

Electrocardiogram interpretation as a basis for thrombolysis

ABSTRACT – *Objective:* to assess the skills and opinions of different grades of doctors and cardiac-trained nurses in interpreting electrocardiographic changes when deciding upon administration of thrombolysis to patients with chest pain.

Design: a questionnaire was distributed to staff in several local hospitals.

Subjects and methods: participants were asked to assess 30 electrocardiograms (ECGs) and determine whether they would prescribe thrombolytic therapy on the basis of each one, assuming an associated typical history of acute myocardial infarction (AMI) and no contraindications to treatment. They were asked to return the questionnaire anonymously, stating only their position.

Results: of the 88 questionnaires, 61 were returned by 15 senior nurses, 10 house officers, 12 senior house officers, 10 medical registrars, eight consultant physicians and six consultant cardiologists. When electrocardiograms showed unequivocal evidence of acute myocardial infarction, all consultant cardiologists gave the correct answer, but only 75.5% of house officers diagnosed AMI. Cardiologists were most in favour of thrombolysis when left bundle branch block was present. Cardiac-trained nurses showed good decision-making skills.

Conclusions: staff involved in assessment of patients with chest pain should have specific training in electrocardiographic diagnosis of myocardial infarction in order to minimise in-hospital delay when thrombolysis is indicated. The management of patients with left bundle branch block remains uncertain; cardiologists are more likely to recommend thrombolytic therapy than any of the other participants in the study.

The immediate assessment of patients with chest pain of possible cardiac origin has become a high priority in emergency medicine. This is in the light of major thrombolysis trials which have shown a reduction in morbidity and mortality when thrombolytic agents are administered to patients with evolving myocardial infarction, at least to those whose ECG shows diagnostic ST elevation or bundle branch block¹. However, audit of the time to thrombolysis from the onset of pain continues to show that in many centres in-hospital delay is a significant component of overall delay. One of the features that correlates with in-

hospital delay is the extent of ST elevation on the presenting electrocardiogram: the greater the magnitude of ST elevation, the less the delay to thrombolysis². Previous studies have analysed the abilities of doctors to interpret ECGs and have identified general deficiencies^{3,4} but have not demonstrated clearly how this might affect patients presenting with AMI. The purpose of this study was to look at the interpretative skills and obtain the opinions of different grades of hospital doctor, with specific regard to the ECG diagnosis of myocardial infarction and the decision to administer thrombolysis.

Methods

Questionnaires were sent to house physicians, medical or casualty senior house officers, medical registrars, consultant physicians and consultant cardiologists in several different centres. Questionnaires were also sent to senior nurses involved in the management of patients with AMI. Doctors were presented with the case of a 45-year-old male smoker in the accident and emergency department, complaining of sudden onset of severe indigestion-like retrosternal pain two hours previously, associated with nausea, sweating and shortness of breath; the pain had now lessened but there was persistent nausea, malaise and sweating; he had no previous medical history to note, and no allergic or drug history; history and clinical examination revealed no contraindications to thrombolysis. Doctors were then asked to analyse 30 ECGs taken from a variety of sources and say whether or not, in the light of this case and the individual ECG findings, they would administer thrombolysis. They were asked to assess the ECG as rapidly as they would do routinely without conferring with colleagues. They were assured that the study was entirely anonymous, but were asked to indicate their position. It was suggested that if they were unsure whether to administer thrombolysis, they should indicate that they would not do so (on the basis that, in practice, review by a more experienced colleague may incur a significant delay). Questionnaires were returned by post and the results of the study were sent to all participants.

Of the 30 ECGs in this study, 19 were recorded on patients presenting in the acute phase of myocardial infarction and 11 were recorded on patients in whom AMI was not suspected or later excluded. Some ECGs were from patients with Q wave myocardial infarction to see whether the presence of Q waves influenced decisions on thrombolysis.

ROBERT F STOREY, MRCP, Registrar in Medicine

JOHN M ROWLEY, MA, MRCP, Consultant Cardiologist

King's Mill Hospital, Sutton-in-Ashfield

Results

Sixty-one of the 88 questionnaires were returned fully completed, by 15 senior nurses, 10 house officers, 12 senior house officers, 10 medical registrars, 8 consultant physicians and 6 consultant cardiologists.

The answers were analysed according to the position of the respondent and the following categories of ECG:

1. ECGs recorded on patients presenting in the acute phase of myocardial infarction:

(a) Unequivocal evidence of acute injury: more than 2mm ST elevation of 'acute' morphology in all inferior leads or at least two contiguous chest leads,

without pathological Q waves in these leads and with accompanying reciprocal changes ($n=11$).

