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# Tissue Doppler imaging following paediatric cardiac surgery: early patterns of change and relationship to outcome<sup>☆</sup>

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## Abstract

In this study, tissue Doppler imaging (TDI) was used to assess changes in ventricular function following repair of congenital heart defects. The relationship between TDI indices, myocardial injury and clinical outcome was explored. Forty-five children were studied; 35 with cardiac lesions and 10 controls. TDI was performed preoperatively, on admission to paediatric intensive care unit (PICU) and day 1. Regional myocardial Doppler signals were acquired from the right ventricle (RV), left ventricle (LV) and septum. TDI indices included: peak systolic velocities, isovolumetric velocities (IVV) and isovolumetric acceleration (IVA). Preoperatively, bi-ventricular TDI velocities in the study group were reduced compared with normal controls. Postoperatively, RV velocities were significantly reduced and this persisted to day-1 (PreOp vs. PICU and day-1:  $7.7 \pm 2.2$  vs.  $3.4 \pm 1.0$ ,  $P < 0.0001$  and  $3.55 \pm 1.29$ ,  $P < 0.0001$ ). LV velocities initially declined but recovered towards baseline by day-1 (PreOp vs. PICU:  $5.31 \pm 1.50$  vs.  $3.51 \pm 1.23$ ,  $P < 0.0001$ ). Isovolumetric parameters in all regions were reduced throughout the postoperative period. Troponin-I release correlated with longer X-clamp times ( $r = 0.82$ ,  $P < 0.0001$ ) and reduced RV velocities ( $r = 0.42$ ,  $P = 0.028$ ). Reduced pre- and postoperative LV velocities correlated with longer ventilation (PreOp:  $r = 0.54$ ,  $P = 0.002$ ; PostOp:  $r = 0.42$ ,  $P = 0.026$ ). This study identified reduced postoperative RV velocities correlated with myocardial injury while reduced LV TDI correlated with longer postoperative ventilation.

**Keywords:** Tissue Doppler imaging; Isovolumetric; Paediatric cardiac surgery; Troponin-I

## 1. Introduction

Advances in surgical technique and ITU management have contributed to the improved survival of children undergoing repair of congenital heart disease. Despite this, peri-operative myocardial injury and ventricular dysfunction continue to be major contributors to morbidity and mortality.

Tissue Doppler imaging (TDI), an echocardiography based technique, measures Doppler shift frequencies produced by the contraction and relaxation of longitudinal muscle fibres that run from the plane of atrio-ventricular annuli to the heart apex [1]. The resulting Doppler spectrum and measured myocardial velocities have allowed quantification of systolic and diastolic function in many clinical and experimental studies [2]. More recently myocardial acceleration and peak velocity measured during the period of isovolumetric contraction have been proposed and validated as systolic contractile indices independent of changing loading conditions [3].

In children undergoing congenital heart surgery TDI may offer many potential advantages over conventional echo-

cardiography and other ventricular imaging modalities. Of particular interest is the ability to quantify right ventricular (RV) function independent of its complex, non-geometric shape [4]. Published data on normal TDI parameter values in children with normal anatomical hearts are available [5]. To date, however, there is little information on the pattern of change in TDI parameters that follows cardiac surgery in children.

The objective of this study was to investigate the variations in systolic TDI parameters of myocardial velocity and acceleration that occur before and after congenital heart surgery and explore the association between TDI change, myocardial injury and clinical outcome.

## 2. Methods

The study was approved by the Local Ethical Committee. Informed consent was obtained prior to study enrolment. In total, 35 subjects with a range of septation defects and 10 control subjects with structurally normal hearts were studied (Table 1).

Thirty patients underwent surgical repair. TDI was performed using a Vivid 7 ultrasound scanner (GE Vingmed, Norway) with a 7-MHz probe. Measurements were obtained: (1) preoperatively, under general anaesthesia (PreOp); (2) 1 h postoperatively in paediatric intensive care unit (PICU);

