Effect of Veno-venous Ultrafiltration on Myocardial Performance Immediately After Cardiac Surgery in Children

A Prospective Randomized Study

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Objectives. This study sought to evaluate the effects of veno-venous ultrafiltration on myocardial contractility in children undergoing cardiopulmonary bypass (CPB) for repair of congenital heart defects.

Background. Ultrafiltration (UF) is currently used to diminish postoperative fluid accumulation following CPB in children. Previous reports indicate improvement in hemodynamics immediately after UF, but the mechanism of its action is unknown.

Methods. Twenty-three patients (ages 2 months to 9.1 years; 13 males, 10 females) underwent UF for 10 min after CPB. Twelve patients underwent UF immediately after CPB (Group A). They were studied: (1) before and (2) after CPB, (3) after UF, and (4) 10 min after UF. Eleven patients underwent UF 10 min after CPB (Group B). They were studied: (1) before and (2) after CPB, (3) after a 10-min delay before UF, and (4) after UF. Contractility was determined by the difference in the observed and predicted velocity of circumferential fiber shortening for the measured wall stress, using transesophageal echocardiography. Left ventricular wall thickness was also measured.

Results. There was significant improvement in contractility after UF in both groups (mean ± SD, Group A: −0.28 ± 0.13 to −0.01 ± 0.21 circ/s, p < 0.05; Group B: −0.26 ± 0.16 to −0.11 ± 0.17 circ/s, p < 0.05). Myocardial thickness to cavity dimension decreased in both groups following UF (Group A: 0.19 ± 0.04 to 0.14 ± 0.03, p < 0.05; Group B: 0.18 ± 0.04 to 0.14 ± 0.03, p < 0.05).

Conclusions. UF improves hemodynamics by improving contractility and possibly by reducing myocardial edema in children following cardiac surgery. Enhanced patient outcome after ultrafiltration may in part be due to these changes.

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Children undergoing cardiopulmonary bypass (CPB) surgery for repair of congenital heart defects often suffer from capillary leak syndrome. This is brought about by the whole body inflammatory response to abnormal conditions during CPB and leads to an increase in total body water, tissue edema, and organ dysfunction, especially of the myocardium (1–3). Several treatment modalities, such as extensive use of diuretics, colloid administration, peritoneal dialysis, and ultrafiltration (UF), have been used in an attempt to ameliorate these harmful effects (4,5).

Investigators have found that arterio-venous UF is a safe and effective way of removing body water to correct hemodilution during CPB (6–8). Naik et al. (4,5) modified the procedure performing arterio-venous UF after CPB. They reported improved hemodynamics and better patient outcome after the procedure. Although it has been shown that UF improves LV performance and removes body water, the mechanisms of these improvements are not known.

Determination of myocardial contractility has been traditionally obtained by construction of pressure-volume loops utilizing invasive monitoring (9). Colan et al. (10,11) have shown that the relation of end-systolic relation of wall stress (WS) or fiber stress (FS) to corrected velocity of circumferential fiber shortening (VCFc) are reliable indexes of myocardial contractility that are independent of preload, corrected for heart rate and incorporate afterload in a noninvasive manner using echocardiography.

The purpose of this randomized study was to determine the effect of veno-venous UF on myocardial contractility after CPB in children utilizing the echocardiographic stress-shortening relation.

Methods

The study protocol was approved by the Institutional Review Board, and parental informed consent was obtained for each subject.

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Patients. All patients weighing more than 4.0 kg undergoing CPB for repair of congenital heart defects with anticipated biventricular outcome and with the LV in the systemic position were eligible to participate in the study. Ten females and 13 males, ages 2 months to 9 years, were included from May 1995 to November 1995.

The standard surgical, anesthetic, and CPB techniques used in our institution at the time of the study were employed. Myocardial protection was obtained by multidose crystalloid cardioplegia. Several covariates that could influence myocardial function, including age, weight, body surface area, diagnoses, repair procedure, duration of CPB, duration of aortic cross clamp, duration of UF, volume of prime circuit, volume of ultrafiltrate, and medications after CPB, were measured. Patients were excluded if they could not be weaned off CPB after surgery, and thus could not receive the intervention.

