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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).*

How do the clinical findings in patients with pericardial effusions influence the success of aspiration?

SIR,—Cooper and colleagues make a solid contribution to predicting the success of pericardial aspirations.[1] Although breathlessness is by no means universal among patients with cardiac tamponade, their quite original definition of tamponade is apt for the purposes of this paper: "a pericardial effusion causing dyspnoea which is relieved by aspiration". Moreover, their useful definition and relief of dyspnoea by drainage of effusions in patients without classic symptoms provide new evidence that cardiac tamponade is a continuum, not "all or none".[2] Indeed, even minimal effusions are not physiologically inert.[3]

My quarrel with Cooper *et al* is their promotion of Kussmaul's sign as being characteristic of tamponade: "a patient with tamponade typically (sic) develops a positive Kussmaul's sign" (p 353). Indeed, six of their patients were said to develop one.

Kussmaul's sign is in no way typical of cardiac tamponade—it does not develop in pure tamponade.[4] During pericardial effusion Kussmaul's sign is either the result of epicardial constriction underlying the tamponading fluid layer or of inaccurate observation of neck veins. I am sure that on reflection Cooper *et al* will recall this and perhaps amend their publication to this effect because less sophisticated readers may accept Kussmaul's sign as typifying cardiac tamponade.

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1 Cooper JP, Oliver RM, Currie P, Walter JM, Swanton RH. How do the clinical findings in patients with pericardial effusions influence the success of aspiration? *Br Heart J* 1995;73:351–4.
2 Reddy PS, Curtiss EI, O'Toole JD. Cardiac tamponade: Observation in man. *Circulation* 1976;58;265–70.
3 Spodick DH, Paladino D, Flessas AP. Respiratory effects on systolic time intervals during pericardial effusion. *Am J Cardiol* 1983;51;1033–5.
4 Spodick DH. Diseases of the pericardium. In: Parmley WW, Chatterjee K, eds. Cardiology. Lippencot: Philadelphia, 1992; vol 2, 1–34.

This letter was shown to the author, who replies as follows:

SIR,—I agree with Professor Spodick that a positive Kussmaul's sign is not typical of patients with *pure* cardiac tamponade. Measurement of intracardiac pressures and flow during experimental tamponade[1] and in humans during cardiac tamponade[2 3] showed that inspiration causes a decrease in intrapericardial and right atrial pressures. Thus the jugular venous pressure would be expected to fall, not rise, during inspiration in these patients.

However, a positive Kussmaul's sign was recorded in five of our patients (26%) in whom pericardiocentesis relieved symptoms of breathlessness.[4] This compares with six patients (30%) who were found to be hypotensive: a sign that is commonly thought of as an indicator of pure tamponade. Why Kussmaul's sign was present in the five patients is not clear. If epicardial constriction were the reason we might expect a correlation between pericardial loculation and a positive Kussmaul's sign. This was not the case.

By replying to Professor Spodick's comments about a specific physical sign I would not like to detract from the main message of our paper which was that physical signs may be unreliable as markers of a pericardial effusion causing symptoms and their absence does not preclude symptomatic benefit being derived from pericardial aspiration.

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1 Shabetai R, Fowler NO, Fenton JC, *et al.* Pulsus paradoxus. *J Clin Invest* 1965;**44**: 1882.
2 Ruskin J, Bache JR, Rembert JC, *et al.* Pressure flow studies in man: Effects of respiration on left ventricular stroke volume. *Circulation* 1973;**48**:79.
3 Shabetai R, Fowler NO, Gueron M. The effects of respiration on aortic pressure and flow. *Am Heart J* 1963;**65**:525.
4 Cooper JP, Oliver RM, Currie P, Walker JM, Swanton RH. How do the clinical findings in patients with pericardial effusions influence the success of aspiration? *Br Heart J* 1995;**73**:351–4.

Is thrombolytic therapy really better than conventional treatment in acute inferior myocardial infarction?

SIR,—Dr Tobé's suggestion of not thrombolysing patients with acute inferior myocardial infarction (*Br Heart J* 1995;73:108–9) seems unjustified. The meta-analysis performed by the Fibrinolytic Therapy Trialists suggests that thrombolytic treatment produces a non-significant reduction in mortality of eight lives per 1000 patients treated for inferior infarction.[1]

This analysis, however, included an appreciable number of patients who were given thrombolytic agents between 12 and 24 hours after the onset of chest pain, when fibrinolytic therapy is known to be less effective and may even be harmful. Domiciliary thrombolytic trials showed an additional gain of more than 20 lives per hour saved for every 1000 patients treated early.[2 3] Great effort has been made to fast track infarct patients for earlier inhospital thrombolysis.[4]

Mortality should not be the only end point considered in thrombolytic trials. Better preservation of left ventricular function in thrombolysed patients with inferior myocardial infarction is likely to result in long-term reduction of both morbidity and mortality.[5] Earlier treatment with fibrinolytic therapy within 3 hours of the onset of chest pain also reduces the release of creatine kinase MB, reflecting better preservation of left ventricular function.[6]

Patients with inferior myocardial infarction have a lower mortality than those with anterior infarction. This may account for the lower benefit derived from thrombolytic therapy. The presenting electrocardiogram is helpful in risk stratification. For example, patients with inferior ST elevation and reciprocal ST depression have a poorer prognosis[7] and may benefit more from thrombolytic intervention.

For patients with inferior infarcts the aim must surely be to achieve earlier thrombolytic treatment to obtain maximum benefit. It may be appropriate to thrombolyse only those patients with a poorer prognosis if they present late.

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1 Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. *Lancet* 1994;**343**:311–22.
2 GREAT Group. Feasibility, safety, and efficacy of domiciliary thrombolysis by general practitioners: Grampian region early anistreplase trial. *BMJ* 1992;**305**:548–53.
3 Weaver WD, Cerqueira M, Hallstrom AP, Litwin PE, Martin JS, Kudenchuk PJ, *et al* for the Myocardial Infarction Triage and Intervention Project group. Pre-hospital initiated *vs* hospital-initiated thrombolytic therapy. *JAMA* 1993;**270**:1211–6.
4 Pell A, Miller H, Robertson C, Fox KA. Effect of "fast track" admission for acute myocardial infarction on delay to thrombolysis. *BMJ* 1992;**304**:83–7.
5 White HD, Norris RM, Brown MA, *et al.* Effect of intravenous streptokinase on left ventricular function and early survival after acute myocardial infarction. *N Engl J Med* 1987;**317**:850–5.
6 ISAM study Group. A prospective trial of intravenous streptokinase in acute myocardial infarction (ISAM): mortality, morbidity, and infarct size at 21 days. *N Engl J Med* 1986;**314**:1465–71.
7 Bates ER, Clemmensen PM, Califf RM, *et al* for the TAMI Study Group. Precordial ST segment depression predicts a worse prognosis in inferior infarction despite reperfusion therapy. *J Am Coll Cardiol* 1990;**16**: 1538–44.

SIR,—We think Dr Tobé (*Br Heart J* 1995;73:108–9)is wrong to advocate conventional treatment and not thrombolysis for patients with acute inferior myocardial infarction. On the basis of the currently available evidence, patients without contraindications presenting with inferior ST segment elevation (≥1 mm in two or more leads) within 12 hours of the onset of chest pain should receive thrombolytic therapy.

The Fibrinolytic Therapy Trialists' (FTT) overview of large, randomised, controlled trials of thrombolytic therapy for acute infarction showed an 18% (SD 2) proportional reduction in mortality at 35 days from 11·5% to 9·6% (95% CI 23 to 13% reduction). For the subgroup of patients with inferior infarction the reduction was