LETTERS TO THE EDITOR

Myocardial infarction and left bundle branch block

EDITOR,—We congratulate Edhouse *et al*¹ on their attempt to try and produce some clarity in the murky waters of diagnosing and treating patients with acute myocardial infarction (AMI), who present with left bundle branch block (LBBB). Unfortunately, we feel that the study has some limitations that could provide for some confusing “take home” messages. There are four points to consider:

(1) The original article, Sgarbossa *et al*,² was published with an editorial and generated a number of subsequent letters,^{3,4} which were rightly critical of the cohort chosen and the subsequent extrapolation of results. These views are not adequately reviewed and the “spin” in the discussion by Edhouse *et al* in our opinion, is overly supportive of Sgarbossa’s criteria.

(2) The prevalence of AMI in Edhouse’s article is 0.5 and is unusually high for patients presenting to accident and emergency with cardiac sounding chest pain. The method section seems to suggest that these patients were derived from a database of patients eligible for thrombolysis, which would not be an appropriate study population. This is an important point that requires clarification by the authors.

(3) In the conclusion, the first sentence rightly points out the need for thrombolysing all patients with LBBB and persisting cardiac pain. The last sentence suggests serial ECGs. It is unclear which side of the fence the authors wish to sit on. The assertion that serial ECGs should be used if “the diagnosis is in doubt” is based upon five patients. We would suggest this recommendation is untenable on the evidence provided.

(4) Shlipak *et al*⁵ have published the most robust study thus far (with a more representative prevalence of 28% for AMI) for this group of patients. It is disappointing that this study is not reviewed in more detail by Edhouse *et al*. The result and conclusions in Shlipak’s paper invalidate using any of the Sgarbossa criteria for detection of AMI in the presence of LBBB. They also performed a decision analysis suggesting that an extra 10 to 12 lives per thousand patients could be saved by adhering to the American College of Cardiology/American Heart Association guidelines on the subject.⁶ These recommend that in the absence of contraindications, thrombolysis should be used in all patients with LBBB who have a clinical presentation of AMI.

Future research strategies using new technologies may herald greater precision and provide the solution for this difficult group of patients. These could include rapid bedside cardiac enzyme analyses, vectorcardiography and/or neural networks. In the meantime we should adhere to the best available evidence.

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- 1 Edhouse JA, Sakr M, Angus J, *et al*. Suspected myocardial infarction and left bundle branch block. Electrocardiographic indicators of acute ischaemia. *J Accid Emerg Med* 1999;16:331-5.
- 2 Sgarbossa EB, Pinski SL, Barbagelata A, *et al*. Electrocardiographic diagnoses of evolving acute myocardial infarction in the presence of left bundle branch block. *N Engl J Med* 1996;334:481-7.
- 3 Ackermann RJ, Vogel RL. Electrocardiographic diagnoses of acute myocardial infarction in the presence of left bundle branch block [letter]. *N Engl J Med* 1996;335:131-2.
- 4 Byrne J. Electrocardiographic diagnoses of acute myocardial infarction in the presence of left bundle branch block [letter]. *N Engl J Med* 1996;335:132-3.
- 5 Shlipak MG, Lyons WL, Go AS, *et al*. Should the electrocardiogram be used to guide therapy for patients with left bundle branch block and suspected myocardial infarction. *JAMA* 1999;281:714-19.
- 6 Ryan TJ, Anderson JL, Antman EM, *et al*. ACC/AHA guidelines for the management of patients with acute myocardial infarction. Executive summary. *Circulation* 1996;94:2341-50.

The authors reply

We welcome the opportunity to discuss the points raised by Mohammed *et al*. Our data include all patients with acute chest pain and left bundle branch block (LBBB) on presentation or within 12 hours of admission. Thus we have complete data on all patients with LBBB and acute myocardial infarction (AMI) during the study period.

Mohammed states that a 52% prevalence of AMI is unusually high, and claims the prevalence of 28% quoted by Shlipak is a “more representative” figure, but provides no evidence to support this assertion. Hands *et al*¹ found a prevalence of 57%, a figure very similar to our own. We note that Shlipak⁵ mistakenly attributes a prevalence of 25% to the study by Hands.

The recommendation that all patients with chest pain and LBBB receive thrombolysis is correct and evidence-based, but is also rather simplistic. This is reflected in the significant under-use of thrombolysis both in the UK³ and the United States,⁴ and the lengthy treatment delays these patients experience.³

At least half the patients with LBBB and chest pain are not infarcting.¹ While the overall mortality reduction justifies the administration of thrombolysis to all such patients,⁵ this does not make the decision to treat an individual patient any easier. It is hard to think of another circumstance in which we expose patients to a significant stroke risk on the strength of a diagnosis of which we are only 50% certain.

Management decisions are further complicated if the presentation is not classic, or if relative contraindications exist. The decision to administer thrombolysis, especially in the elderly population, is often a careful balancing act between potential benefit and complication risk. The manifest reluctance of doctors to expose patients to significant risk without a definite diagnosis is perfectly understandable, and merely reiterating the guidelines does not help the clinician at the sharp end.

We acknowledge the limitations of our small, retrospective study. Nevertheless, the differences between our results and those of Shlipak are remarkable. Ischaemic change evolves over time, even in the presence of LBBB.⁶ If only the presenting ECG is analysed, evolving changes will be missed and the sensitivity of the predictive criteria⁷ underestimated, particularly if patients present early in the course of their infarct.

