Abnormal patterns of intraventricular flow and diastolic filling after the Fontan operation: evidence for incoordinate ventricular wall motion

D J Penny, M L Rigby, A N Redington

Abstract
Objective—To assess whether regional abnormalities of ventricular function are present in patients after the Fontan operation and to explore the implications of such abnormalities for ventricular filling.

Design and patients—Prospective study in which 25 patients after the Fontan operation were compared with 25 healthy controls and 12 patients with a univentricular atrioventricular connection, before the Fontan operation.

Interventions—Doppler echocardiography, with simultaneous electrocardiogram, phonocardiogram, and respiratory tracing, to incoordinate ventricular filling patterns of diastolic filling and pulmonary and right ventricular wall motion during ventricular contraction and relaxation. The presence of this intraventricular flow correlates closely with abnormalities of regional wall motion, identified by cineangiography in patients with left ventricular disease.

When present during early systole, abnormalities of wall motion may have important implications for overall ventricular efficiency and rate of pressure development, whereas non-uniformity of ventricular wall motion during isovolumic relaxation may slow ventricular pressure decline, resulting in a prolongation of isovolumic relaxation time and a reduction in the rate of ventricular filling during early diastole. So far, regional ventricular wall motion or ventricular diastolic function have not been formally studied in patients after the Fontan operation. In this study, Doppler echocardiography was used to examine atrioventricular and intraventricular flow patterns in patients before and after the Fontan operation and in healthy controls, so that possible regional abnormalities of ventricular function might be explored.

Methods
PATIENTS
Twenty-five patients (14 male and 11 female) with a median age of 94 (range 12–221 months) were studied at a median interval of 28 (0.63–105) months after the Fontan operation. Fifteen patients had tricuspid atresia (with the ventriculoarterial connection concordant in 13, discordant in one, and double outlet in one). Eight patients had double inlet left ventricle (with the ventriculoarterial connection discordant in six and double outlet in two). One patient had pulmonary atresia with...
Results

TIME INTERVALS

The RR interval was similar in patients after the Fontan operation (708 (141) ms), normal children (704 (139) ms), and in patients with a univentricular atrioventricular connection before the Fontan operation (629 (90) ms). The isovolumic relaxation time, however, was significantly longer in patients after the Fontan operation (93-8 (14-5) ms) than in controls (55-2 (8-6) ms) (p < 0.0001) and the preoperative patients (66-4 (22-4) ms) (p = 0.001).

ATRIOVENTRICULAR FLOW

Atrioventricular Doppler flow showed early rapid E wave and atrial systolic A wave filling in all patients and controls, except in one patient with a univentricular atrioventricular connection before the Fontan operation, who had summation flow, so that E and A wave filling could not be separated for the purposes of analysis.

The E:A and the E:E + A velocity ratios were similar in patients before and after the Fontan operation (table). The E:A and the E:E + A ratios were higher in the normal children, than in both patient groups, however. The E wave velocity was significantly lower in patients after the Fontan operation than in either the controls or the preoperative patients. In patients after the Fontan operation an inverse relation was observed between isovolumic relaxation time and E wave velocity (p = 0.007). Whereas A wave velocity was similar in the control and post operative groups, A wave velocity was significantly higher in patients with a univentricular atrioventricular connection before the Fontan operation.

INTRAVENTRICULAR FLOW

Fifteen of the 25 patients after the Fontan operation (60%) showed the presence of intraventricular Doppler flow during early systole. Twenty patients had diastolic flow during isovolumic relaxation (80%), and 12 displayed both. Intraventricular flow was not detected in two patients after the Fontan operation.

SYSTOLIC INTRAVENTRICULAR FLOW

Systolic intracavitary flow (fig 1), as found in the patients after the Fontan operation, was directed from base to apex and had a mean velocity of 23-7 (5-26) cm/s. This flow began in early systole and persisted for 241-5 (59) ms after the onset of the QRS complex on the electrocardiogram—that is, 71 (11-2)% of total electromechanical systole. The intraventricular flow during systole was distinct from the atrioventricular flow signal and persisted after atrioventricular valve closure.

