is the shortening of the QTc interval in Q-wave leads showing ST-segment shift during exercise testing a new ECG marker of myocardial ischemia and viability in patients with previous myocardial infarction? *G Ital Cardiol* 1999;**29**:647–57.
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6 Dilsizian V, Freedman NMT, Bachach SL, *et al*. Regional Tl uptake in irreversible defects: magnitude of change in Tl activity after reinjection distinguishes viable from non viable myocardium. *Circulation* 1992;**85**:627–34.

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

We are pleased that Bertella and colleagues observed results similar to ours regarding the diagnostic significance of exercise induced ST segment elevation in detecting viable myocardium in MI patients. We reported that exercise induced ST segment elevation could detect the viable myocardium in the infarct region with high sensitivity and specificity, especially in patients with acute MI. However, in patients with old MIs and reduced left ventricular function, profound and possibly irreversible ultrastructural changes might occur in areas of hibernation, such as loss of contractile protein.[1] Such myocardial damage might affect the diagnostic accuracy of exercise induced ST segment elevation in detecting myocardial viability.

Bertella *et al* introduced a new ECG marker of hibernating myocardium in chronic MI—the exercise induced shortening of QTc interval in Q wave leads—to increase the specificity of exercise induced ST segment elevation. We are interested in this novel marker; however, how many leads with Q wave were analysed? QT dispersion significantly increases during ischaemia in coronary occlusion[2–4] and exercise stress testing.[5] Brief ischaemia does not change maximum QT, but shortens minimum QT.[4] To understand their results we need to know which leads were selected for analysis. It might be the most sensitive way for detecting myocardial ischaemia to select the lead with the greatest decrease in QTc. Moreover, exercise induced increase in QT dispersion could be a more sensitive marker.

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2 Russel DC, Oliver MF, Wojtczak J. Combined electrophysiological techniques for assessment of the cellular basis of early ventricular arrhythmias. *Lancet* 1977;ii:686–8.
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4 Michelucci A, Padeletti L, Frati M, *et al*. Effects of ischemia and reperfusion on QT dispersion during coronary angioplasty. *PACE* 1996;**19**: 1905–8.
5 Stoletniy LN, Pai RG. Value of QT dispersion in the interpretation of exercise stress test in women. *Circulation* 1997;**96**:904–10.

Quality of life four years after myocardial infarction: short form 36 scores compared with a normal population

EDITOR,—Brown *et al* compared the quality of life of patients after myocardial infarction with age and sex adjusted population norms from Oxford (age < 65 years) and Sheffield (age > 65 years).[1] This takes no account of social class or place of residence, which are known to influence health profile results.[2] Why not use controls and patients from the same community? Also, a comparison of the change in physical functioning score between the two age ranges shows a much greater fall in the controls (24.65 *v* 12.06). This suggests that the Oxford and Sheffield norms are not comparable and therefore confounds any attempt to make inferences by age group. The eight (short form) SF-36 scales can be summarised into physical and mental components, which are standardised to a mean score of 50, the population norm.[3] This allows interpretation of the quality of life of patients in relation to a general population and has been validated for the UK version of the SF-36.[4] Surely this is preferable, and more clinically meaningful, to using something as obscure as principal components analysis, which few readers are likely to understand.

Patients who have had a myocardial infarction commonly have a cluster of coronary risk factors such as diabetes mellitus, hypertension, and obesity. Furthermore, atherosclerosis is a systemic disease with many manifestations, and these patients may also suffer from other smoking related conditions. This total burden of illness is likely to have a profound effect on their health profile, swamping the contribution of any single condition.[5] Brown *et al* provide no detailed information on comorbidities, instead focusing on their patients' treatment, which may be an inadequate surrogate. Additionally, employment status is often considered a measure of health related quality of life for men of working age, but failure to return to work can be a cause as well as a consequence of declining health. How many of their patients who were initially employed were still working at follow up?