(b) Strong evidence of acute injury: 1mm or more ST elevation in at least two inferior leads, or 2mm in two other contiguous leads, or 1–2mm ST elevation in two contiguous V leads of typical 'hyperacute' morphology, without pathological Q waves in those leads, or left bundle branch block ($n=8$).

(c) Non diagnostic initial ECG: isolated 2mm ST elevation in V2 of suspicious morphology ($n=1$).

2. ECGs recorded on patients not in the acute phase of myocardial infarction:

(a) Abnormal ECGs: Q waves only, left ventricular

Table 1a. Number (percentage) of answers advocating thrombolysis according to grade and ECG criteria - presenting ECGs from patients with proven acute myocardial infarction.

ECG criteria	Senior nurse $n=15$ (%)	House officer $n=10$ (%)	Senior house officer $n=12$ (%)	Medical registrar $n=10$ (%)	Consultant physician $n=8$ (%)	Consultant cardiologist $n=6$ (%)
Unequivocal evidence of acute injury $n=11$	150/165 (91)	83/110 (75)	111/132 (84)	104/110 (95)	80/88 (91)	66/66 (100)
Strongly suggestive of acute injury (including left bundle branch block) $n=8$	84/120 (70)	32/80 (40)	58/96 (60)	48/80 (60)	42/64 (66)	33/48 (69)
Left bundle branch block only $n=1$	10/15 (67)	0/10 (0)	4/12 (33)	5/10 (50)	3/8 (38)	5/6 (83)
Non diagnostic initial ECG $n=1$	1/15 (7)	1/10 (10)	1/12 (8)	0/10 (0)	0/8 (0)	0/6 (0)

Table 1b. Number (percentage) of answers advocating thrombolysis according to grade and ECG criteria - presenting ECGs from patients without proven acute myocardial infarction.

ECG criteria	Senior nurse $n=15$ (%)	House officer $n=10$ (%)	Senior house officer $n=12$ (%)	Medical registrar $n=10$ (%)	Consultant physician $n=8$ (%)	Consultant cardiologist $n=6$ (%)
Abnormal ECGs $n=8$	15/120 (13)	9/80 (11)	18/96 (18)	15/80 (19)	20/64 (31)	14/48 (29)
Normal ECGs $n=2$	2/30 (7)	2/20 (10)	0/24 (0)	0/20 (0)	0/16 (0)	0/12 (0)

hypertrophy, ST depression or T wave changes related to previous non-Q wave myocardial infarction ($n=8$).

(b) Normal ECGs ($n=2$).

Tables 1a and 1b show the responses of the different grades of respondents to these categories. Tables 2a and 2b list the features of ECGs where all grades of respondent either answered 'yes' to thrombolysis for an abnormal ECG which did not in fact represent acute infarction, or answered 'no' to thrombolysis for an abnormal ECG which did in fact show some changes of acute injury and was recorded from a patient in the acute phase of infarction.

Figures 1 and 2 are two of the ECGs used in this study. You are invited to examine these and decide whether to give thrombolysis. The 'correct' answer is given at the end of this article (see *Description of ECGs*).

Discussion

This study illustrates a skills gap in diagnosing AMI between cardiologists and medical registrars on the one hand, and junior and senior house officers on the other, in cases where there is unequivocal evidence of acute myocardial injury. Although this questionnaire is a crude assessment of clinical decision-making, it does suggest that some of the observed delay (more than 60 minutes) in administering thrombolysis after a patient has presented to the hospital may relate to lack of confidence and skills in ECG interpretation amongst staff who are charged with the responsibility of first-line management of patients with chest pain. Medical students and house officers should be given better training in ECG diagnosis of AMI, to shorten in-hospital delays in administering thrombolysis. Further

consideration should also be given to a centralised ECG interpretation service so that an experienced assessment is made with minimum delay.

This study also illustrates a general lack of consensus in cases where pre-existing abnormalities or the particular morphology of the ST segment elevation confound electrocardiographic diagnosis. All groups were divided in deciding whether to give thrombolysis, in the context of the history presented, when the ECGs showed ST elevation associated only with pathological Q waves. Also, cardiologists were more likely to advocate thrombolysis in cases where the segment of ST elevation was linear rather than 'saddle-shaped' morphology (for the same degree of elevation), recognising that the latter may sometimes be caused by a repolarisation variant or pericarditis. Identification of reciprocal ST depression in opposing leads in acute myocardial injury is important since, excepting lead aVR, this is not a feature of early repolarisation syndrome or acute pericarditis⁵. Two ECGs in this study, recorded from the same patient with AMI, showed more than 2mm ST elevation in V5 and V6 but this was associated with a prominent J wave and slurred ST segment ('saddle-shaped'). A significant proportion of all groups of respondent, in making a decision on thrombolysis, failed to attach significance to reciprocal changes seen in opposing leads.

