to children. A three year old who presented with polymorphic ventricular tachycardia with episodes of syncope had her arrhythmia controlled with a combination of propranolol and flecainide. Trough blood levels of flecainide measured 320 (target range 200–700) when she was on a dose of 2 mg/kg/day in two divided doses. The reported blood level remained high despite reducing the dose of flecainide, so it was transpired that the laboratory carrying out the assay was using high performance liquid chromatography that was also detecting fluorescence from the concomitant use of propranolol.

Using gas chromatography instead, it was possible to separate the blood levels of the two antiarrhythmic drugs demonstrating a subtherapeutic level of flecainide. It is, therefore, important for the laboratory to be aware of all drugs being administered at the time of sampling and, equally, for clinicians to be aware of the type of assay used for sensible interpretation and sound clinical decision.

Probng of significance of ST-T segment alterations in patients with non-Q wave myocardial infarction

Sir,—I was interested to read the report by Ramires et al1 regarding the prognostic significance of T wave inversion and ST segment depression in patients with non-Q wave myocardial infarction; I have reported similar but slightly different results.2 Their results showed that the presence of ST segment depression, when compared with T wave inversion, is related to higher rates of short and long term cardiac events (9-6% and 30-8% v 0% and 9-8%), and mortality for the same observation periods (5-8% and 9-6% v 4-9% and 7-3%). However, prognostic implications for non-Q wave myocardial infarction seem to be differently related between ST depression and T wave inversion in my study. Mortality at one month was 41% in patients with ST depression and 0% in those with T wave inversion.

One possible reason for the difference between these results may originate in that Ramires et al excluded patients who developed either ST elevation or ST segment depression associated with tall R waves. In my observation ST elevation was recognised in the very acute phase (before T wave inversion) in 80% of patients, and was associated with preserved or reappearing R waves. Furthermore, most of the patients with ST depression showed preserved or normal R waves in leads with ST segment depression. Thus, some patients with typical non-Q wave myocardial infarction with ST depression or T wave inversion may have been excluded from their study.

I was equally interested to read a related paper regarding the morphology of T wave inversion by Agestuma and al2 who explained that the difference in the repolarisation property between the severely ischaemic area with a shortened action potential duration and the adjacent mildly ischaemic area with a prolonged duration of excitation may result in giant negative T waves. I suppose that the difference in repolarisation period between the mildly ischaemic area and the adjacent severely ischaemic subendocardial area was not sufficient to reverse the direction of T wave vector in surface electrocardiograms, in particular to cause giant negative T waves, although it may contribute to intensify the amplitude of negative T waves. Instead of the difference between the mildly ischaemic area and the severely ischaemic area, I feel that the difference in repolarisation period between the area with ischaemic (injured) myocardial cells associated with prolonged repolarisation and the non-ischaemic (non-injured) area with a normal repolarisation period is an important factor causing the negative T waves.

With regard to the mechanism of T wave inversion and ST depression in non-Q wave myocardial infarction, I speculated that T wave inversion does not reflect the presence of ischaemic or necrotic myocardial cells within the subendocardium. Instead, it suggests that injured myocardial cells, which are in the recovery phase from ischaemia and associated with the prolongation of the repolarisation period, are present in enough layers (transmural or near transmural layers) in a one-vessel territory of a one-vessel area wall to reverse the direction of the T wave vector between the injured and normal myocardium. On the other hand, ST depression in non-Q wave myocardial infarction reflects subendocardial ischaemia, mainly in multi-vessel territories, from the beginning of infarction, unlike T wave inversion, which appeared to start with transmural or near transmural ischaemia in a one-vessel territory. In both types of non-Q wave myocardial infarction, necrosis would develop in the subendocardial layer of each ischaemic lesion.

My colleagues and I have also recently reported the implications of persistent negative T waves and restored positive T waves following Q wave myocardial infarction.3 In this study we showed that persistent negative T waves indicated pathologically transmural infarction and restored positive T waves indicated non-transmural infarction. I believe “T wave inversion” is much more meaningful than currently used terms such as “negative T waves”.

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

We found very interesting Dr Maeda’s comments regarding the importance of the prognostic significance of T wave inversion in patients with non-Q wave