What was Cronbach's α for each scale? This coefficient assesses reliability by looking at the internal consistency of item responses and is an important measure of data quality.[6] The UK SF-36 has a few ambiguously worded items and suffers from floor and ceiling effects in the two role performance scales. The improved UK SF-36 version 2 has eliminated these problems but was not used by Brown *et al*. Healthy survivor and volunteer effects clearly made the study patients unrepresentative of the initial group. In addition, there is evidence that, as patients come to terms with chronic illness, psychological adaptation occurs such that patients can consider their quality of life as good even when severely limited.[6] An earlier administration of the instrument would have mitigated these problems. Finally, the SF-36 health profile adopts a fixed format "medical model" of health related quality of life. Newer questionnaires, such as the schedule for the evaluation of individual quality of life (SEIQoL), take account of individual patient preferences and priorities, and may therefore improve the appropriateness and responsiveness of these outcome measures. Concurrent administration of the SEIQoL, rather than the Nottingham health profile, would have been more interesting and informative.

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1 Brown N, Melville M, Gray D, *et al*. Quality of life four years after acute myocardial infarction: short form 36 scores compared with a normal population. *Heart* 1999;**81**:352–8.
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4 Jenkinson C, Layte R, Lawrence K. Development and testing of the medical outcomes study 36-item short form health survey summary scale scores in the United Kingdom. *Med Care* 1997;**35**:410–16.
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6 Mayou R, Bryant B. Quality of life in cardiovascular disease. *Br Heart J* 1993;**69**:460–6.

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

Quality of life issues and their measurement are rightly assuming an increasingly important role in health outcomes; however, they are relatively new, not without limitation, and are subject to continuing modification. As Mazeika points out, social class and place of residence can influence health profile results. Even so, assigning social class to housewives and the retired, for example, can be difficult and recent evidence suggests that ecological data are prone to error.[1]

We do not share Mazeika's concerns over the use of normative data from two cities in the UK. We had intended to use controls from the same community but we rejected this as the logistics of generating a potential list of age and sex matched "historical" controls four years later for a cohort that was

initially assembled in 1992 were enormous. The regional differences between younger patients from these cities are small and not of the order of magnitude suggested by the designers of the tool as significant. Mazeika's interpretation of the change in physical functioning scores between controls compared to patients is feasible, but we believe that four year survivors of myocardial infarction over age 65 have a quality of life similar to their peers. This may be due either to increasing comorbidity with age or to reduced expectations in the elderly "norms" as we originally discussed.

Patients with atherosclerotic disease may indeed have significant comorbidity and we did attempt to measure this, albeit using surrogates. Approximately 16% of our cohort described their main physical limitation as non-cardiac. We discussed return to work in the original text and accept that it is influenced by economic, social, and personal factors. However, establishing causality in the relation between quality of life and ability to return to work is contentious.

It is ironic that Mazeika describes principal components analysis as "obscure" when this technique was used to standardise scores into the summary scales of physical and mental components of health which he recommends. The analysis of our data took place before validation data on the summary scale scores he cites were published. Because of space constraints, we omitted Cronbach's α, a measure of internal consistency, from the final draft of our paper. For items in the same domain, α exceeded the recommended value[2] of 0.8 for patients younger and older than 65 years, with the exception of the domain *mental health* in patients over 65 years where α was 0.74. All domains were significantly correlated with each other, with Spearman's correlation coefficients exceeding 0.3 for all domains as recommended in the SF-36 manual.

Mazeika expresses surprise that we did not use the improved UK SF-36 version 2.[3] Research projects take time to design, implement, analyse results, and finally undergo peer review and modification before publication. Our questionnaires were distributed in 1996, before the UK SF-36 version 2 was developed. At that time, the original SF-36 was recommended and considered the most appropriate tool for this type of study.

Mazeika states that "Healthy survivor and volunteer effects clearly made the study patients unrepresentative of the initial group". There is no suggestion in our paper that these four year survivors are representative of all patients with myocardial infarction. The purpose of our study was to describe medium to long term survivors, whether healthy or not. Survivorship may form part of the explanation for some of our findings in the elderly, nevertheless younger patients' demonstrably poor quality of life is hardly likely to be described as "healthy survival".