Protocol (Fig. 1). In order to reduce the possibility that time alone was accounting for the changes following CPB, the patients were randomized after CPB into two groups. Group A (n = 12) had veno-venous UF immediately after weaning off CPB, and Group B (n = 11) had veno-venous UF 10 min after weaning off CPB. This grouping allowed all patients to undergo UF, the endpoint of UF was determined by the surgeon based on the volume remaining in the pump reservoir, hematocrit, or 10 minutes of UF, whichever was earlier.

Echocardiographic techniques. A Hewlett-Packard Sonos 2500 Ultrasound Imaging System with a Hewlett-Packard biplane 9-mm tip 7.5/5.5 MHz transesophageal echocardiography (TEE) probe was used. The TEE probe was inserted in accordance to previously set guidelines after the airway and all monitoring lines were secured (12,13). The probe was positioned in the stomach and anteflexed to obtain a short axis view of the left ventricle at the level of the mid-papillary muscles. M-mode echocardiography with superimposed arterial tracings from an arterial catheter placed in the radial artery were obtained at each study time.

Echocardiographic indices. All indices in each study time were obtained using three consecutive cardiac cycles without arrhythmia. The indices were measured by a single investigator (ESR) blinded to patient group.

Left ventricular systolic performance. Shortening fraction (SF) and VCFc, derived by M-Mode echocardiography served as indices of left ventricular performance. SF was derived by the following formula:

\[ \text{SF} (%) = \left( \frac{\text{LVED} - \text{LVES}}{\text{LVED}} \right) \times 100, \]

where LVED = left ventricular end-diastolic dimension; LVES = left ventricular end-systolic dimension. The observed VCFc was measured as follows:

\[ \text{VCFc} = \left( \frac{\text{SF} \times R}{100} \right) / ET, \]

where SF = shortening fraction; ET = ejection time measured in seconds; RR = R to R interval measured in seconds.

LV mass and wall thickness. LV mass was calculated by previously described techniques (14). LV wall thickness can change not only because of a primary change in wall thickness (e.g., edema), but also because of a secondary effect of a change in LV cavity volume (e.g., decreased preload). Therefore, we chose to measure the ratio between wall thickness and
LV dimension, which is a more meaningful measure to discriminate between these effects. This ratio was derived by dividing M-mode measurements of LV end-diastolic wall thickness (hₜ) by LV end-diastolic dimension (LVED).

**Left ventricular afterload.** End-systolic wall stress (WS) served as an index of afterload (14). This was derived by M-mode measurements as follows:

\[ WS = (1.35 \times P_{es} \times \text{LVES}) + \left[ 4 \times h_{es} \times \{1 + (h_{es}/\text{LVES})\} \right] \]

where \(1.35\) = conversion factor for mm Hg to g/cm²; \(P_{es}\) = end-systolic pressure derived from linear interpolation of the arterial pulse trace to the dicrotic notch; \(\text{LVES}\) = left ventricular end-systolic dimension; \(h_{es}\) = end-systolic wall thickness.

As Regan points out (15), wall stress provides an estimate of fiber pulling force, which is biased on wall thickness. Fiber stress provides an unbiased estimate of the forces resisting shortening regardless of wall thickness. It was measured from M-mode data as follows:

\[ \text{FS} = (1.35 \times P_{es} \times b_{m}) / (2 h_{es}) \]

where \(b_{m}\) = midwall cavity dimension at end-systole = \(h_{es}/\ln(\text{LVES}/2 + h_{es}) - \ln(\text{LVES}/2)\); \(P_{es}\) = end-systolic pressure; \(h_{es}\) = end-systolic wall thickness; \(\text{LVES}\) = left ventricular end-systolic dimension; \(1.35\) = conversion factor from mm Hg to g/cm².

**Contractility.** The difference between the measured and predicted VCFc for measured WS, \(\Delta \text{VCFc} - \text{WS}\), served as an index of contractility for each patient in each study period. The predicted VCFc for age for measured wall stress was obtained from our previously studied normal patients (16). In addition, these same normal patients served as the population for which the normal VCFc to fiber stress was calculated and regression equations were derived. These regression equations were then used to obtain the predicted VCFc for the measured fiber stress in the study population.

