

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).

Assessment by general practitioners of suitability for thrombolysis in patients with suspected acute myocardial infarction

SIR—The paper by Gemmill *et al* raises three issues.¹ (a) A general practitioner may refer a patient with chest pain to a coronary care unit because he thinks the patient has had an acute myocardial infarction or because he thinks he has not—but, to be on the safe side, he would like acute myocardial infarction ruled out. To expedite admission a general practitioner will plead either case with similar conviction. The admitting medical officer therefore has a biased perception of the general practitioner's diagnostic skills. If, at the time of request for admission, the general practitioner is questioned about his diagnosis and the patient's suitability for thrombolysis, he will exaggerate the severity of the illness and the patient's need for treatment in order to justify his request. What the general practitioner says he believes and will do and what he really believes and will do are different.

Similarly with recording and interpreting the electrocardiogram (ECG): the general practitioner's skills in this area cannot be reliably assessed under simulated conditions. He will take much more care if a therapeutic decision of his own is contingent on the result than if he is recording and reporting an ECG as part of someone else's research project. Moreover, by producing interpretable ECGs in only 60% of cases, the urban doctors who took part in this study were conveniently able to prove to everyone's satisfaction their own unsuitability to undertake the unwanted, onerous responsibility of domiciliary thrombolysis.

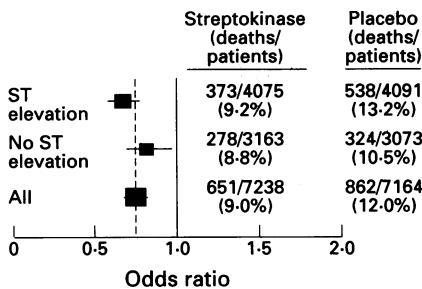
Thus the method of this study is seriously flawed; though the general conclusion, that general practitioners need more knowledge and experience of recording and interpreting the ECG before they use thrombolysis, is probably correct.

The study seems to vindicate the reluctance of cardiologists to let general practitioners use thrombolytic therapy. But the real message is that you can take a horse to water but you cannot make it drink.

(b) Gemmill *et al* should not equate the transit time of 35 minutes with the time that would have been saved by domiciliary thrombolysis. Was their door-to-needle time really zero?

(c) A thrombolysis policy that restricts treatment to 51% of those with acute myocardial infarction must be questioned. The figure shows that patients with suspected acute myocardial infarction with or without ST segment elevation on the presenting ECG benefit from streptokinase. The data are from ISIS-2.²

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Data from ISIS-II: streptokinase compared with placebo. 95% confidence intervals are shown (the size of the box is proportional to the number of patients).

- Gemmill JD, Lifson WK, Rae AP, Hillis WS, Dunn FG. Assessment by general practitioners of suitability of thrombolysis in patients with suspected acute myocardial infarction. *Br Heart J* 1993;70:503-6.
- Second international study of infarct survival collaborative group. Randomised trial of intravenous streptokinase. Oral aspirin, both, or neither among 17187 cases of suspected acute myocardial infarction: ISIS-2. *Lancet* 1988;ii:349-60.

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

SIR,—We are grateful for being given the opportunity to respond to the points raised by Dr Rawles, who of course has extensive experience in administration of thrombolytic therapy in a rural setting.

His first point relates to the fact that the general practitioners did not actually administer thrombolytic therapy. We considered this point very carefully before embarking on the study, and ideally, if at the time it had been possible, it would have been preferable for the general practitioners to administer the agent. This, however, was not an option open to us when the study was designed. We were fortunate to have a back door admission policy to the coronary care unit with which the GPs had been familiar for several years. Analysis of the data indicates that they did not respond differently in terms of the type of patients they sent to the back door during the study. Therefore there is no evidence that the GP exaggerated the severity of the patient's illness. Furthermore the actual recording and interpretation of the ECG was the basis of a real life admission to hospital, with inherent audit.

Only GPs who carried out their own evening on call system were included in this study. They were all interested and committed GPs who showed their willingness to volunteer for participation in the study. This makes Dr Rawles' comments about these GPs singularly inappropriate. It is disappointing that only 60% of ECGs were

interpretable, but important information such as this must be faced before widespread prescription of domiciliary thrombolysis can be considered.

Secondly, Dr Rawles thinks that we should not have equated the transit time of 35 minutes with the time that would have been saved by domiciliary thrombolysis. Of course we are not suggesting that our door to needle time was zero. What we are saying, however, is that door to needle time at home and in hospital is the same if you have a back door policy such as we have and therefore the time saved is 35 minutes.

Thirdly, Dr Rawles expresses concern that only 51% of the patients with acute myocardial infarction were given thrombolytic therapy. He attempts to support this statement by quoting some retrospective subgroup analysis from ISIS-2. A more balanced view is given in a recent editorial in the *British Medical Journal*¹ indicating that the only situation in addition to ST elevation where the benefit of thrombolytic therapy is clear-cut is in patients with bundle branch block. We concur with this view and therefore do not believe that there is anything questionable about 51% of the patients with discharge diagnosis of myocardial infarction receiving thrombolytic therapy at the time of our study.