The majority of cardiologists advocated the administration of thrombolysis when the ECG showed left bundle branch block, since it could be inferred from the history that no previous ECG record existed for this case; in the context of a typical presentation of AMI, it was felt appropriate to regard this as a new change, appreciating the evidence that the association of left bundle branch block with acute infarction carries a particularly high mortality which is greatly reduced by thrombolysis. However, most of the other doctors did not advocate thrombolysis in this setting, implying either that they failed to recognise the presence or significance of left bundle branch block or that they were not prepared to risk a false positive diagnosis of acute infarction. Evidence presented from the GUSTO-I (Global utilisation of streptokinase and tissue plasminogen activator for occluded coronary arteries) trial suggests that there are three useful markers that may help in the diagnosis of acute infarction in the presence of left bundle branch block: ST depression greater than or equal to 1mm concordant with the QRS complex; ST elevation greater than or equal to 1mm in lead VI, V2 or V3; and ST elevation greater than or equal to 5mm, discordant with the QRS complex⁶. However, an overview of the subject by Wellens⁷ favours the administration of thrombolysis to patients admitted with chest pain suggestive of acute cardiac ischaemia and left bundle branch block, whether or not these particular changes are present.

Previous thrombolysis trials have shown that most false positive diagnoses of myocardial infarction are due to the presence of ST segment abnormalities

Table 2a. Features of ECGs where false positive diagnosis of acute myocardial infarction is made.

- ST elevation associated only with Q waves due to previous myocardial infarction.
- Early repolarisation in vagotonic individual.
- Early repolarisation due to left ventricular hypertrophy.
- Extreme ST depression only.
- Marked biphasic T wave abnormality and loss of R wave representing previous non-Q wave myocardial infarction.
- Early repolarisation in V2 and ST depression elsewhere.

Table 2b. Features of ECGs where false negative diagnosis of myocardial infarction is made.

- Changes of previous myocardial infarction but ST elevation in leads with residual R wave representing reinfarction.
- 'Saddle-shaped' ST elevation in lateral chest leads but with reciprocal changes.
- Left bundle branch block.
- 1mm acute inferior ST elevation with isolated Q wave in lead III and minor reciprocal changes.

