

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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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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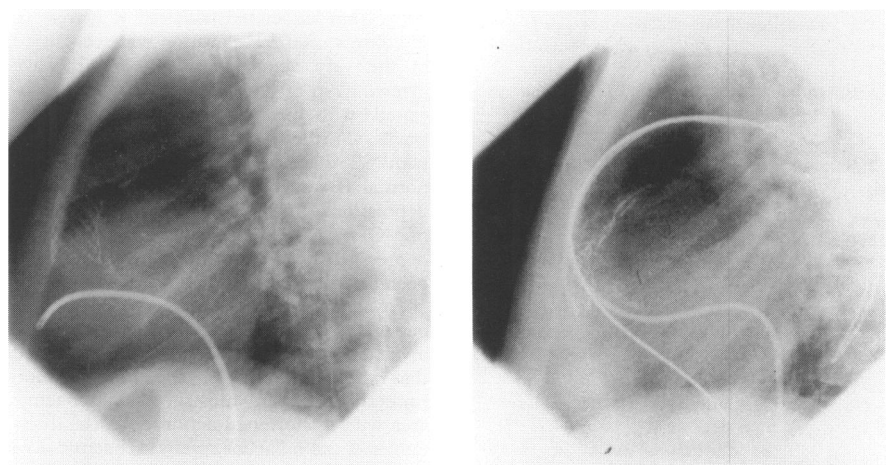
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Raised plasma BNP in a patient with acute pulmonary thromboembolism

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.