

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 modification³ 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.
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- 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 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.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

myocardial infarction.^{1,2} Both studies suggest that during a non-Q wave myocardial infarction the presence of ST-T segment changes in the diagnostic ECG could be a predictor of an adverse outcome. His results are more impressive on the notability of ST segment shift during the acute phase of a non-Q wave myocardial infarction. Dr Maeda noted a 41% one month mortality for patients that presented with ST segment depression and 0% mortality for patients with T wave inversion. In contrast, for the same study period we reported mortalities of 5.8% and 4.9%, respectively, for these groups of patients.

A possible explanation for the exceptional prognosis for T wave inversion myocardial infarction in Dr Maeda's study is the fact that 80% of his patients that presented with ST segment elevation in the very acute phase evolved to T wave inversion—with preserved or reappearing R waves. In accordance with Agetsuma *et al.*,³ the presentation of a giant negative T wave may predict both a return of the R wave and a better left ventricular function in patients in the chronic stage of anterior myocardial infarction. However, Agetsuma *et al.*'s study showed no significant differences in the rate of patency of the infarct related coronary artery. In our study, patency of the infarct related artery was much more frequent in patients with ST segment depression (76.9%) than those with T wave inversion (14.6%). Patients with patent infarct vessels are subjected to a higher incidence of subsequent ischaemic cardiac events than those with total occlusion of the infarct related artery as more residual myocardium is at risk.^{4,5} With respect to left ventricular dysfunction as a prognostic factor in our study, we noticed that both patients with T wave inversion myocardial infarction and ST segment depression presented similar and normal left ventricular ejection fractions.

Dr Maeda describes a severely dim first month post-myocardial infarction mortality for patients that presented with ST depression (41%) compared with patients in our study (5.8%). A conceivable justification is that some patients with non-Q wave myocardial infarction with ST depression may have been excluded from our study. However, other clinical variables and standard risk factors that have an important predictive value in risk stratification after a myocardial infarction must be considered.

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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 Scheinman MM, Abbott JA. Clinical significance of transmural versus nontransmural electrocardiographic changes in patients with acute myocardial infarction. *Am J Med* 1973; 55:602-7.
- 5 Rigo P, Murray M, Taylor DR, Weisfeldt ML, Strauss HW, Pitt B. Hemodynamic and prognostic findings in patients with transmural and nontransmural infarction. *Circulation* 1975;51,1064-70.

Stent placement in the outlet of the right ventricle

SIR,—Gibbs and colleagues have demonstrated the feasibility of stent placement in the abnormal, but normally connected, outflow to the right ventricle, and in discussion have drawn attention to possible mechanisms of stent failure including fracture.¹ We have placed stents in the reconstructed outflow (within a conduit, containing a homograft, between right ventricle and pulmonary artery) on three occasions with follow up of a year or more, and wish to draw attention to a complication in this group that may limit its application.

Recurrent balloon distensible obstruction at the proximal conduit anastomosis was demonstrated in a 7 year old girl who had had a previous conduit replacement following a Rastelli procedure for transposition of the great arteries, ventricular septal defect, and left ventricular outflow obstruction. This obstruction was overcome by placement of a single 12 × 300 mm stent delivered on a 12 × 40 mm balloon and fully distended. One year later (on reinvestigation for recurrent symptoms) the stent was shown to be severely deformed with a configuration similar to the original stenosis (fig).

We believe the deformation took place because the stent was placed effectively between a muscular dynamic structure (the original wall of the right ventricle) and a rigid structure (the back of the sternum). We would thus recommend care in stent placement in the reconstructed outflow, if the conduit lies behind, particularly if adherent to, the sternum. Such a relation would not be present with stent placement in a normally sited right ventricular outflow. This patient was included in a previous report.²

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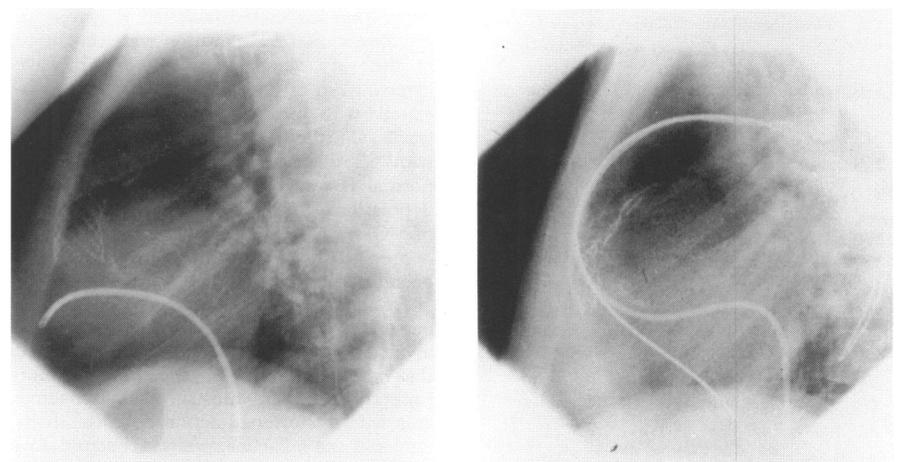
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Raised plasma BNP in a patient with acute pulmonary thromboembolism

SIR,—B-type or brain natriuretic peptide (BNP) is mainly secreted from the ventricles, and the plasma concentrations of BNP are increased more than those of A-type or atrial natriuretic peptide (ANP) in patients with chronic heart failure as well as in patients with acute myocardial infarction.¹⁻⁴ ANP and BNP play important roles in the improvement of cardiac function by vasodilatation, natriuretic action, and inhibition of the renin-angiotensin-aldosterone system and sympathetic nervous system in patients with heart failure.⁵ We present a case of acute pulmonary thromboembolism in which plasma BNP concentration was remarkably increased and thereafter decreased rapidly following treatment of pulmonary hypertension. To our knowledge, this is the first case in which plasma BNP was remarkably increased in a patient with right ventricular overload.

The patient was an 80 year old female who had suffered from varicose veins of the lower extremities and knee arthrosis. Two days before admission, she had dyspnoea when walking and, when admitted to hospital, even at rest. She was conscious and complained of severe dyspnoea with coldness in her fingers and toes. Echocardiography showed the right ventricle was remarkably enlarged but there was no intracardiac shunt. Pulmonary arterial pressure was 78/25 mm Hg and angiography revealed thrombi in the right and left pulmonary arteries. When the thrombus was partially suctioned through the catheter, pulmonary systolic pressure fell to 50 mm Hg. Urokinase (480 000 U) was administered into the pulmonary artery over 30 minutes and a further 480 000 U given over 24 hours. Heparin was given to maintain activated coagulation time at 150-200 seconds until warfarin treatment became effective. With the decrease of pulmonary arterial pressure, symptoms improved and the enlargement of the right ventricle disappeared in serial echocardiograms.

When the patient could walk without dyspnoea, two weeks after admission, angiography of the pulmonary arteries did not show any thrombus, and pulmonary arterial pressure was decreased to 32/10 mm Hg. Over the clinical course, serum creatine



Stent placement in a conduit from right ventricle to pulmonary artery (left) immediately after placement; (right) one year later showing deformation produced between the muscular right ventricular wall behind the stent, and the rigid sternum anteriorly.