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 1998 issue of *Heart* (page 104).

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

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,[2–4] and, as it was related to mortality in cardiac failure patients, M mode echocardiography was recommended for general use.[1]

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.[5] 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 dyssynergic sites with functional improvement after revascularisation. In the dyssynergic 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 dyssynergies 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 dyssynergy recovery after revascularisation in patients with heart failure caused by ischaemic heart disease.

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1 Willenheimer R, Cline C, Erhart L, *et al*. Left ventricular atrioventricular plane displacement: an echocardiographic technique for rapid assessment of prognosis in heart failure. *Heart* 1997;**78**:230–6.
2 Alam M, Hoglund C, Thorstrand C, *et al*. Atrioventricular plane displacement in severe congestive heart failure following dilated cardiomyopathy or myocardial infarction. *J Intern Med* 1990;**228**:569–75.
3 Kranidis A, Kostopoulos K, Margaris N, *et al*. Significance of echocardiographic atrioventricular plane displacement for the evaluation of left ventricular function and end diastolic pressure in patients with coronary artery disease. *Int J Cardiac Imaging* 1995;**11**:185–92.
4 Kranidis A, Kostopoulos K, Filippatos G, *et al*. Analysis of left atrioventricular plane movement during diastole in ischemic heart disease. *Jpn Heart J* 1995;**36**:545–56.
5 Kranidis A, Bouki T, Kostopoulos K, *et al*. The contribution of the left atrioventricular plane displacement during low dose dobutamine stress echocardiography in predicting recovery of left ventricular dyssynergies. *Echocardiography* 1996;**13**:587–97.

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

We read with great interest the letter by Kranidis *et al* concerning their study on the value of AVPD in predicting functional recovery following revascularisation of akinetic/dyskinetic myocardium.[1] These findings add further to the knowledge about the nature of AVPD.

Why is AVPD improved in response to dobutamine infusion? The most obvious answer seems to be that dobutamine improves contractility of hibernating subendocardial fibres. In addition, improved diastolic function may play a role. In 54 patients with heart failure we found a clear relation between reduced AVPD and impaired transmitral Doppler indices of left ventricular diastolic function, especially a short deceleration time of early diastolic flow (Willenheimer *et al*, unpublished data). In patients with similar fractional shortening—that is, contractility in circumferential fibres, AVPD was lower in those with more compromised diastolic function. We believe that our results indicate that impaired myocardial diastolic properties result in decreased long axis lengthening. Consequently, because the long axis shortening must equal the lengthening, AVPD is reduced; concurrently, diastolic transmitral flow is disturbed. Diastolic dysfunction might thus be the primary cause of impaired AVPD.

Our findings are somewhat supported by those of Henein and Gibson.[2] They suggested that, because of a primarily disturbed long axis function, incoordination between long and short axis function can result in asynchrony of left ventricular diastolic function and associated filling abnormalities. An increased AVPD in response to dobutamine stimulation may thus, at least partly, be the result of improved diastolic function, which may coincide with improved contractility of circumferential fibres.

An interesting finding in the study by Kranidis *et al* was that the regional (septal, lateral, inferoposterior, and anterior) AVPD increase during dobutamine infusion corresponded to the areas of functional recovery following revascularisation.[1] Alam *et al* found that, following exercise, regions of decreased AVPD corresponded to areas of reversible ischaemia on thallium scan.[3] Furthermore, Höglund *et al* found that regional AVPD at rest was decreased corresponding to the site of first time acute Q wave myocardial infarction.[4] These findings support a connection between regional AVPD and an area of ischaemic,[3] hibernating,[1] or infarcted[4] myocardium. In contrast, in 173 patients with coronary artery disease undergoing coronary angiography, regional AVPD at rest was not related to areas of coronary artery stenosis or areas of prior myocardial infarction, although AVPD was decreased corresponding to the degree and extent of coronary artery disease (Willenheimer *et al*, unpublished data).

The time elapsed between the ischaemic event and the assessment of AVPD may explain these contrasting findings. Short term alterations in the functional status of subendocardial, longitudinal fibres (in response to ischaemia or dobutamine infusion) may cause corresponding regional changes in AVPD. However, changes in functional status may, after some time, lead to an interdependence between longitudinal and circumferential fibres, smoothing out regional contractility differences, thus causing a more generalised decrease in AVPD. This hypothesis might not only explain the differences between our findings at rest and the findings by Kranidis *et al* and Alam *et al* using stress echocardiography,[1 3] but also the different findings by us and Höglund *et al* in the resting situation.[4] In the latter study, patients were examined shortly after an acute Q wave myocardial infarction, whereas most patients in our study were examined a longer time after a major ischaemic event.

