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:

• 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 January 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 Wil- lenheimer et al regarding the prognostic significance of left atrioventricular plane dis- placement (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 atrio- ventricular 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.2,3 and, as it was related to mortality in cardiac failure patients, m mode echocardiography was recommended for general use.4 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, which may coincide with improved contractility of circumferential fibres. An interesting finding in the study by Kra- nidis et al was that the regional (septal, lateral, inferoposterior, and anterior) AVPD increase during dobutamine infusion corresponded to the area of dobutamine recovery and associated filling abnormalities.6 These findings support a connection between regional AVPD and an area of ischaemic hibernating,1 or infarcted1 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 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 recovery test), may cause corresponding regional changes in AVPD. However, changes in functional status may, after some time, lead to an independ- ence between longitudinal and circumferen- tial 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 and the study by Kranidis et al and Alam et al using stress echocardiography,1 but also the different findings by us and Höglund et al in the rest- ing situation.7 In the latter study, patients were examined shortly after an area of acute 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.


Changes in pulmonary artery size before and after total cavo pulmonary 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 cavo pulmonary connection.1 We congratulate the authors on their contribu- tion 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 or the timing of 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 part of a right heart bypass operation as a completion of previous semi-Fontan or bili-rectangular superior cavopulmonary anastomosis. 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 months) 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 arteries) although remaining below the volume of systemic blood flow. These patients underwent a total cavopulmonary connection within a mean interval of 3.3 years (mean Z score 0.5 and −0.5 for the right and left pulmonary arteries) 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 can 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 physiologic 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).

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 the Fontan 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.

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 important questions 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 childhood. 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 septomectomies, 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, three atrial septomectomies 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 anastomosis we performed enlargement of a central pulmonary artery stenosis 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 II) 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 arterial 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 total 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 the changes in patients following 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 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

Sin.—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 poor hypopnœa and pulmonary congestion on chest radiography from compensated chronic heart failure without pulmonary congestion. The causes of deceleration 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 deceleration of chronic heart failure to acute heart failure are unknown, five of our patients demonstrated continuously increased serum concentrations of TnT, suggesting ongoing subclinical myocardial degeneration even in the compensated stage of chronic heart failure. We concluded that subclinical myocardial 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.

COMPARATIVE STUDY OF CHEST PAIN CHARACTERISTICS IN PATIENTS WITH NORMAL AND ABNORMAL CORONARY ANGIOGRAPHS


Fax machines for thrombolysis


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


Fax machines for thrombolysis


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

ATHANASIOS KRANIDIS, GERASIMOS FILIPPATOS, KOSTAS KAPPOLOS and LAMBROS ANTHOPOULOS

Heart 1998 80: 208
doi: 10.1136/hrt.80.2.208

Updated information and services can be found at:
http://heart.bmj.com/content/80/2/208.1

These include:

References
This article cites 9 articles, 2 of which you can access for free at:
http://heart.bmj.com/content/80/2/208.1#ref-list-1

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/