LETTERS TO THE EDITOR

Myocardial infarction and left bundle branch block

EDITOR,—We congratulate Edhouse et al 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. Sgarbossa et al, was published with an editorial and generated a number of subsequent letters, which were rightly critical of the cohort chosen and the subsequent extrapolation of results. These views were 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 (0.5%) is unusually high for patients presenting to accident and emergency with cardiac sounding chest pain. The method section suggests that in this group patients were asked if “the diagnosis is in doubt” based upon five patients. We would suggest this recommendation is untenable on the evidence provided.

3. The recommendation that all patients with chest pain and LBBB receive thrombolysis is made with no evidence-based, but is also rather simplistic. This is reflected in the significant under-use of thrombolysis both in the UK and the United States, and the lengthy treatment delay these patients experience. While the overall mortality reduction justifies the administration of thrombolysis to some patients, 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.

4. Schlipak et al 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. They also performed a decision analysis that requires clarification by the authors.

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 unusual high, and claims the prevalence of 28% quoted by Schlipak is a “more representative” figure, but provides no evidence to support this assertion. Hands et al. found a prevalence of 57%, a figure very similar to our own. We note that Schlipak’s mistaken attributes a prevalence of 25% to the study by Hands.

The recommendation that all patients with chest pain and LBBB receive thrombolysis is not evidence-based, and is also rather simplistic. This is reflected in the significant under-use of thrombolysis both in the UK and the United States, and the lengthy treatment delay these patients experience. While the overall mortality reduction justifies the administration of thrombolysis to some patients, 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 difficult 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 Sgarbossa et al are remarkable. Ischaemic change evolves over time, even in the presence of LBBB. If only the presenting ECG is analysed, evolving changes will be missed and the sensitivity of the predictive criteria will decrease, particularly if patients present early in the course of their infarct.

Schlipak noted the ECG criteria infrequently; in contrast we found at least one of the criteria in 19 of 24 patients with AMI. 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%).

We note that in Shlipak’s study only the presenting ECG was analysed.

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 reports a median of 89 minutes. 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. This is of immediate practical benefit to clinicians and should facilitate considerable reductions in treatment delays. Thrombolysis should also be recommended when relative contraindications 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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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 déjà 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.1 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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