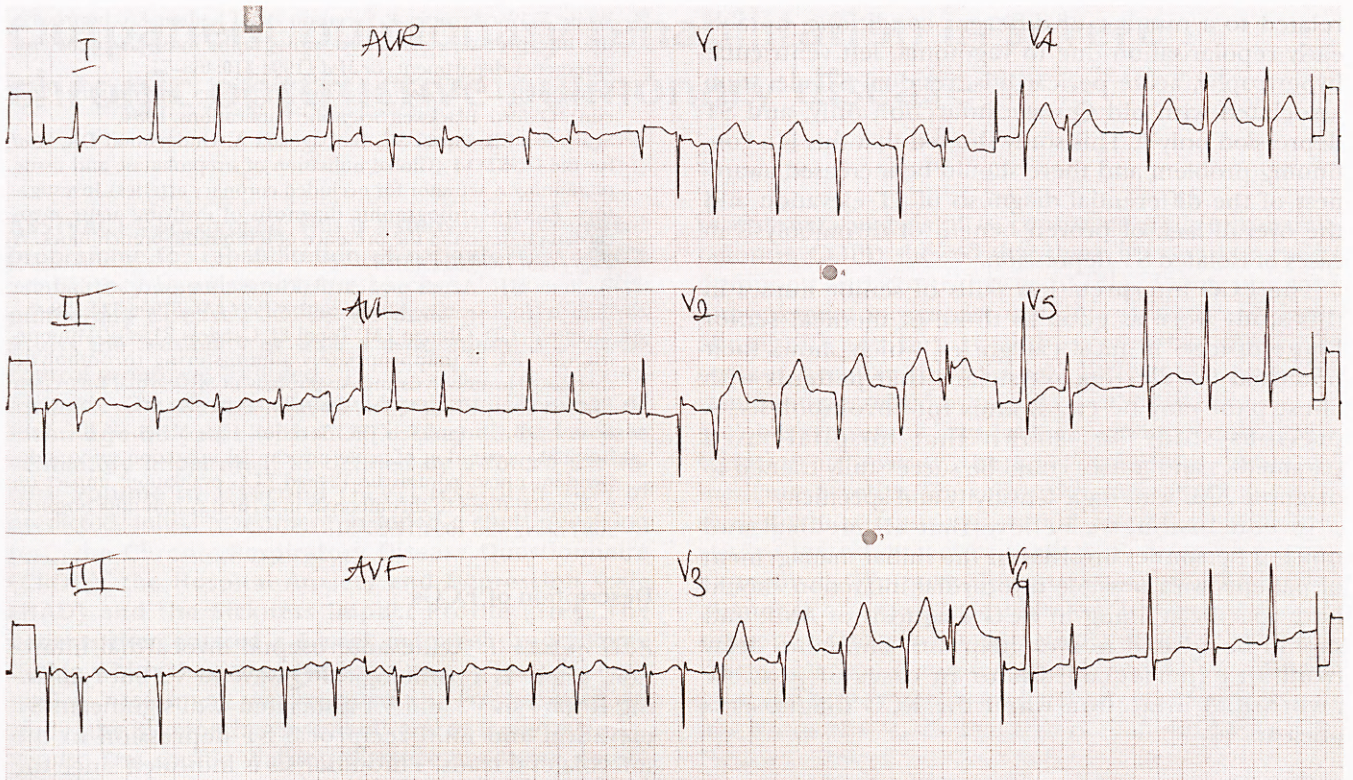


Fig 1. ECG number 1.

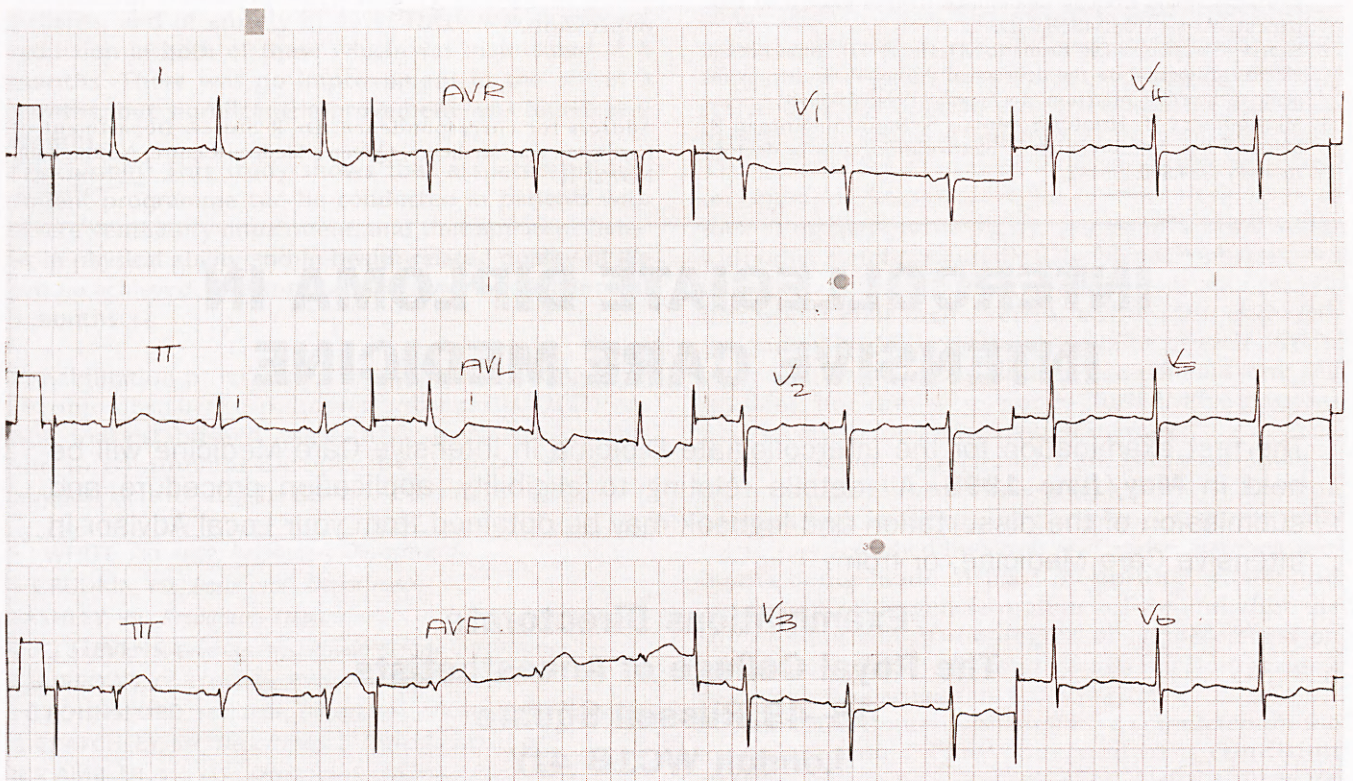


Fig 2. ECG number 2.

