Thrombolytic therapy in A&E departments in the U.K.

Sir
Recent multi-centre clinical trials have demonstrated a reduction in early mortality from acute myocardial infarction when intravenous thrombolytic therapy was used. The outcome was better, the earlier treatment was started.

However, in-hospital delays in the initiation of thrombolytic therapy have been demonstrated both in this country (Burrell et al., 1988; Burns et al., 1989; Dalton et al., 1989) and in the U.S.A. (Sharkey et al., 1989). Analysis of the cited publications suggests that delays in the A&E department are contributory suggesting that treatment should be commenced in the department whenever feasible.

I carried out a postal survey of the current policies regarding thrombolytic usage in A&E departments in the U.K. A&E departments led by 170 consultants were identified from the Casualty Surgeons Association Handbook for 1988. A questionnaire was sent to all 170 consultants in charge in the month of January 1990.

A total of 132 replies were received (a response rate of 77.6%). Of the hospitals responding, 30 were teaching hospitals and the remainder district general hospitals.

In 83 of the responding departments, thrombolytic therapy is at present not initiated in the A&E department. Twenty-two consultants stated that this was due to facilities for rapid transfer to the coronary care unit. Six consultants stated that local physicians were not in favour of initiating thrombolysis in the A&E department. Two consultants stated that there was an apparent lack of unanimity among local physicians about the benefits of thrombolytic therapy. A further seven stated that they were hoping to initiate the use of thrombolytic therapy in their departments in the near future.

Of the 49 A&E departments in which thrombolytic therapy is at present initiated, streptokinase alone is used in 26. Tissue plasminogen activator is used as an alternative to streptokinase in three and to anistreplase in one department. Anistreplase is used as an alternative to streptokinase in a further nine departments, and alone in two. All three agents are used in eight departments.

The decision to initiate thrombolytic therapy in these 49 departments is made solely by A&E staff in seven, by medical and/or cardiology resident staff in 17, and by either of the preceding in the remaining 25.

The accepted time interval from onset of symptoms to commencement of thrombolysis was stated to be within the first 4 h in five, within the first 6 h in 27, and at any time within the first 24 h in the remaining 17 departments.

The survey shows that there is wide variation in practice in the U.K. and that the role of the A&E department in the initiation of thrombolytic therapy awaits clarification. Undoubtedly the potential for delay in the A&E departments does exist and may be beyond the direct control of the departmental staff. This includes involvement with other seriously ill patients, delays in contacting resident medical staff, lack of immediate bed availability, lack of porters and other causes of delayed
transfer to the ward. Besides, resident medical staff may be busy with other urgent clinical duties.

There seems little justification for not considering commencing thrombolysis in the A&E department in the face of such problems. Also, an agreed written protocol with clear indications and contraindications should help minimise the risks of inadvertent inappropriate administration of these agents.

Greater uniformity of procedure and clearer guidelines for A&E staff are eagerly awaited.

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REFERENCES

Spontaneous pneumomediastinum
Sir
A 25-year-old male presented to the A&E Department with a 6-h history of increasingly severe retrosternal chest pain, radiating to the left supraclavicular region, associated with dyspnoea and unproductive coughing. The pain was exacerbated by deep inspiration and eased by sitting forward. The patient initially denied any precipitating event but on repeated interrogation admitted to taking a teaspoonful of powdered amphetamine sublingually 8h prior to admission, during an ‘acid house’ party. He had smoked approximately 20 cigarettes since taking the amphetamine but categorically denied other substance abuse. The patient usually smoked 10 cigarettes daily but had no history of overt lung disease.

On examination, the patient was pale, distressed and tachypnoeic (respiratory rate 22 min⁻¹). The pulse rate was 110 and the blood pressure was 145/85; he was acyanotic and apyrexial. Surgical emphysema was faintly palpable in the left supraclavicular fossa. The heart sounds were muffled and Hamman’s sign was positive (i.e. a crunching noise on auscultation over the left third to fifth intercostal spaces parasternally, varying with systole and accentuated by expiration in the left lateral decubitus position). Air entry was reduced symmetrically and there were scattered wheezes throughout both upper lung fields. The peak expiratory flow rate (PEFR) was 2501 min⁻¹ (50% of expected PEFR). Pulse oximetry revealed an