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. In their recent paper Soo and colleagues 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 (21.9% v 10.5%) 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 “adequate” compared with paramedic crews.

The interventions that offer the greatest benefit in cardiac arrest are immediate basic life support and early defibrillation. 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 may have an influence on survival. Fire and police service have already taken many interventions—such selective populations are a danger of interpreting results by just examining the survival advantage 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 six British health districts. BMJ 1998;316:1065–70.
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.

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

Dr 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 institution1 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 data from the complete population, failing to account for just 3% of all patients (as our Ustien 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.

J SOAR


Exercise four hour redistribution through Tl-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.3 We also studied this in patients with previous MI and agree with the results;4 however, viable myocardium may persist for a long time after an MI,5 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, ECG marker of transmural ischaemia—the 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.6 We evaluated 21 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 201Tl myocardial scintigraphy of viable myocardium in the infarct zone (akinetetic segments with normal echorereflectivity plus > 7mm end diastolic wall thickness and significant 201Tl redistribution after reinjection (> 50% of the reference myocardium in any scan)).

The control group (group B) comprised 15 patients with previous anterior MI, critical stenosis of the LAD, and evidence of scar (increased echoreactivity, associated to < 6 mm end diastolic wall thickness, and no 201Tl redistribution) in infarcted areas. Groups A and B were consecutively 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 (AQTC) was calculated. The AQTC was measured again during exercise testing in 11 patients of group A (group AI) who had significant contractility recovery in akinetic areas (83% of akinetic segments) three months after myocardial revascularisation. We considered significant QTc interval shortening as AQTC < −10%. Data were presented as mean ± SD.

There was no significant difference between patients in group A, B, and AI (before and after revascularisation) regarding age, sex, number of pathological Q waves in resting ECG, exercise duration and 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).

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2 We also studied this in patients with previous MI and agree with the results.
3 However, viable myocardium may persist for a long time after an MI, and in these cases ST segment shift is not considered a specific indicator of transmural ischaemia and viability.
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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 ± 12.06). This suggests that the Oxford and Sheffield norms are not comparable and the SF-36 scale founds 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

This was known 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 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 old MIs and patients with left ventricular function, profound and possibly irreversible ultrastructural changes might occur in areas of hibernation, such as loss of contractile protein. Such myocardial damage might limit 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 models.5 Briefly, ischaemia does not change maximum QT, but shortens minimum QT. 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.


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

We do not share Mazeika's concerns over 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 a 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 $u$ 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 function 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.7 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 short form 36 (SF 36), health survey questionnaire: normative data for adults of working age. BMY 1993:861437–40.

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 function 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 do not measure this, although 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 component 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 $\alpha$, a measure of internal consistency, from the final draft of our paper. For items in the same domain, we exceeded the recommended value 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 $\alpha = 0.74$. 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. Research projects take time to design, implement, analyse results, and finally undergo peer review and modification before publication. Indeed, the summary scales were published 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.

We note 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, 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.


Exercice 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 prior to 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”.4 This statement is not supported by the data. 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.5 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 clinical 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 as a means of recruiting 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.6 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 the 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”.7 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.8 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.

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 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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Value of echocardiography in predicting long term outcome after heart transplantation

EDITOR,—The recent study by Fraud et al has highlighted the improving survival of cardiac transplant recipients with a 10 year survival rate of approaching 50%.9 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 recognised 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, although most current revascularisation options currently exist, the value of screening that exposes many stable patients to the risk of an expensive invasive procedure has been questioned.10 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 and left ventricular fraction, it did not feature in Fraud 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-
Good outcomes from cardiac surgery in the over 70s

Burgess et al. We read with interest the manuscript by Zaidi and colleagues who reported their experience on 575 patients aged 70 years and older, in whom various cardiac surgical procedures were performed. Their study, along with other investigations published in the literature in recent years, 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. 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 70 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 neurological events, carotid artery stenosis, aortic atherosclerosis, and diabetes) were more frequently encountered in older patients. Based on these considerations, 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 better 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 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 surgery have been made in combination with adequate exposure of all target vessels and mechanical stabilisation, which 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 1%. The presence of diastolic dysfunction, which could jeopardise the value of the myocardium, 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 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, often with the development of restrictive physiology in a proportion of recipients characterised by an increase of left ventricular end diastolic pressure. The histological appearance in these hearts 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. Those with restrictive physiology are significantly more likely to experience NYHA class III or IV symptoms. Ross et al. have shown that presentation of normal Doppler parameters of diastolic function in the early post-transplantation period presages a significant actuarial survival advantage, which is independent of the influence of other factors such as allograft rejection. In experienced hands both restowing Doppler and dobutamine stress echocardiography allow the non-invasive identification of heart transplant recipients at high risk of an adverse outcome. Thus, this group will benefit from more aggressive treatment and careful follow up remains to be determined.

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REFERENCES


NOTICES

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

For further details please contact Lil Wagner, Aligned Management Associates Inc, tel: +858 541 1444; fax: +858 541 1447; email: llw213@ mindspring.com

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@confcom.co.uk; www.confcom.demon.co.uk/AME2000/AMEHome.html