LETTERS TO THE EDITOR

Scope

Heart welcomes letters commenting on papers published in the journal in the previous six months. Topics not related to papers published earlier in the journal may be introduced as a letter: letters reporting original data may be sent for peer review.

Presentation

Letters should be:

- not more than 600 words and six references in length
- typed in double spacing (fax copies and paper copy only)
- signed by all authors.

They may contain short tables or a small figure. **Please send a copy of your letter on disk**. Full instructions to authors appear in the July 1997 issue of *Heart* (page 97).

Iatrogenic atrioventricular bypass tract following a Fontan operation for tricuspid atresia

SIR,—We read with interest the article by Rosenthal *et al*[1] that describes the creation of a functioning accessory connection by anastomosis of the atrial appendage to the right ventricular outflow tract in a patient with tricuspid atresia. We would like to draw the authors' attention to the previous description of this complication.[2] The patient in our report had electrocardiographic evidence of pre-excitation, recurrent supraventricular tachycardia, and successful surgical ablation of the functioning atrioventricular connection. We also reported that three of 21 patients with the Bjork modification[3] had new evidence of pre-excitation following surgery. We agree with Rosenthal *et al* that the patient reported by Case *et al*[4] was probably the second description of this interesting complication.

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1 Rosenthal E, Bostock J, Gill J. Iatrogenic atrioventricular bypass tract following a Fontan operation for tricuspid atresia. *Heart* 1997; 77:283–5.
2 Razzouk AJ, Gow R, Finley J, Murphy D, Williams WG. Surgically created Wolff-Parkinson-White syndrome after Fontan operation. *Ann Thorac Surg* 1992;54:974–7.
3 Bjork VO, Olin CL, Bjarke BB, Thoren CA. Right atrial-right ventricular anastomosis for correction of tricuspid atresia. *J Thorac Cardiovasc Surg* 1979;77:452–8.
4 Case CL, Schaffer MS, Dhala AA, Gillette PC, Fletcher SE. Radiofrequency ablation of an accessory atrioventricular connection in a Fontan patient. *Pacing Clin Electrophysiol* 1993;16:1434–6.

Flecainide levels—a cautionary note

SIR,—Monitoring blood levels of flecainide is essential particularly when administered 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 3200 μg/l (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. It 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.

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Prognostic 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 al*[1] 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 seemed significantly different 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 mechanism of T wave inversion by Agetsuma *et al*[3] 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 the 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 the ventricular 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 multivessel 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.[4] 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 recognised.

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1 Ramires JAF, Serrano CV, Solimene MC, Moffa PJ, Caramelli B, Pileggi F. Prognostic significance of ST-T segment alterations in patients with non-Q wave myocardial infarction. *Heart* 1996;75:582–7.
2 Maeda S. Different clinical implications for ST depression and T wave inversion in non-Q wave myocardial infarction. *J Cardiol* 1994; 24:357–66.
3 Agetsuma H, Hirai M, Hirayama H, Suzuki A, Takanaka C, Yabe S, *et al*. Transient giant negative T wave in acute anterior myocardial infarction predicts R wave recovery and preservation of left ventricular function. *Heart* 1996;75:229–34.
4 Maeda S, Imai T, Kuboki K, Chida K, Watanabe C, Ohkawa S. Pathological implications of restored positive T waves and persistent negative T waves after Q wave myocardial infarction. *J Am Coll Cardiol* 1996;28:1514–18.

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

We found very interesting Dr Maeda's comments regarding the importance on the prognostic significance of ST-T segment alterations in patients with non-Q wave