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Table 1  
Demographic data

Demographics	Control	ASD	VSD	AVSD
<i>n</i>	10	8 <sup>a</sup>	16	11
Male	4	3	11	5
Age (months)	23.6 ± 20.9 <sup>a</sup>	37.8 ± 15.2 <sup>a</sup>	8.0 ± 5.5	6.1 ± 6.0
Weight (kg)	13.2 ± 2.6 <sup>a</sup>	13.2 ± 2.6 <sup>a</sup>	6.4 ± 2.5	5.1 ± 0.8
XC (min)	44.0 ± 30.1	44.0 ± 30.1	50.1 ± 15.9	108.8 ± 19.8 <sup>***</sup>
CPB (min)	73.8 ± 38.3	73.8 ± 38.3	83.56 ± 20.9	155.2 ± 24.9 <sup>**</sup>
Ventilation time (h)	18.3 ± 21.9	18.3 ± 21.9	61.1 ± 57.5	107.6 ± 57.4 <sup>***</sup>
PICU stay (days)	1.38 ± 0.7	1.38 ± 0.7	3.89 ± 2.7	6.00 ± 2.6 <sup>***</sup>
cTnI (day-1, µg/l)	3.82 ± 3.7	3.82 ± 3.7	3.83 ± 2.2	12.9 ± 4.26 <sup>***</sup>

Data expressed as mean ± S.D.

Control, structurally normal heart; ASD, atrial septal defect; VSD, ventricular septal defect; AVSD, atrio-ventricular septal defect; XC, cross-clamp; CPB, cardiopulmonary bypass; PICU, paediatric intensive care unit; cTnI, cardiac Troponin-I.

<sup>a</sup>5 primum, 3 secundum; <sup>\*</sup>control and ASD groups were significantly older (ASD; and heavier) than VSD and AVSD groups ( $P < 0.01$ ); <sup>\*\*</sup>cross-clamp and cardiopulmonary bypass duration were significantly longer for AVSD group compared to VSD and ASD groups ( $P < 0.0001$ ); <sup>\*\*\*</sup>ventilation time, PICU stay and cTnI was significantly higher in AVSD group compared to VSD and ASD groups ( $P < 0.001$ ).

and (3) 15 h postoperatively (day 1). Colour-coded myocardial velocities were recorded from the basal septum and the lateral free walls of both ventricles immediately below (0.5 cm) the insertion of the mural leaflet of the left and right AV valves as previously described [6]. Three consecutive cardiac cycles were recorded and stored digitally for later off-line analysis.

An arterial blood sample for cardiac Troponin-I (cTnI) was taken 15 h postoperatively.

### 2.1. TDI analysis

Stored TDI data were displayed (Fig. 1) and analysed using Echopac software (GE Vingmed). Each TDI parameter was derived by analysis of the TDI annular signal and represented the functional description of corresponding ventricle i.e. tricuspid valve for RV, mitral valve for left ventricle (LV) and basal septum for septum. Peak myocardial velocity (Sa) was measured as the wave height during systole. Peak myocardial isovolumetric velocity (IVV) was defined as the maximum velocity during isovolumetric contraction. Isovolumetric acceleration (IVA) was calculated by dividing the peak IVV by the isovolumetric contraction time (Fig. 1) [3].

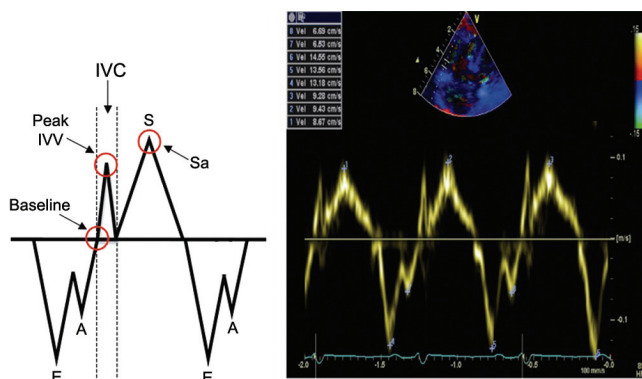


Fig. 1. Normal TDI waveform depicting measured and acquired parameters. E, early diastolic wave; A, late diastolic wave; S, systolic wave; Sa, absolute peak systolic velocity; Baseline, onset of isovolumetric contraction; Peak IVV, absolute peak isovolumetric velocity; IVC, period of isovolumetric contraction.

### 2.2. Statistical analysis

Data are expressed as means ± S.D. Demographic and outcome data (age, bypass and cross-clamp times, cTnI, ventilation time, and PICU stay), TDI comparisons with controls and the change in TDI parameters with time were all analysed by ANOVA with post-hoc Bonferroni comparison. Correlations were performed between quantitative variables to assess the degree of linear association using Pearson method. All analyses were done using Minitab (Version 14) with a significance level of 5%.

## 3. Results

There was no mortality or significant complication. Study and control patient demographics are summarised in Table 1. There was an increase in operative duration, troponin release and ventilation/PICU times with increasing cardiac complexity.