We would like to emphasise again that though domiciliary administration of thrombolytic therapy does have some benefits in the rural setting,² this information cannot be extrapolated to the urban setting where with a fast track admission policy there is likely to be little if any advantage to the patient.

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- Cobbe SM. Thrombolysis in myocardial infarction. *Br Med J* 1994;308:216-7.
- GREAT Group. Feasibility, safety and efficacy of domiciliary thrombolysis by general practitioners: Grampian region early anistreplase trial. *Br Med J* 1992;305:548-53.

Streptokinase antibodies are of clinical importance and they can be measured in half an hour by a simple enzyme-linked immunosorbent assay

SIR,—Buchalter and Patel *et al* implied that the relation between anti-streptokinase antibodies and the lytic efficacy of streptokinase or its derivative, anistreplase, in patients with acute myocardial infarction is unknown.^{1,2} Furthermore, they seemed to be unaware that there is a rapid assay for streptokinase antibodies.

We and others reported a strong relation between a systemic non-lytic state and angiographic non-patency of the infarct related vessel in patients with myocardial infarction.^{3,4} In addition when titres of streptokinase antibodies were high before thrombolytic therapy with anistreplase a systemic lytic state and subsequent patency of the infarct related vessel were less likely to be achieved.⁵ Finally, there is a simple enzyme-linked immunosorbent assay that can measure these antibodies in half an hour.⁶ Because this method is quick and easy to perform, its use may lead to addi-

tional or alternative thrombolytic therapy being given and so to early coronary artery reperfusion.

Sigwart *et al* recommended that streptokinase antibodies should be assayed routinely in patients being given thrombolytic therapy after myocardial infarction.⁷ The commonly used radioimmunoassay, however, is time consuming and inconvenient and therefore not suitable as a guide to clinical strategies in critically ill patients. Some clinicians routinely measure serum fibrinogen immediately after administration of streptokinase to identify patients in whom delayed or failed reperfusion is likely because streptokinase antibodies are present.⁸ This method is still useful, but there is a quick and easy assay for streptokinase antibodies that can be used for screening and to guide clinical treatment.

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- 1 Buchalher MB. Are streptokinase antibodies clinically important? *Br Heart J* 1993;70:101-2.
- 2 Patel S, Galihal S, Dutka DP, Morris GK. Streptokinase neutralisation titres up to 866 days after intravenous streptokinase for acute myocardial infarction. *Br Heart J* 1993;70:119-21.
- 3 Brügemann J, van der Meer J, Takens LH, Hillege H, Lie KI. A systemic non-lytic state and local thrombolytic failure of anistreplase (anisoylated plasminogen streptokinase activator complex, APSAC) in acute myocardial infarction. *Br Heart J* 1990;64:355-8.
- 4 Lew AS, Neer T, Rodriguez L, Geft IL, Shah PK, Ganz W. Clinical failure of streptokinase due to unsuspected high titer of anti-streptokinase antibody. *J Am Coll Cardiol* 1984;4:183-5.
- 5 Brügemann J, van der Meer J, Bom VJJ, van der Schaaf W, de Graeff PA, Lie KI. Anti-streptokinase antibodies inhibit fibrinolytic effects of anistreplase in acute myocardial infarction. *Am J Cardiol* 1993;72:462-4.
- 6 Bom VJJ, Brügemann J, van der Schaaf W, van Wijk RT, van der Meer J. Rapid enzyme immunoassay of anti-streptokinase antibodies in human plasma. *Clin Chim Acta* 1993;218:121-9.
- 7 Sigwart U, Grbic M, Bachmann F. Measurement of antistreptokinase antibodies. *J Am Coll Cardiol* 1985;5:1500.
- 8 Lew AS, Laramée P, Ganz W. Reply to Sigwart, *et al.* *J Am Coll Cardiol* 1985;5:1500.

Traumatic rupture of the thoracic aorta diagnosed by transoesophageal echocardiography

SIR,—The case reported by de Belder *et al* confirms the diagnostic value of transoesophageal echocardiography (TOE) in traumatic aortic rupture.¹ They say that the echocardiographic appearances were "previously undescribed". However, I and others first reported the role of TOE in traumatic aortic rupture in 1991,² with echocardiographic images similar to their case. Others subsequently confirmed our findings.^{3,4} I hope that the interesting report of de Belder *et al* will further promote this rapid, accurate bedside technique to diagnose a frequently fatal condition.

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- 1 de Belder A, Thomas M, Marrinan M. Traumatic rupture of the thoracic aorta diagnosed by transoesophageal echocardiography. *Br Heart J* 1993;70:393-4.

- 2 Galvin IF, Black IW, Lee CL, Horton DA. Transoesophageal echocardiography in acute aortic transection. *Ann Thorac Surg* 1991;51:310-1.
- 3 Sparks MB, Burchard KW, Marrin CA, Bean CH, Nugent WC Jr, Plehn JF. Transoesophageal echocardiography. Preliminary results in patients with traumatic aortic rupture. *Arch Surg* 1991;126:711-3.
- 4 Shapiro MJ, Yanofsky SD, Trapp J, *et al*. Cardiovascular evaluation in blunt thoracic trauma using transoesophageal echocardiography (TEE). *J Trauma* 1991;31:835-9.

