conclusions regarding the sensitivity and specificity of electrocardiographic criteria for myocardial infarction in their study therefore remains in doubt.

Receiver operator characteristic curves should aim for the highest accuracy because of their value in portraying grades of diagnostic uncertainty, the therapeutic implications of diagnostic uncertainty being especially important in the risk:benefit analysis of potentially dangerous drug treatments. In this context, ST segment elevation should continue to be used as a precondition for thrombolytic treatment because of evidence that streptokinase given within two hours of the onset of chest pain produces the most beneficial effects on left ventricular function and survival in the subset of patients with a sum of ST segment elevation of $\geq 1{\cdot}2$ mV.[5]

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Disappearing ST elevation could mean reperfusion

EDITOR,—Jacqueline Adams and colleagues suggest that "elevation of the ST segment . . . is an unsatisfactory precondition for giving thrombolytic treatment to patients with suspected acute myocardial infarction."[1] They express particular concern about those patients with confirmed myocardial infarction in whom ST elevation that was present in the initial electrocardiogram has resolved spontaneously by the time of their admission to hospital. The authors suggest that it is a mistake to deny such patients thrombolytic treatment. I wish to make two points.

Firstly, patients in whom ST elevation resolves spontaneously before admission may have a relatively favourable prognosis even without thrombolysis. A rapid reduction in ST elevation has been suggested to be a useful surrogate marker of reperfusion and of better preserved ventricular function.[2,3] Spontaneous reperfusion is well recognised in myocardial infarction: DeWood *et al* showed a rate of coronary occlusion of only 65% at 12-24 hours in transmural myocardial infarction[4] and as low as 26% in non-Q wave myocardial infarction.[5]

Secondly, thrombolysis carries a small risk of serious side effects (major bleeding 0·2-1%, allergy 0·1-1·7%, hypotension 1·7-10%, plus an excess of haemorrhagic but not total strokes), which must be balanced against its potential benefits.

It remains common practice to reserve thrombolysis for patients with ST elevation or new left bundle branch block in their electrocardiogram. The risk:benefit ratio for thrombolysis in those presenting with other patterns is less certain. There is insufficient evidence to recommend wholesale changes to the present criteria for giving thrombolytic agents in hospital. Further work is required to determine whether the same applies for patients presenting at an earlier stage in the community and starting treatment at home.

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Domiciliary ECGs unreliable

EDITOR,—Although all doctors would like to have easy criteria for giving thrombolysis in acute myocardial infarction, in practice we are still left with individual risk:benefit assessments. Unfortunately, Jacqueline Adams and colleagues' paper does not help in this respect.[1] Clinicians recognise the difficulty of diagnosing myocardial infarction on the basis of electrocardiographic changes, particularly when the electrocardiogram is obtained early after the onset of symptoms, and it is no surprise that infarction can occur despite the lack of classic ST elevation.

It is quite another question, however, whether patients will benefit from thrombolysis given for "abnormal" electrocardiograms. An unpublished overview of major trials of thrombolysis by the Oxford-ISIS group has shown that benefits are seen only in those with ST elevation or bundle branch block. Those with ST depression do worse in terms of mortality. Furthermore, it is sometimes difficult for hospital clinicians to interpret electrocardiograms, let alone general practitioners in far from ideal circumstances at the patient's home.

Shouldn't we concentrate on improving policies for rapid admission to allow early, safe thrombolysis rather than wasting time in obtaining and pondering over domiciliary electrocardiograms?

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Acronyms must be explained

EDITOR,—I was surprised that the acronym GREAT was not explained in the recent article by Jacqueline Adams and colleagues.[1] According to the International Committee of Medical Journal Editors, of which the *BMJ*'s editor is a member, abbreviations should be avoided in the title of articles and the full term for which an abbreviation stands should precede its first use in the text.[2] As an acronym is like an abbreviation the same rule should apply. I do not think that many readers will know what GREAT means unless they search through all the references of the article and look carefully at the title of reference 3[3]: GREAT stands for Grampian region early anistreplase trial.

Physicians, especially cardiologists, like to use or invent acronyms. Unless these are explained, however, they lead to confusion and sometimes frustration. Acronyms are often necessary but can be perplexing if you do not know what they stand for.[4] That was why I prepared a list of acronyms of major cardiological trials,[5] which is currently being updated. Acronyms can sometimes create additional problems because several trials share the same acronyms.[5]

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Authors' reply

EDITOR,—The result of a clinical trial is applicable only to patients with the same characteristics as those who participated in the trial. The conclusion from the second international study of infarct survival is that thrombolytic treatment reduces mortality in patients with suspected acute myocardial infarction.[1] That conclusion has not been overturned by any new evidence. Post hoc subgroup analysis has shown that patients with ST elevation or bundle branch block do particularly well, but a reduction in mortality is not precluded in subgroups with other electrocardiographic abnormalities, even ST depression.

But for most patients with acute myocardial infarction, especially those presenting with electrocardiographic abnormalities that are less specific for infarction than ST elevation, the immediate benefit of thrombolytic treatment is not saving life but saving myocardium. Taking to absurd lengths the argument that thrombolytic treatment should be given only to those who have been shown to benefit would result in this treatment being restricted to just the 2-3% of patients who would die without it. But surely all patients with myocardial infarction stand to gain from thrombolytic treatment, whatever the size of the untreated infarct. The losers are those without myocardial infarction, who are exposed to the risks of thrombolysis without possibility of benefit.

We agree that there is no gold standard for diagnosing myocardial infarction, but we assayed the most specific enzyme available (myocardial isoenzyme of creatine kinase, not creatine kinase); had we measured it four hourly or used a different threshold the shape of the receiver operator characteristic curves would have been much the same and our conclusion that ST elevation is an unsatisfactory precondition for thrombolytic treatment would have been unchanged. Had the paper stopped there the conclusion would have been negative but non-controversial. We went on to suggest an alternative precondition for thrombolytic treatment—the presence of any electrocardiographic abnormality—citing the support of a benefit:risk analysis.[2]

The worst result of thrombolytic treatment is cerebral haemorrhage in a patient without myocardial infarction. With the incidence of this complication being used as the denominator, benefit:risk ratios have been calculated for subgroups of patients with suspected myocardial infarction and various electrocardiographic abnormalities. These ratios are unacceptably low only in patients whose electrocardiogram on presentation is normal. Giving thrombolytic treatment in hospital to all patients with suspected acute myocardial infarction and an abnormal electrocardio-