The nature of AVPD is still largely unknown and somewhat confusing. Future research in this field will hopefully provide important insight into systolic and diastolic left ventricular function.

1 Kranidis A, Bouki T, Kostopoulos K, *et al*. The contribution of the left atrioventricular plane displacement during low dose dobutamine stress echocardiography in predicting recovery of left ventricular dyssynergies. *Echocardiography* 1996;**13**:587–97.
2 Henein MY, Gibson DG. Suppression of left ventricular diastolic filling by long axis asynchrony. *Br Heart J* 1995;**73**:151–7.
3 Alam M, Höglund C, Thorstrand C, *et al*. Effects of exercise on the displacement of the atrioventricular plane in patients with coronary artery disease. A new echocardiographic method of detecting reversible myocardial ischemia. *Eur Heart J* 1991;**12**:760–5.
4 Höglund C, Alam M, Thorstrand C. Effects of acute myocardial infarction on the displacement of the atrioventricular plane: an echocardiographic study. *J Intern Med* 1989;**226**:251–6.

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.[2 3] 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 techniques used in the 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 primary right heart bypass operation or as a completion of previous hemi-Fontan or bidirectional superior cavopulmonary anastomosis. We believe that this information is crucial if the results of this study are to be compared with other published series.[4 5]

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 3.34 years from the time of their first cardiac catheterisation, at the relatively young mean age of 3.46 years. This operation was followed by relatively poor pulmonary arterial growth (change of mean Z score −2.9 and −4.4 for the right and left pulmonary arteries).

The second, older group of patients (group II; mean age 10 months) had mildly underdeveloped pulmonary arteries at the time of their first cardiac catheterisation. One is left to speculate as to the proportions of this group who had either well balanced or increased pulmonary blood flow at first assessment, and how many received initial palliation to restrict pulmonary blood flow. Subsequent assessment demonstrated pulmonary blood flow in excess of systemic blood flow and pronounced enlargement of both pulmonary arteries (mean Z score > 8.0) over a much longer period of time (mean 7.3 years) leading to the total cavopulmonary connection. The ensuing reduction of high pulmonary blood flow to more physiological levels after this definitive procedure led to an encouraging return of the size of both pulmonary arteries to close to normal values (mean Z score 2.2 and −0.7 for the right and left pulmonary arteries).

These are certainly interesting findings, which to a certain extent support our previous conclusions[6] that candidates for any Fontan-type operation will initially fall into a group with hypoplastic pulmonary arteries or a group with adequate or even larger than normal pulmonary arteries at initial assessment. This division is in most cases matched by the volume of pulmonary blood flow. The aim of subsequent palliative procedures will be different in these groups of patients. Promotion of growth of originally hypoplastic pulmonary arteries is mandatory; however, the paper of Buheitel *et al* appears to imply that an early total cavopulmonary connection will give a suboptimal result in terms of pulmonary arterial growth. In patients with larger than normal pulmonary arteries, the role of an early total cavopulmonary connection is not clear. The relevance of these data to clinical outcome is also far from clear and is not addressed in this paper. We support the conclusion of the authors that more information about the long term development of pulmonary arteries following Fontan-type operations is required. We look forward to the next report on their patients' pulmonary arterial growth. However, the lack of correlation between the size of pulmonary arteries and pulmonary arteriolar resistance or clinical findings may question the value of central pulmonary arterial measurement for the long term outcome of patients after total cavopulmonary connection.

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1 Buheitel G, Hofbeck M, Tenbrink U, *et al.* Changes in pulmonary artery size before and after total cavopulmonary connection. *Heart* 1997;**78**:488–492.
2 Reddy VM, McElhinney DB, Moore P, *et al.* Pulmonary artery growth after bidirectional cavopulmonary shunt: is there a cause for concern? *J Thorac Cardiovasc Surg* 1996;**112**:1180–92.
3 Mendelsohn AM, Bove EL, Lupinetti FM, *et al.* Central pulmonary artery growth pattern after the bidirectional Glenn procedure. *J Thorac Cardiovasc Surg* 1994;**107**:1284–90.
4 Lundstrom U, Nisula L, Pesonen E, *et al.* Pulmonary artery growth failure after Fontan operation—cause and consequence [abstract]. *Cardiology in the Young* 1993;**3**(suppl 1):148.
5 Sigfusson G, Webber SA, Myers JL, *et al.* Growth of pulmonary arteries following Fontan operation: influence of age and type of connection [abstract]. *Circulation* 1995;**92**(suppl 2):55.
6 Slavik Z, Webber SA, Lamb RK, *et al.* Influence of bidirectional cavopulmonary anastomosis on pulmonary arterial growth. *Am J Cardiol* 1995; **76**:1085–7.