Following our experience with quality of life tools, we believe that the combination of a *disease specific* tool, such as the quality of life after myocardial infarction instrument,[5] or perhaps the schedule for the evaluation of individual quality of life (a new, patient weighted measure, not without limitation) and a *generic* tool such as the SF-36 may well offer a more complete assessment of the impact of illness and comorbidity on health related quality of life. Even so, the SF-36 did provide us with evidence that a myocardial infarction makes a young man feel old and an old man feel a bit older. Perhaps most important, Mazeika seems to be missing the essential point of our paper: the quality of life of infarct *survivors* younger than 65 is significantly impaired four years after their acute illness.

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5 Heller RF, Lim L, Valenti L, *et al*. Predictors of quality of life after hospital admission for heart attack or angina. *Int J Cardiol* 1997;**59**:161–6.

Exercise testing, symptoms, and clinical outcome in aortic stenosis

EDITOR,—We read the recent editorials on aortic stenosis with interest.[1 2] Otto rightly highlights the importance of classifying patients with aortic stenosis into those at risk of future clinical events. Earlier studies on the natural history of aortic stenosis have shown that patients with symptomatic aortic stenosis have a very poor prognosis.[3] The difficulty arises in classifying patients with asymptomatic aortic stenosis as they are generally considered to be at low risk of future events, even in the presence of severe disease. Otto has suggested defining severe aortic stenosis as a peak jet velocity > 4 m/s as "about 80% of asymptomatic patients with a jet velocity > 4 m/s will develop symptoms requiring valve replacement within two years".[1] This statement is not strictly correct. Although almost 80% of these patients did indeed have aortic valve replacement carried out within two years, the most common reason for valve replacement was reduced exercise tolerance.[3] Having reduced exercise capacity does not mean patients are symptomatic per se and, although it is a fine point, it is of critical importance. We do not know whether reduced exercise capacity in aortic stenosis is an independent predictor of outcome, and Otto's study did not address this question. In a previous study on asymptomatic aortic stenosis it was deemed unethical to withhold exercise testing results from the primary care physicians of the patients concerned, despite the fact there is no evidence in adults to support reduced exercise capacity as a predictor of clinical outcome. In Otto *et al*'s study, of 48 patients undergoing aortic valve replacement, 18 had reduced exercise time stated as the primary reason for surgery.[4] This proportion is even higher when patients with severe asymptomatic aortic stenosis and those having incidental valve replacement at the time of coronary artery bypass surgery are excluded. These data clearly show that the primary care physicians were influenced by the results of the exercise tolerance testing and may invalidate Otto's use of a jet velocity of 4 m/s as a predictor of clinical outcome.

Chambers stated that "if chest tightness develops, it is reasonable to prepare for aortic valve replacement".[2] We do not agree that angina confers additional prognostic information compared to other symptoms. In Ross and Braunwald's classic study on aortic stenosis, angina was shown to have a relatively good prognosis compared to symptoms of breathlessness, heart failure, and syncope.[3] It is also difficult to distinguish whether chest pain is a result of severe aortic stenosis or underlying coronary artery disease, as approximately 50% of aortic stenosis patients requiring valve replacement will have significant obstructive coronary artery disease.[3]

We do agree that exercise testing in aortic stenosis confers additional valuable information regarding patients' functional status; however, whether it confers added prognostic significance is not known. Prospective blinded studies on the results of exercise tolerance testing are required before surgery is recommended on this basis in addition to currently accepted echocardiographic and symptomatic variables.

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1 Otto CM. The difficulties in assessing patients with moderate aortic stenosis [editorial]. *Heart* 1999;**82**:5–6.
2 Chambers J. Exercise testing to guide surgery in aortic stenosis [editorial]. *Heart* 1999;**82**:7–8.
3 Ross J Jr, Braunwald E. Aortic stenosis. *Circulation* 1968;**37–38**(suppl V):V61–7.
4 Otto CM, Burwash IG, Legget ME, *et al*. Prospective study of asymptomatic valvular aortic stenosis. *Circulation* 1997;**95**:2262–70.