Maldevelopment of conotruncal and aorto-pulmonary septum with absent left central pulmonary artery: anatomical and clinical implications.

SIR,—We were most interested in the excellent report by Schulze-Neick *et al* in which they described a case of atresia of the pulmonary valve associated with absence of the central component of the left pulmonary artery.¹ We would like to suggest an alternative interpretation of the underlying morphological malformation.

We are confused by their assertion that there was no arterial ligament or duct. As

the left pulmonary artery had no central connection with the pulmonary trunk, from where did it receive its blood flow before the establishment of the modified Blalock-Taussig shunt? If, as we suspect, there was a connection with the concavity of the aortic arch, surely this vessel should be regarded as representing the arterial duct?

Failure of incorporation of the orifice of the right sixth arch into the confluence of the pulmonary arteries, caused by deviation of this orifice or of the aorto-pulmonary septum, results in a single (right) anomalous pulmonary artery arising from the ascending aorta (a condition sometimes erroneously called a "hemitruncus"), in which case the anomalous pulmonary artery remains entirely separated from the pulmonary trunk and the left pulmonary artery. It is difficult to see how the right sixth arch could have two proximal ends with one opening into the aorta and the other joining the pulmonary trunk. The distal end of this arch cannot be involved because had it persisted, it would be connected with the right subclavian artery, and because the non-arcuate portion of the right pulmonary artery terminates in the right lung. We consider that this anomaly was more likely to

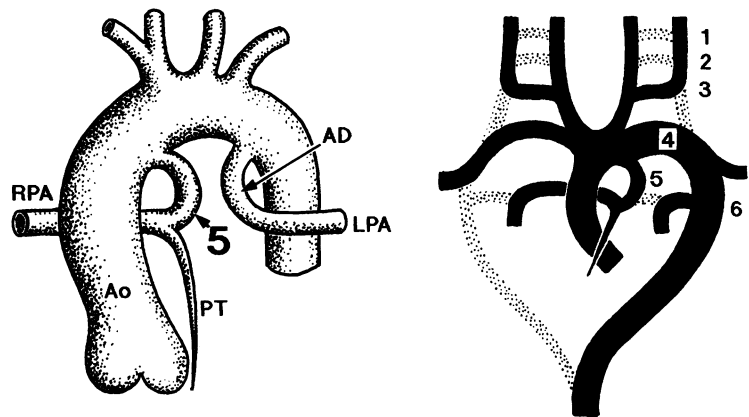


Figure 1 Diagrams of our interpretation of the state of the great arteries in the case described by Schulze Neick, *et al*¹ and in our case (fig 2), and of the underlying embryological arrangement. Ao, aorta; AD, arterial duct; LPA, left pulmonary artery; PT, central pulmonary trunk; RPA, right pulmonary artery; 1-6, embryological aortic arches.

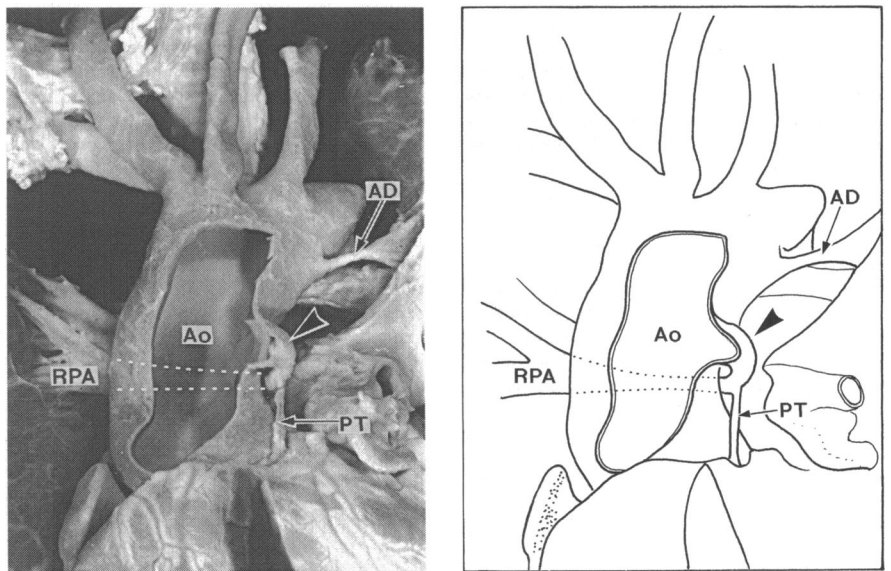


Figure 2 Photograph and key diagram of the specimen in the Brompton collection. The anomalous origin of the right pulmonary artery is indicated by the large arrow head. Ao, aorta; AD, arterial duct; LPA, left pulmonary artery; PT, central pulmonary trunk; RPA, right pulmonary artery; 1-6, embryological aortic arches.