related to a number of different conditions, notably early repolarisation due to vagotonia, left ventricular hypertrophy, acute pericarditis, residual ST elevation associated with old Q wave infarction only, and ST depression only^{1,8}. This study showed that this is a continuing problem and there should be increased awareness of the differential diagnosis of ST elevation and the overall lack of proven benefit of thrombolysis in cases of isolated ST depression.

The ECG interpretation skills of senior nurses in this study were as good as those of medical senior house officers in most categories, which shows their potential for alerting physicians that patients with chest pain may be candidates for thrombolysis. A previous study has shown that hospital-based paramedics and nurses could be successfully trained to diagnose AMI with high sensitivity and specificity⁹.

In conclusion, we believe that all medical and nursing personnel involved in the initial management of patients with possible myocardial infarction should have specific training in ECG diagnosis of AMI, and should be advised to seek help with minimum delay from an experienced doctor in cases of possible myocardial infarction when the ECG diagnosis is unclear.

References

- 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 Sharkey SW, Berger CR, Brunette DD, Henry TD. Impact of the electrocardiogram on the delivery of thrombolytic therapy for acute myocardial infarction. *Am J Cardiol* 1994;**73**:550-3.
- 3 Montgomery H, Hunter S, Morris S, Naunton-Morgan R, Marshall RM. Interpretation of electrocardiograms by doctors. *Br Med J* 1994;**309**:1551-2.
- 4 White T, Woodmansey P, Ferguson DG, Channer KS. Improving the interpretation of electrocardiographs in an accident and emergency department. *Br Med J* 1994;**310**:468-71.
- 5 Schamroth L. *The electrocardiology of coronary artery disease* (2nd edn). Oxford: Blackwell Scientific Publications, 1984.
- 6 Sgarbossa EB, Pinski SC, Barbagelata A, Underwood DA, *et al* for the GUSTO-1 (Global utilisation of streptokinase and tissue plasminogen activator for occluded coronary arteries) Investigators. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle branch block. *N Engl J Med* 1996;**334**:481-7.
- 7 Wellens HJJ. Acute myocardial infarction and left bundle branch block - can we lift the veil? *N Engl J Med* 1996;**334**:528-9.
- 8 Chapman GD, Ohman EM, Topol EJ, Candela RJ, *et al*. Minimizing the risk of inappropriately administering thrombolytic therapy (Thrombolysis and Angioplasty in Myocardial Infarction [TAMI] Study Group). *Am J Cardiol* 1993;**71**:783-7.
- 9 Foster DB, Dufenbach JH, Barkdoll CM, Mitchell BK. Pre-hospital recognition of acute myocardial infarction using independent nurse/paramedic 12-lead ECG evaluation: impact on in-hospital times to thrombolysis in a rural community hospital. *Am J Emerg Med* 1994;**12**:25-31.

Description of ECGs

ECG number 1: Acute anteroseptal myocardial infarction. There is a pathological Q wave in V2 but remaining R wave in V3 and V4 associated with significant ST elevation and mild reciprocal ST depression in the inferolateral leads. Thrombolysis is indicated since the onset of pain was less than 12 hours previously.

ECG number 2: Acute inferior myocardial infarction. There is significant inferior ST elevation of typical acute morphology and widespread reciprocal ST depression.

Address for correspondence: Dr R Storey, Department of Cardiovascular Medicine, Queen's Medical Centre, Nottingham, NG7 2UH.

INTERCOLLEGIATE DIPLOMA IN INTENSIVE CARE MEDICINE

The first examination for the Intercollegiate Diploma in Intensive Care Medicine will be held in **May/June 1998**. All details relating to eligibility, application procedure and submission of the dissertation and logbook may be obtained from your Local Advisor in Intensive Care Medicine, or from:

**Examinations Directorate
The Royal College of Anaesthetists
48-49 Russell Square
London WC1B 4JY**