A similar pattern was detected during early systole in five of the 12 patients (42%) with a univentricular atrioventricular connection before the Fontan operation. This flow had a mean velocity of 23 (3-46) cm/s and lasted for 237 (65-7) ms after the onset of the QRS complex and was not significantly different from that detected in patients after operation. A lower velocity (15 cm/s) systolic flow, which
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Table: Atrioventricular velocity profiles and velocity ratios in the three groups of subjects studied. All indices are expressed as mean (SD). The statistical significance of differences between groups was analysed by Student’s t test.

<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th>Pre-Fontan</th>
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<td>25</td>
<td>25</td>
<td>NS</td>
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<tr>
<td>RR interval (ms)</td>
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<td>12</td>
<td>25</td>
<td>NS</td>
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<td>NS</td>
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<tr>
<td>E wave velocity (cm/s)</td>
<td>69.9 (18)</td>
<td>65.2 (36.7)</td>
<td>44.8 (17.1)</td>
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<td>NS</td>
<td>NS</td>
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<tr>
<td>A wave velocity (cm/s)</td>
<td>29.08 (11.67)</td>
<td>47.8 (23.6)</td>
<td>33.76 (12.6)</td>
<td>p = 0.003</td>
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<td>p = 0.025</td>
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<td>E:A ratio</td>
<td>2.6 (0.9)</td>
<td>1.4 (0.4)</td>
<td>1.4 (0.5)</td>
<td>p &lt; 0.001</td>
<td>NS</td>
<td>p &lt; 0.001</td>
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<td>E:E ratio</td>
<td>0.71 (0.07)</td>
<td>0.58 (0.07)</td>
<td>0.57 (0.08)</td>
<td>NS</td>
<td>p &lt; 0.001</td>
<td>NS</td>
</tr>
<tr>
<td>Isovolumic relaxation time (ms)</td>
<td>55.2 (6)</td>
<td>66.4 (22.4)</td>
<td>93.8 (14.5)</td>
<td>NS</td>
<td>p &lt; 0.001</td>
<td>NS</td>
</tr>
</tbody>
</table>

Figure 1: Pulsed wave Doppler velocity tracing with simultaneous electrocardiogram (ECG), phonocardiogram (PCG), and respirometer (RESP) in a patient after the Fontan operation. Base to apex. Systolic flow (S) is shown, which begins around the time of the first heart sound on the phonocardiogram (S1). There is continued atrioventricular flow during mid-diastole (diastasis flow) in this patient.

Figure 2: Pulsed wave Doppler velocity tracing in a patient after Fontan operation. Isovolumic relaxation flow (R) peaks at the time of the second heart sound on the phonocardiogram (S2), and persists until the development of early rapid ventricular filling. ECG, electrocardiogram; PCG, phonocardiogram; Resp, respirometer.

Fontan operation (80%) (fig 2). This peaked at or just after the time of aortic valve closure, had a mean velocity of 21.4 (6.7) cm/s, and ended with the development of atrioventricular flow. Intraventricular flow with a velocity of 10 cm/s was detected in two normal children (8%), but was not seen in any of the patients with a univentricular atrioventricular connection before the Fontan operation.

There was no relation between the duration of follow up and the presence of abnormal intraventricular flow. Respiration had no significant effect on intraventricular flow.

Discussion

This study shows the presence of two distinct patterns of intraventricular flow in patients after the Fontan operation; the first occurred during early systole and the second during isovolumic relaxation.

SYSTOLIC INTRAVENTRICULAR FLOW

In 15 of 25 patients (60%) an intraventricular Doppler signal, reflecting blood flow towards the apex, occurred in early systole. This was distinct from the atrioventricular flow, and persisted for about 70% of electromechanical systole. A similar signal was seen in five of 12 patients (42%) with a univentricular atrioventricular connection before the Fontan operation, but in only two of 25 normal children. Although the precise mechanism for this flow remains uncertain we postulate that as in other groups of patients with left ventricular disease, it occurs as a result of incoordinate ventricular contraction during early systole.

