Letters to the Editor

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 1998 issue of Heart (page 104).

Atrioventricular plane displacement during low dose dobutamine infusion predicts recovery of left ventricular dysynergies

Sir,—We read with interest the study by Willenheimer et al regarding the prognostic significance of left atrioventricular plane displacement (AVPD) in patients with heart failure.1 This study showed that mortality in patients with heart failure is strongly related to systolic left atrioventricular plane motion. The movement of the left atrioventricular plane is a result of the longitudinal shortening of left ventricular fibres. During systole, the contraction of the longitudinal fibres of the left ventricle leads to a descent of the atrioventricular plane towards the relatively immobile cardiac apex. It should not be forgotten that assessment of left ventricular systolic function by cross sectional echocardiography is sometimes difficult because endocardium is inadequately visualised, especially in the elderly. In contrast, the mitral ring is distinctly outlined and easily studied by M mode recording,2 and, as it was related to mortality in cardiac failure patients, M mode echocardiography was recommended for general use.3

Willenheimer et al proposed further studies of the effects of different drugs on systolic AVPD. We recently assessed the value of left AVPD during low dose dobutamine infusion to predict functional improvement of asynergic infarcted myocardial areas after revascularisation in patients with heart failure caused by ischaemic heart disease.4 In healthy subjects we found a significant increase of left AVPD at all four recorded sites (septal, lateral, anterior, and inferior walls of the left ventricle) after dobutamine infusion. Patients also had a significant increase of left systolic AVPD during dobutamine infusion, but only in the dysynergic sites with functional improvement after revascularisation. In the dysynergic areas without functional improvement left AVPD did not change. Selecting a maximum left AVPD increase of more than 2 mm at any site of the left ventricle to predict recovery of the regional dysynergies resulted in a sensitivity of 91%, specificity of 83%, positive predictive value of 88%, and negative predictive value of 87%. Willenheimer et al found that mortality in patients with heart failure is related to systolic AVPD. We found that assessment of left systolic AVPD during low dose dobutamine infusion predicted left ventricular dysynergy recovery after revascularisation in patients with heart failure caused by ischaemic heart disease.

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

We read with great interest the letter by Kr


Changes in pulmonary artery size before and after total cavopulmonary connection

Sir.—We read with interest the paper of Buheitel et al dealing with the important topic of pulmonary artery size in children before and after total cavopulmonary connection.1 We congratulate the authors on their contribution to the ongoing discussion about the

fate of pulmonary arteries following various forms of right heart bypass operation. However, there are several issues related to the paper that were not clear.

In our opinion the authors do not provide enough information about the surgical technique and the type of palliative treatment of the patients either at primary palliation or during the so-called total cavopulmonary connection. We particularly miss data related to the type of systemic–pulmonary arterial shunt preceding the total cavopulmonary connection, the extent of surgical reconstruction of the central pulmonary arteries, or the use of atrial baffle fenestration. It is not clear whether the total cavopulmonary connection was done as a completion of previous semi-Fontan or bidirectional superior cavopulmonary connection. We believe that this information is crucial if the results of this study are to be compared with other published series.

The authors studied two distinctly different groups of patients, which deserve closer analysis. The first, much younger group of patients (group I; mean age 1.5 month) had severely hypoplastic pulmonary arteries (Z score of the right and left pulmonary arteries −6.0 and −9.6, respectively) at the time of their first cardiac catheterisation. One can only assume that these patients had very low pulmonary blood flow and that they went on to have some form of initial palliation to augment pulmonary blood flow. This provided sufficient pulmonary blood flow to enhance pulmonary arterial growth to reach normal values (mean Z score 0.5 and −0.5 for the right and left pulmonary artery) although remaining below the volume of systemic blood flow. These patients underwent a total cavopulmonary connection within a mean interval of 5.3 years. Initial pulmonary artery size before and after total cavopulmonary connection. Heart 1997;

We emphasise that the decrease in pulmonary artery size found during medium term follow up should not be interpreted as a lack of pulmonary artery growth. As we pointed out in our paper, there was turbulent flow in the central pulmonary arteries in almost all our patients before total cavopulmonary connection (with the exception of the three children who underwent a bi-directional Glenn anastomosis). The abolition of turbulent blood flow in the central pulmonary arteries could well explain their reduction in size following total cavopulmonary connection.

We certainly did not want to imply that an early total cavopulmonary connection will give a suboptimal result in terms of pulmonary arterial growth, and we have expressed this explicitly in our discussion.