Value of echocardiography in predicting long term outcome after heart transplantation

EDITOR,—The recent study by Fraund *et al* has highlighted the improving survival of cardiac transplant recipients with a 10 year survival rate of approaching 50%.[1] Functional status in long term survivors was encouraging with fewer than one in five patients experiencing (New York Heart Association) NYHA class III or IV symptoms. These findings reinforce cardiac transplantation as a valuable treatment option for patients with symptomatic severe left ventricular systolic dysfunction.

Disappointingly, the authors were unable to identify any useful factors that potentially could be used to predict long term outcome. Allograft vasculopathy is now emerging as the main factor limiting long term survival, and 39% of all deaths in the study were attributable to this complication. Angiographic screening programmes for the detection of allograft vasculopathy have been instituted but, without the routine use of intravascular ultrasound techniques, coronary angiography has been shown systematically to underestimate this form of coronary disease. Furthermore, as no adequate therapeutic options currently exist, the value of screening that exposes many stable patients to the risk of an expensive invasive procedure has been questioned.[2] Clearly, a non-invasive method of identifying high risk patients would be highly desirable.

Echocardiography plays an important role in the follow up of recipients after cardiac transplantation but, other than assessment of left ventricular systolic function by ejection fraction, it did not feature in Fraund *et al*'s article. In the past efforts have focused specifically on the use of Doppler studies in detecting acute allograft rejection. Its clinical utility is now expanding. Recently, dob-

utamine stress echocardiography has been scrutinised for the detection of allograft vasculopathy. This method of assessment focuses on the functional significance of ischaemia rather than the specific coronary anatomy; therefore, it has introduced a new approach to the evaluation of recipients. Importantly, stress echocardiography has been shown to have a high negative predictive value for determining future cardiac events and death.[3] A major advantage is that it is non-invasive, but reservations exist regarding the potential for high interobserver variability, which could jeopardise the value of the information derived.

The importance of abnormalities of left ventricular diastolic function is now being appreciated. The presence of a restrictive pattern of left ventricular filling independently predicts an adverse outcome in patients with a range of conditions including acute myocardial infarction. In cardiac allografts, diastolic dysfunction has a multifactorial cause. Valentine *et al* have shown that recovery of diastolic function after allograft rejection may often be incomplete, with the development of restrictive physiology in a proportion of recipients characterised by an increase of left ventricular end diastolic pressure.[4] The histological appearance in these circumstances is one of myocyte loss and fibrous replacement. An irreversible decline in compliance may develop leading to chronically deranged diastolic function while systolic function may be preserved by hypertrophy of intact myocytes.

Cumulative myocardial damage leading to chronic diastolic dysfunction has important implications for the long term prognosis of heart transplant recipients.[5 6] Those with restrictive physiology are significantly more likely to experience NYHA class III or IV symptoms.[4] Ross *et al* have shown that preservation of normal Doppler parameters of diastolic function in the early post-transplantation period confers a significant actuarial survival advantage, which is independent of the influence of other factors such as allograft vasculopathy.[5]

In experienced hands both resting Doppler and dobutamine stress echocardiography allow the non-invasive identification of heart transplant recipients at high risk of an adverse outcome. Whether this group will benefit from more aggressive treatment and careful follow up remains to be determined.