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

We thank Drs Slavik and Franklin for their interesting comments on our study. To answer the questions raised in their comment we provide some additional information.

We agree that possible candidates for a later Fontan-type operation can be divided initially into patients with diminished pulmonary blood flow and rather small pulmonary arteries, and patients with adequate or increased pulmonary blood flow and normal or even larger than normal pulmonary arteries. As we did not perform complete haemodynamic studies in all children at the time of initial cardiac catheterisation (especially in children with duct dependent pulmonary circulation), we were unable to provide information about Qp/Qs in all our children in the neonatal period or in early infancy. We decided not to include this incomplete information in our study and rather preferred to divide our patients into two groups based on the findings of the complete haemodynamic assessment before the total cavopulmonary connection.

In group I (patients with Qp/Qs ⩽ 1) nine of 16 children had one, four of 16 had two, and only three of 16 children had no palliative procedure before the creation of the total cavopulmonary connection. The palliative procedures included 11 systemic–pulmonary artery shunt procedures (using Goretex prostheses), three bidirectional cavopulmonary shunt procedures, two atrial septectomies, and only one pulmonary banding. Among children of group II (Qp/Qs > 1 before total cavopulmonary connection) 10 of 16 patients had one, three of 16 had two, and three of 16 had no palliative procedure. These palliative procedures included eight systemic–pulmonary artery shunts, one atrial septectomy, and seven pulmonary bandings.

The total cavopulmonary connection was performed using an intra-atrial tunnel without fenestration in all patients. Together with the total cavopulmonary connection, patch enlargement of a central pulmonary artery stenosis had to be performed in both groups with an equal frequency and therefore should have no major influence on our results (four children of group I and three children of group II). In 29 of 32 patients the total cavopulmonary connection was performed as a primary right heart bypass operation, in three of 32 (all group I) it was done as a completion following a bidirectional Glenn procedure.

Our study aimed to give a description of the behaviour of pulmonary arterial size following total cavopulmonary connection. 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 prior bidirectional 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 size 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 serious 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

SIR,—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.[1] These patients had poor 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 orthopnoea 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 about 0.04–0.09 ng/ml as measured by the second generation kit.

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1 Sato Y, Kataoka K, Matsumori A, *et al*. Measuring serum aminoterminal type III procollagen peptide, 7S domain of type IV collagen, and cardiac troponin T in patients with idiopathic dilated cardiomyopathy and secondary cardiomyopathy. *Heart* 1997;**78**:505–8.

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

SIR,—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,[1] and that this is true for women aged ⩾ 52 with negative exercise tolerance tests and ⩾ 3 risk factors for CHD,[2] is a validation of the proposition, consistent with Bayes's 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.[3] 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,[3] 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.[4]

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1 Cooke RA, Smeeton N, Chambers JB. Comparative study of chest pain characteristics in patients with normal and abnormal coronary angiograms. *Heart* 1997;**78**:142–6.
2 Curzen N, Patel D, Clarke D, *et al*. Women with chest pain: is exercising testing worthwhile? *Heart* 1996;**76**:156–60.
3 Berman D, Hachamovitch R, Lewin H, *et al*. Risk stratification in coronary artery disease: implications for stabilisation and prevention. *Am J Cardiol* 1997;**79**:10–16.
4 Elveback L, Lie JT. Continued high incidence of coronary artery disease at autopsy in Olmstead County, Minnesota, 1950–1979. *Circulation* 1984;**70**:345–9.

Fax machines for thrombolysis

SIR,—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[1] but also on the two original articles,[2 3] 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[4–6] 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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1 Hooghoudt THE, Drenth JPH, Lamfers EJP. Fool proof fax facilities: a valuable tool in thrombolysis decision making [letter]. *Heart* 1998;**79**:314.
2 Chamberlain D. Fax machines for thrombolysis? [editorial] *Heart* 1997;**78**:108.
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5 Cheng TO. Comparing ECGs. *South Med J* 1991;**84**:1507.
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