Since wall stress provides an estimate of fiber pulling force, which is biased on wall thickness, acute changes in wall thickness may artifactually effect its measurement and the measurement of contractility. Therefore, the VCFc to fiber stress relationship was also established in this normal patient population as well. The difference between the measured and predicted VCFc for measured fiber stress, \(\Delta \text{VCFc} - \text{FS}\), was also used as an index of contractility.

Even when contractility is calculated using fiber (rather than wall) stress, errors may occur because of acute changes in wall thickness. Stress (either wall stress or fiber stress) is fundamentally dependent on pressure and dimension and inversely dependent on thickness \([\text{Stress} \propto (P)(D)/(h)]\). If pressure and dimension remain constant but thickness increases because of edema, the calculated stress will appear to be lower. True stress will be underestimated. An underestimation of stress will result in an underestimation of contractility. In reality, an increase in myocardial thickness after cardiopulmonary bypass does not denote existence of additional myofibers developed acutely to carry the load but rather reflects increase in myofilibril density. Therefore, contractility was calculated by a third method. In this method, we assumed that mass was conserved during cardiopulmonary bypass. End-systolic wall thickness at Time 1 (before surgery and, therefore, before edema) was assumed to be the dry-weight thickness and therefore assumed to reflect the most “normal” myofiber density. Wall and fiber stress at Times 2, 3, and 4 were then calculated using wall thickness at Time 1. Contractility was then recalculated using these new stress values \((\Delta \text{VCFc}, \text{“dry”})\).

**Statistical analysis.** All data are presented as mean ± SD. A contrast transformation using repeated measures analysis of variance was utilized with each time point compared to the baseline study within each group (SAS GLM Contrast transformation within the Repeated statement). The repeated measures analysis was supplemented with paired t tests for other relevant comparisons between time points. A2 and A3 data were compared to determine changes due to UF in Group A, and B2 and B3 data were compared to determine changes due to UF alone in Group B. A2 and A3 data were compared to determine changes due to time in Group A, and B3 and B4 were compared to determine changes due to UF in Group B. Finally, A3 data were compared with B4 data to determine the difference in contractility due to immediate versus delayed UF. Adjustments to p values were not made for multiple comparisons.

**Results**

**Patient profiles (Table 1).** The study groups A and B were comparable in demographics, length of procedure, UF dura-
There was a significant increase in heart rate in both groups. Baseline heart rate was similar in the two groups (p = 0.5). There was no significant difference in the different study periods in each group. There was no significant difference in the medications used between the two groups. One patient was excluded from the study because of inability to wean off CPB eventually requiring extracorporeal oxygenation.

Echocardiographic indices (Table 2). Heart rate. The baseline heart rate was similar in the two groups (p = 0.77). There was a significant increase in heart rate in both groups after CPB (p = 0.001). There was no significant difference between the two groups in each study period. There was no significant difference in the different study periods in each group after CPB.

Blood pressure (Fig. 2). The baseline systolic, diastolic, and end-systolic blood pressures (BP) were similar in the two groups (p = 0.65, 0.32, and 0.25, respectively). There was a significant decrease in systolic and diastolic BP in both groups after CPB (p = 0.002 and 0.01, respectively). The end-systolic BP decreased after CPB in both groups, significantly so in Group A (p = 0.001). Comparing the change from A2 to A3 with that from B2 to B3, the systolic, diastolic, and end-systolic BP increased with UF compared with no UF (p = 0.02, 0.003, 0.04, respectively).

Figure 2. Changes in systolic blood pressure during the different study times. There was significant fall in systolic blood pressure after CPB followed by significant increase in blood pressure after UF in both groups.