Shlipak noted the ECG criteria infrequently²; in contrast we found at least one of the criteria in 19 of 24 patients with AMI.³ When we analysed only the first ECG, the

presence of any of the criteria indicated a diagnosis of AMI with sensitivity = 62.5%. A series of ECGs was available in only 33% of patients, but incorporating even this small number into the analysis increased the sensitivity of the criteria to 79% (specificity 100%).³ We note that in Shlipak’s study only the presenting ECG was analysed.²

Our unpublished data on 797 consecutive patients presenting with AMI revealed a median interval between onset of pain and arrival at hospital of 135 minutes, whereas audit data from the United States report a median of 89 minutes.⁴ A relatively early presentation, coupled with analysis of only the first ECG, may partly explain the low sensitivity found in Shlipak’s study.

Our findings strongly support those of Sgarbossa in showing that a significant number of infarcting patients can be identified quickly and counselled confidently regarding their need for thrombolysis.³ This is of immediate practical benefit to clinicians and should facilitate considerable reductions in treatment delays. Thrombolysis should also be recommended when the ECG criteria are absent, along with an individual risk benefit assessment to allow patients to participate in the management decision. Where patients do not receive immediate thrombolysis serial ECGs are essential; if evolving changes indicate a definite infarct, the balance of risk and benefit may change considerably.

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- 1 Hands ME, Cook EF, Stone PH, *et al*. Electrocardiographic diagnosis of myocardial infarction in the presence of complete left bundle branch block. *Am Heart J* 1988;116:23-30.
- 2 Shlipak MG, Lyons WL, Go AS, *et al*. Should the electrocardiogram be used to guide therapy for patients with left bundle-branch block and suspected myocardial infarction? *JAMA* 1999;281:714-19.
- 3 Edhouse JA, Sakr M, Angus J, *et al*. Suspected myocardial infarction and left bundle-branch block: electrocardiographic indicators of acute ischaemia. *J Accid Emerg Med* 1999;16:331-5.
- 4 Barron HV, Bowly LJ, Breen T, *et al*. Use of reperfusion therapy for acute myocardial infarction in the United States. Data from the National Registry of Myocardial Infarction 2. *Circulation* 1998;97:1150-6.
- 5 ISIS2 Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17 187 cases of suspected acute myocardial infarction: ISIS-2. ISIS-2 Collaborative Group. *Lancet* 1988;ii:349-60.
- 6 Fesmire FM. ECG diagnosis of acute myocardial infarction in the presence of left bundle-branch block in patients undergoing continuous ECG monitoring. *Ann Emerg Med* 1995;26:69-82.
- 7 Sgarbossa EB, Pinski SL, Barbagelata A, *et al*. The GUSTO-1 Investigators. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. *N Engl J Med* 1996;334:481-7.

Pedestrian injuries sustained in negotiating traffic calming measures

EDITOR,—We have recently seen two patients who were injured while walking across raised traffic calming features (sleeping policemen). The first patient, a 56 year old woman could not walk on the pavement as it was blocked by cars. In negotiating the traffic restraint she failed to clear the restraint and caught her foot, stumbling forwards cutting both of her

knees. Six months after the injury she has right anterior knee pain caused by the contusional injury. The second, a 60 year old woman, was walking across her road and walked across a parking restraint. She caught her foot and fell forwards onto her outstretched left hand. She sustained an undisplaced distal radial fracture requiring cast treatment.

Sleeping policemen are usually made of the same materials as the road and are usually gently sloping. However, they may be made of different material than the road with different frictional characteristics, they may be sharp, angular and low. It would seem that our patients both misjudged their foot clearance when walking. In both cases the restraints were curved and of the same material as the road, about six inches high. In one case a pavement could not be used. If the restraints are made of a different material to the road surface the friction characteristics may be different and cause difficulties in foot placement and lifting. If they are low and angular they may resemble the ubiquitous raised paving stone. We have heard that these restraints may be inconvenient for emergency traffic including ambulances.

Traffic restraints should be placed in well lit areas of the road, they should have the same frictional or material characteristics of the road, it may be reasonable to place warning notices to take care in crossing these obstructions.

We feel it unlikely that ours are the only injuries caused by this mechanism.

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Shock and ipsilateral pulmonary oedema

EDITOR,—I had a distinct *deja vu* phenomenon on reading the article by Desai and colleagues.¹

In 1970 (30 years ago) David Trapnell and I reported four patients with unilateral pulmonary oedema after pleural aspiration.² This paper described two patients with pneumothorax but also two with pleural effusions. One of the patients, an 18 year old, died.

The common feature of these four patients was that the air or fluid had occupied the pleural space for some time and were not acute presentations. We concluded "It is established practice in genito-urinary circles to decompress the bladder of a patient with chronic urinary retention slowly. Acute retention is relieved rapidly after insertion of a urethral catheter. We would like to suggest that the same principle of therapeutic relief be

applied to the pleural space". We believed that this important concept had not been reported previously in a group of patients and felt that medical practitioners treating chronic or relatively longstanding pleural effusions or pneumothoraces should be aware of the need to decompress the pleural space with caution.

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- 1 Desai AV, Phipps PR, Barnes DJ. Shock and ipsilateral pulmonary oedema after tube thoracostomy for spontaneous pneumothorax. *J Accid Emerg Med* 1999;16:454-5.
- 2 Trapnell DH, Thurston JGB. Unilateral pulmonary oedema after pleural aspiration. *Lancet* 1970;i:1367-9.

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