This hypothesis is supported by the angiographic data of Gibson and coworkers, which showed the presence of delayed onset of ventricular contraction and areas of regional hypokinesis in 11 of 20 patients with a univentricular atrioventricular connection. There was evidence of incoordinate relaxation in only four patients, although this study did not include patients after the Fontan operation.

The aetiology of this systolic incoordination may be related to an alteration in the arrangement of muscle fibres and ventricular geometry resulting from the underlying congenital heart lesion, or from chronic volume overload, or which may be secondary to myocardial necrosis or fibrosis manifest in the myocardium of some patients with congenital heart disease from an early age. It is not surprising that the incidence of systolic intraventricular flow is not increased by the Fontan operation, as neither afterload nor global ventricular systolic function seems to be significantly worsened.

lasted for only 145 ms after the onset of the QRS complex (42% of total electromechanical systole), was detected in two of the 25 normal children.

INTRAVENTRICULAR FLOW DURING ISOVOLUMIC RELAXATION

An early diastolic intraventricular base to apex flow signal was detected in 20 patients after the
INTRAVENTRICULAR FLOW DURING ISOVOLUMIC RELAXATION

By contrast with systolic flow, the incidence of intraventricular flow during isovolumic relaxation was much higher in patients after the Fontan operation (80%) than in the healthy children (89%) and in the children with a univentricular atrioventricular connection before the Fontan operation (0%). Again, the exact mechanism for this phenomenon is unclear, but intraventricular relaxation may be an explanation. M-mode echocardiography studies showed that an acute reduction in ventricular preload precipitated the development of incoordinate left ventricular relaxation in patients with ischaemic heart disease. Furthermore, in patients with left ventricular hypertrophy a similar pattern of base to apex intraventricular flow during isovolumic relaxation was attributed to incoordinate relaxation demonstrated by angiography. By separating the pulmonary and systemic circulations, the Fontan operation imposes an acute and dramatic preload reduction on a previously volume loaded ventricle. Furthermore, our own findings suggest that considerable systemic ventricular "hypertrophy" may develop at the time of the Fontan operation. Presumably this is related to an acute reduction in preload in a ventricle in which shortening fraction and muscle mass remain constant. Indeed in patients studied two months after this operation posterior wall thickness was increased. It thus seems reasonable to postulate that the diastolic flow in patients after the Fontan operation reflects the development of incoordinate ventricular relaxation resulting from an acute preload reduction and the development of a "hypertrophic" left ventricle at the time of operation.

VENTRICULAR RELAXATION AND ATRIOVENTRICULAR FLOW

Incoordinate ventricular relaxation results in an impairment of overall ventricular relaxation with prolonged isovolumic relaxation time, which may cause a reduction in atrioventricular flow velocity during early diastole. In our patients after Fontan operation, the isovolumic relaxation time was prolonged and early atrioventricular filling was lower than in healthy children and patients before operation.

Both E and A wave atrioventricular velocities were proportionately lower in postoperative patients, however, than in patients before the Fontan operation, so that E:A and E:E + A ratios were similar in the two groups. Thus the lower rate of ventricular filling in the patients after operation may merely reflect a reduction in total stroke volume consequent on separation of the systemic and pulmonary circulations. It would be premature to assume that these data provide evidence for changes in ventricular compliance characteristics resulting from the Fontan operation. The inverse relation between isovolumic relaxation time and early filling velocity in these patients suggests that impaired ventricular relaxation may be the primary cause of altered filling after this operation.

This study does provide evidence for the development of incoordinate ventricular relaxation in patients after the Fontan operation. We postulate that the underlying mechanism for these abnormalities is related to a combination of preload reduction and acquired ventricular hypertrophy. The presence of incoordinate ventricular relaxation may alter ventricular diastolic function, which in turn may influence pulmonary blood flow after the Fontan operation. The possible influence of abnormal ventricular relaxation on diastolic filling and cardiac output after the Fontan operation warrant further study.

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