<table>
<thead>
<tr>
<th>Table 2. Echocardiographic Values</th>
<th>Grp/Time</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
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<tbody>
<tr>
<td>HR (bpm)</td>
<td>A</td>
<td>116 ± 22</td>
<td>151 ± 20</td>
<td>136 ± 20</td>
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<tr>
<td></td>
<td>B</td>
<td>111 ± 17</td>
<td>153 ± 11</td>
<td>136 ± 19</td>
<td>129 ± 17</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>A</td>
<td>86 ± 13</td>
<td>69 ± 11</td>
<td>101 ± 8</td>
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<tr>
<td></td>
<td>B</td>
<td>83 ± 13</td>
<td>72 ± 15</td>
<td>79 ± 15</td>
<td>92 ± 14</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>A</td>
<td>45 ± 9</td>
<td>37 ± 8</td>
<td>61 ± 9</td>
<td>59 ± 6</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>45 ± 10</td>
<td>38 ± 3</td>
<td>45 ± 10</td>
<td>52 ± 10</td>
</tr>
<tr>
<td>Psa (mm Hg)</td>
<td>A</td>
<td>65 ± 9</td>
<td>45 ± 9</td>
<td>78 ± 12</td>
<td>75 ± 8</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>59 ± 14</td>
<td>53 ± 16</td>
<td>56 ± 10</td>
<td>68 ± 13</td>
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<tr>
<td>LVED (cm)</td>
<td>A</td>
<td>2.9 ± 0.9</td>
<td>2.9 ± 0.8</td>
<td>3.1 ± 0.8</td>
<td>2.9 ± 0.6</td>
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<tr>
<td></td>
<td>B</td>
<td>3.4 ± 0.8</td>
<td>3.6 ± 0.8</td>
<td>3.2 ± 0.7</td>
<td>3.4 ± 0.7</td>
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<td>h0 (cm)</td>
<td>A</td>
<td>0.44 ± 0.14</td>
<td>0.53 ± 0.09</td>
<td>0.43 ± 0.07</td>
<td>0.45 ± 0.14</td>
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<td></td>
<td>B</td>
<td>0.46 ± 0.09</td>
<td>0.60 ± 0.13</td>
<td>0.59 ± 0.16</td>
<td>0.48 ± 0.09</td>
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<tr>
<td>h0/LVED</td>
<td>A</td>
<td>0.15 ± 0.03</td>
<td>0.19 ± 0.04</td>
<td>0.14 ± 0.03</td>
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<td></td>
<td>B</td>
<td>0.14 ± 0.03</td>
<td>0.17 ± 0.03</td>
<td>0.18 ± 0.04</td>
<td>0.14 ± 0.03</td>
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<td>LV mass (g)</td>
<td>A</td>
<td>31 ± 26</td>
<td>34 ± 19</td>
<td>29 ± 18</td>
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<tr>
<td></td>
<td>B</td>
<td>35 ± 18</td>
<td>54 ± 32</td>
<td>49 ± 33</td>
<td>40 ± 21</td>
</tr>
<tr>
<td>ET (msec)</td>
<td>A</td>
<td>277 ± 57</td>
<td>229 ± 49</td>
<td>232 ± 48</td>
<td>215 ± 40</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>284 ± 54</td>
<td>239 ± 32</td>
<td>250 ± 46</td>
<td>259 ± 40</td>
</tr>
<tr>
<td>SF (%)</td>
<td>A</td>
<td>43 ± 9</td>
<td>34 ± 8</td>
<td>36 ± 8</td>
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<tr>
<td></td>
<td>B</td>
<td>42 ± 7</td>
<td>34 ± 6</td>
<td>35 ± 6</td>
<td>35 ± 7</td>
</tr>
<tr>
<td>WS (g/cm²)</td>
<td>A</td>
<td>33 ± 17</td>
<td>26 ± 9</td>
<td>49 ± 18</td>
<td>49 ± 17</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>32 ± 14</td>
<td>27 ± 9</td>
<td>28 ± 7</td>
<td>45 ± 25</td>
</tr>
<tr>
<td>ΔVCFc – WS (circ/s)</td>
<td>A</td>
<td>-0.02 ± 0.16</td>
<td>-0.28 ± 0.13</td>
<td>-0.01 ± 0.21</td>
<td>-0.02 ± 0.21</td>
</tr>
<tr>
<td>(g/cm²)</td>
<td>B</td>
<td>-0.07 ± 0.18</td>
<td>-0.26 ± 0.12</td>
<td>-0.26 ± 0.16</td>
<td>-0.11 ± 0.17</td>
</tr>
<tr>
<td>FS (g/cm²)</td>
<td>A</td>
<td>68 ± 20</td>
<td>51 ± 14</td>
<td>92 ± 23</td>
<td>91 ± 20</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>64 ± 22</td>
<td>56 ± 13</td>
<td>58 ± 12</td>
<td>83 ± 33</td>
</tr>
<tr>
<td>ΔVCFc – FS (circ/s)</td>
<td>A</td>
<td>-0.18 ± 0.19</td>
<td>-0.42 ± 0.19</td>
<td>-0.22 ± 0.26</td>
<td>-0.19 ± 0.29</td>
</tr>
<tr>
<td>&quot;Dry&quot; wall stress (g/cm²)</td>
<td>B</td>
<td>-0.22 ± 0.21</td>
<td>-0.40 ± 0.17</td>
<td>-0.40 ± 0.20</td>
<td>-0.31 ± 0.14</td>
</tr>
<tr>
<td>Δ VCFc – &quot;dry&quot; (circ/s)</td>
<td>A</td>
<td>-0.08 ± 0.20</td>
<td>-0.18 ± 0.21</td>
<td>0.18 ± 0.47</td>
<td>0.26 ± 0.52</td>
</tr>
<tr>
<td>(g/cm²)</td>
<td>B</td>
<td>-0.13 ± 0.21</td>
<td>-0.16 ± 0.14</td>
<td>-0.18 ± 0.16</td>
<td>0.03 ± 0.30</td>
</tr>
</tbody>
</table>