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1 Fraund S, Pethig K, Franke U, *et al*. Ten year survival after heart transplantation: palliative procedure or successful long term treatment? *Heart* 1999;**82**:47–51.
2 Grant SCD, Brooks NH, Levy RD. Routine coronary angiography after heart transplantation. *Heart* 1997;**77**:101–2.
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5 Ross HJ, Gullestad L, Hunt SA, *et al*. Early Doppler echocardiographic dysfunction is associated with an increased mortality after orthotopic cardiac transplantation. *Circulation* 1996;**94**(suppl II):289–93.
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Good outcomes from cardiac surgery in the over 70s

EDITOR,—We read with interest the manuscript by Zaidi and coworkers who reported their experience on 575 patients aged 70 years and older, in whom various cardiac surgical procedures were performed.[1] Their study, along with many others published in the literature in recent years,[2–4] shows that the role of cardiac surgery in the elderly, and particularly coronary artery revascularisation, is still evolving. Despite refinements in the perioperative management of cardiac surgical patients, valve surgery as well as coronary artery revascularisation in the elderly continue to be associated with operative risks considerably greater than those observed in the younger population.[2–4] In addition, older patients are at higher risk for developing devastating complications such as stroke, which often lead to long term disability. In this regard, Hogue *et al* in a recent review of 2972 patients aged 65 years and older subjected to a variety of cardiac operations, reported that although age was not an independent risk factor for perioperative stroke, other significant independent factors (such as previous neurological events, carotid artery stenosis, aortic atherosclerosis, and diabetes) were more frequently encountered in older patients.[5] Based on these consideration, arguments have been made as to whether these therapeutic modalities should be offered to elderly high risk patients who, as a result of their advanced age and comorbid conditions, invariably face reduced life expectancies.

Of the 575 elderly patients included in Zaidi *et al*'s series, 334 underwent isolated coronary artery bypass grafting (CABG). Importantly, the perioperative outcomes noted in these patients compare favourably with, and are actually lower than, those reported in the literature. In fact, they reported a 30 day mortality rate of 3.9% in patients who received isolated CABG. Accordingly, the incidence of postoperative neurological events in CABG patients was remarkably low (1.8%), as was the incidence of other complications, such as renal failure, reoperation, myocardial infarction, and low output syndrome requiring an intra-aortic balloon pump. The proportion of redo operations reported was 7.3% in the two groups combined (valve patients and CABG patients).

We have recently reviewed our experience of coronary revascularisation in more than 450 patients aged 70 years and older, in whom the operation was conducted without using cardiopulmonary bypass ("off pump"). In recent years, advances in techniques of cardiac elevation in combination with adequate exposure of all target vessels and mechanical stabilisation, have made coronary revascularisation on the beating heart safe and effective. Importantly, more than 22% of the patients were octogenarians. Our analysis revealed a 30 day mortality rate of 4.8% (risk adjusted < 2%), along with an overall rate of postoperative complications of 12%, including a stroke rate of 2.1%. None of the patients in the octogenarian subgroup suffered postoperative neurological events. In our study, however, these figures were obtained in the face of a substantial proportion of redo operations (28% of cases), in combination with a 47% rate of urgent and emergent procedures, and a 31% incidence of preoperative cerebrovascular disease. Based on these results, in agreement with Zaidi and colleagues, we believe that elderly patients with surgically correctable, symptomatic coronary occlusive disease should not be denied surgical intervention based solely on their age. Our preliminary data suggest that myocardial revascularisation without using cardiopulmonary bypass may offer these patients an advantage in terms of perioperative outcomes, especially in the presence of meaningful perioperative risk factors, such as those associated with redo operations. Finally, avoiding extracorporeal circulation seems to provide additional benefit in lowering the rate of postoperative stroke, which remains one of the most debilitating sequelae following coronary revascularisation in the elderly.

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NOTICES

Patho2000, the 20th annual San Diego cardiothoracic surgery symposium, will be held 10 to 13 February 2000 in San Diego, California, USA.

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Third acute medical emergencies conference, will take place on 21 and 22 February 2000 at the Royal College of Physicians in London, UK.

The programme covers initial treatment of acute medical emergencies in cardiology, neurology, gastroenterology, and respiratory medicine.

For more details please contact the Secretariat, CCI Limited, 2 Palmerston Court, Palmerston Way, London SW8 4AJ, UK; tel: +44 (0)20 7720 0600; Fax: +44 (0)20 7720 7177; email: AME2000@confcomm.co.uk; www.confcomm.demon.co.uk/AME2000/AMEHome.html