We agree completely with Drs Slavik and Franklin that presently the clinical relevance of these changes in pulmonary artery growth after a Fontan-type repair remains unclear, as all our patients are in good clinical condition. Nevertheless, the growth of the central pulmonary arteries might have long term consequences on the long term outcome following a right heart bypass operation. Therefore, we believe that it is mandatory to obtain further information on the impact of a non-pulsatile flow pattern on pulmonary arterial growth. This applies particularly to children in whom a bidirectional Glenn procedure or a total cavopulmonary anastomosis is performed in infancy or early childhood. We plan a reassessment of the pulmonary arteries and the clinical condition of our patients in three to five years.

Serum concentration of cardiac troponin T in patients with cardiomyopathy: a possible mechanism of acute heart failure

We—We previously reported a group of patients with dilated cardiomyopathy associated with increased concentrations of serum cardiac troponin T (TnT) (measured using a first generation radioimmunoassay kit) and collagen. These patients had short-term
prognosis. Of 11 patients with positive serum concentrations of TnT or collagen, seven died before April 1998 while all 10 negative patients are currently stable in their clinical course. Five of the positive patients developed acute heart failure several times before death, complaining of dyspnoea and with hypotension and pulmonary congestion on chest radiography from compensated chronic heart failure without pulmonary congestion. The causes of decompensation of chronic heart failure to acute heart failure were unclear in most cases—there was no significant infection, no interruption in taking diuretics, and no drinking excess water. Although the mechanisms of decompensation of chronic heart failure to acute heart failure are unknown, five of our patients demonstrated continuously increased serum concentrations of TnT, suggesting ongoing subclinical myocyte degeneration even in the compensated stage of chronic heart failure. We concluded that subclinical myocyte degeneration occurs during compensated chronic heart failure and that this degeneration may lead some patients into acute heart failure.

Since April 1997, we have been using second-generation TnT assays, which are different from the first-generation assays and have a high specificity. Patients with dilated cardiomyopathy whose prognosis is poor have serum concentrations of TnT above 0.04–0.09 ng/ml as measured by the second-generation kit.

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Comparative study of chest pain characteristics in patients with normal and abnormal coronary angiograms

Sin—Owing to the age related prevalence of coronary heart disease (CHD), the observation that angiographically validated CHD is more common in patients aged ≥ 55 with "atypical" chest pain than in their younger counterparts, and that this is true for women aged ≥ 52 with negative exercise tolerance tests and ≥ 3 risk factors for CHD, is a validation of the proposition, consistent with Bayes' theorem, that the higher the prevalence of any disease, the greater the likelihood of atypical presentation. This is a concept that deserves more universal recognition to circumvent omissions of the type exemplified by a recent evaluation (among other considerations) of the diagnostic role of non-invasive nuclear imaging in patients with intermediate likelihood of CHD. This study did not test the hypothesis that the predictive accuracy of nuclear imaging could be age related. In the absence of such information, especially in view of the documentation of poor negative predictive value for the exercise tolerance test, the threshold for angiographic investigation should be lower in the old than in the young, especially in view of the necropsy validation of an age related increase in severity of individual coronary atherosclerotic lesions.

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Fax machines for thrombolysis

Sin—I hate to write a letter to the editor commenting on another letter to the editor, but I wish to comment not only on the remarks made by Hooghoudt and colleagues but also on the two original articles, which escaped my notice when first published.

Fax machines truly contribute to better patient care, not only in decisions regarding thrombolysis but also in other common clinical situations. On many occasions we see patients in the emergency department with chest pain and electrocardiographic abnormalities including Q waves and ST changes. But these changes could be "old," representing previous myocardial infarction or ventricular aneurysm rather than something acute. In the absence of a previous ECG, the usual plan would be to admit the patient to the coronary care unit, put the patient on a monitor, repeat the electrocardiography at frequent intervals, and draw serial blood samples for myocardial enzyme determinations to rule out an acute myocardial infarction. However, if we could see the previous ECGs with the old Q waves and/or ST changes for comparison, all these unnecessary and costly tests could be avoided.

Another clinical situation where a knowledge of any previous ECG abnormality would be critical in decision making is atrial fibrillation. Patients with chronic atrial fibrillation and well controlled ventricular rate often do not experience any cardiac irregularity or palpitation. Thus, when such a patient is seen for the first time with complaint of palpitation and is found to be in atrial fibrillation, one assumes the atrial fibrillation to be acute in onset. The differentiation between acute and chronic atrial fibrillation is particularly important if one is thinking about cardioversion without preceding anticoagulation. This problem could be easily and promptly resolved if the patient's previous ECG is made available for comparison. Therefore, the immediate availability of a previous ECG is extremely relevant, whether for consultation, comparison or confirmation, in most clinical situations. Fax machines can now accomplish this whether across town or around the world. They can really influence the way we practice medicine. As the Chinese saying goes, one picture is better than a thousand words. This is certainly true in the case of comparing ECGs.

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