Data are mean ± SD. DBP = diastolic blood pressure; ET = ejection time; HR = heart rate; Psa = end systolic blood pressure; SBP = systolic blood pressure; ΔVCFc = WS = difference between measured and predicted VCFc for measured WS; ΔVCFc = FS = difference between measured and predicted VCFc for measured fiber stress.
LV mass and wall thickness. LV mass significantly decreased with UF use in both groups. The baseline $h_d$/LVED was similar in the two groups ($p = 0.6$). After CPB, the $h_d$/LVED was significantly increased in both groups ($p = 0.01$), indicating an increase in ventricular wall thickness. Group A, which received UF earlier, showed a significant decrease in ventricular wall thickness immediately after UF, compared with Group B, which did not undergo UF at the same time point ($p = 0.002$). Ventricular wall thickness in Group B eventually decreased significantly after the delayed UF ($p = 0.006$). There was no significant difference between the final $h_d$/LVED in each group ($p = 0.57$) (Fig. 3).

Shortening fraction. The baseline SF was similar in the two groups ($p = 0.6$). There was a significant decrease in the SF after CPB in both groups ($p = 0.008$). There was no significant difference in the SF between the two groups after UF. There was also no significant difference in the different study points within the same group after CPB.

Myocardial contractility (Figure 4). The baseline myocardial contractility using the wall stress-velocity relation was similar in the two groups ($p = 0.33$). There was a significant decrease in contractility in both groups after CPB ($p < 0.001$). There was significant increase in contractility in both groups after UF ($p = 0.04$). Contractility improved greatly between study times A2 and A3 (period of UF), ($p = 0.002$) but did not change between study times B2 and B3 (period of observation), ($p = 0.53$). Contractility was restored immediately in Group A but not in Group B, in whom UF was delayed. Contractility improved in Group B after UF ($p = 0.01$). Though the degree of improvement in UF appeared to be greater in Group A, the difference between A4 and B4 was not significant ($p = 0.11$). Contractility results using the VCFc to fiber stress relation showed the exact same trends as the contractility results using the VCFc to wall stress relation (Table 2). Finally, contractility using the conservation of mass techniques based on “dry” wall stress also showed the same trends as contractility results based on traditional wall and fiber stress equations. For example, contractility was similar in the two groups at Time 1 ($-0.08 \pm 0.1$ for Group A vs. $-0.13 \pm 0.2$ for Group B, $p = 0.57$). Contractility significantly increased in both groups after UF ($p < 0.03$). Importantly, although contractility decreased in both groups following bypass, the difference was not statistically significant. This suggests that the decrease in contractility with bypass using the traditional wall stress calculation may be spurious because this calculation underestimates actual wall stress because of wall edema. However, the increase in contractility associated with UF remained statistically significant even while using the “dry” wall stress modification. This suggests that there is a true increase in contractility resulting from UF use.

Discussion

The significant findings of this study are that UF following open heart surgery in children results in improvement in myocardial contractility and reduction in myocardial wall thickness. These findings are important because they help to explain the hemodynamic improvement seen after UF and may suggest other possible uses of UF.

Pathophysiology of cardiopulmonary bypass. CPB is an unnatural condition leading to changes that may result in unfavorable hemodynamics. The physical attributes of the myocardium itself in the form of its thickness and contractility are altered to the detriment of the patient. The activated leukocytes brought about by the contact of blood with foreign material in the form of tubings and pumps release a variety of cytotoxic products such as lysosomal hydrolases, neutral proteases, and arachidonic acid, some of which have been shown to increase vascular permeability (17). During controlled hypothermic cardiac arrest, there may be impairment of the transmembrane fluid transport brought by hypothermia, which also predisposes the heart to further fluid accumulation. Ischemia has also been shown to predispose the myocardium to pathologic fluid accumulation after restoration of coronary flow (18).

Several substances such as endothelin have been found to be elevated following CPB that are vasoactive or cardioactive,
or both (19). The role of some of these substances on myocardial contractility is still in question.

Ultrafiltration effects on ventricular performance. Studies of adults utilizing pressure-area loops demonstrated a decrease in LV performance and an increase in chamber stiffness immediately after CPB without UF (20,21). Previous work by Naik et al. (22) and Elliott et al. (23) using arterio-venous UF immediately after CPB in children revealed uniform improvement in systolic BP, associated with marked increase in cardiac index, decrease in heart rate, and decrease in pulmonary vascular resistance, without changes in systemic vascular resistance.

Mechanisms of action of ultrafiltration We have shown that with UF there are significant decreases in myocardial wall thickness and mass and a significant increase in myocardial contractility. We believe that the decrease in myocardial wall thickness and thickness/dimension ratio likely represent a decrease in myocardial edema. Although cardiac output was not measured, we believe that the increase in systolic blood pressure following UF probably reflects an increase in cardiac output resulting from enhanced contractility. Lack of change in contractility during the observation period (study times B2 to B3 and A3 to A4) strongly eliminates “recovery time” as the reason for improved contractility during UF (study times A2 to A3 and B3 to B4). It also appears that early UF may improve myocardial contractility to a higher degree than delayed UF. A causality between lesser myocardial wall thickness and improved myocardial contractility could be inferred but not definitely concluded; the increase in myocardial contractility may not be due to a decrease in myocardial edema alone. Several previous studies have shown that UF removes certain factors or molecules that could alter the contractile state (1,19).

An alternative explanation for improved systolic BP after UF may be that myocardial edema is reduced, improving compliance, increasing ventricular filling, and resulting in improved output and blood pressure. Regardless of the exact mechanism, venovenous UF improves myocardial contractility and decreases wall thickness, which may result in improved hemodynamics and patient outcome.

Limitations of the study. Some of the patients were receiving vasoactive medications immediately after CPB, which could affect some of the parameters measured. There was no difference in the use of these medications between the two groups. Attempts were also made for the inotropic infusion to have reached a steady state before beginning study time 2.

Smaller patients who usually retain more water after CPB (1,22) and who may have benefited more from UF were not included in the study because the 9-mm TEE probe was the smallest probe available in our institution, and it was felt that inserting it in patients less than 4 kg would have unduly increased the risk of procedure. Extrapolation of the results of this study to smaller patients may be possible, but not fully conclusive.

The absence of a pure control group that did not undergo UF is a limitation of the study. No conclusions can be made regarding the long-term effects of UF.

Estimation of end-systolic BP may be limited because of the use of the intraradial artery pulse tracings, which may be affected by vasconstriction. Direct aortic and indirect carotid and axillary pulse tracings are more accurate but were not feasible or appropriate in the context of this study.

Conclusions. Ultrafiltration improves hemodynamics by improving myocardial contractility and reducing ventricular wall thickness. The improvement in myocardial contractility by ultrafiltration is likely due to a decrease in myocardial edema in combination with removal of certain cardio-depressant substances brought about by CPB. Also, this study reaffirms the role of this modality in the postoperative management of children following open heart surgery. This modality may also play a role in some disease states with capillary leak such as septic shock. This study also demonstrated the feasibility of semi-invasive quantitation of myocardial contractility intraoperatively.

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References