Postoperative Troponin-I was elevated compared to the normal range. cTnI (all patients; 6.6 ± 5.3) was significantly higher following atrio-ventricular septal defect (AVSD) repair compared with ventricular septal defect (VSD) and atrial septal defect (ASD) (Table 1). There was a strong correlation between cTnI release and cross-clamp ( $r = 0.82$ ,  $P < 0.0001$ ) and bypass duration ( $r = 0.83$ ,  $P < 0.0001$ ).

### 3.1. TDI parameters

Preoperatively in both control and study patients, all TDI parameters were greater in the RV compared with LV and septum, with the highest values in the control group (Table 2). Peak myocardial velocities demonstrated an obvious decrease with increasing complexity of cardiac lesion. By contrast, the isovolumetric parameters, particularly in the septum, remained more consistent across the cardiac lesion spectrum.

LV, RV and septal peak systolic velocities were all significantly reduced in the immediate postoperative period but LV velocities demonstrated a trend to recovery by day 1, (Fig. 2). By contrast, both isovolumetric parameters, IVA (Fig. 3) and IVV (Fig. 4), decreased in the early period and continued to decline at day 1.

Table 2  
Controls vs. preoperative TDI parameters by diagnosis

	Sa (cm/s)			IVV (cm/s)			IVA (cm/s <sup>2</sup> )		
	LV	Sep	RV	LV	Sep	RV	LV	Sep	RV
Control	7.75±1.70	7.43±0.46	12.65±0.99	6.20±1.56	7.03±0.74	11.86±4.93	300.8±62.4	291.8±110.8	470.8±189.3
ASD	6.63±1.47	5.21±0.68**	8.64±1.89**	6.37±1.20	7.61±0.57	9.80±0.83	230.2±153.9	216.4±44.5	263.0±93.0*
VSD	5.46±1.3**	4.92±0.98**	7.73±2.29**	6.86±3.97	7.32±2.45	9.04±3.68	220.9±132.9	233.6±113.7	274.5±116.4*
AVSD	4.19±0.9**	4.52±1.41**	6.93±2.32**	6.17±2.35	6.94±3.00	9.58±2.27	142.2±88.4*	218.6±79.6	317.6±111.6*

TDI data expressed as mean±S.D.; (TDI velocity vs. control: \* $P < 0.05$ , \*\* $P < 0.0001$ ).

Sa, peak systolic myocardial velocity; IVV, peak velocity during isovolumetric contraction; IVA, isovolumetric acceleration; LV, left ventricle; Sep, basal septum; RV, right ventricle; Control, anatomically normal heart; ASD, atrial septal defect; VSD, ventricular septal defect; AVSD, atrio-ventricular septal defect.

### 3.2. Correlation of TDI parameters with myocardial injury and outcome

Reduced preoperative Sa<sub>LV</sub> correlated with greater cTnl and longer ventilation ( $r = 0.54$ ,  $P = 0.002$ ) and PICU times ( $r = 0.53$ ,  $P = 0.02$ ).

Postoperatively, peak systolic velocities in septum correlated with X-clamp ( $r = -0.51$ ,  $P = 0.003$ ), cardiopulmonary bypass (CPB) ( $r = -0.53$ ,  $P = 0.003$ ), Tnl ( $r = -0.64$ ,  $P = 0.001$ ), ventilation ( $r = -0.50$ ,  $P = 0.007$ , Fig. 6a) and PICU stay ( $r = -0.55$ ,  $P = 0.002$ ). RV velocities correlated X-clamp time ( $r = -0.43$ ,  $P = 0.022$ ), CPB ( $r = -0.42$ ,  $P = 0.026$ ), and Tnl ( $r = -0.42$ ,  $P = 0.028$ , Fig. 5). LV velocities correlat-

ed only with ventilation ( $r = -0.42$ ,  $P = 0.025$ , Fig. 6b) and PICU stay ( $r = -0.48$ ,  $P = 0.01$ ).

Postoperative IVV in septum correlate with X-clamp ( $r = -0.44$ ,  $P = 0.028$ ), CPB ( $r = -0.44$ ,  $P = 0.026$ ), Tnl release ( $r = -0.71$ ,  $P = 0.001$ ), ventilation ( $r = -0.47$ ,  $P = 0.024$ ), and PICU stay ( $r = -0.61$ ,  $P = 0.002$ ). RV IVV correlated with X-clamp ( $r = -0.43$ ,  $P = 0.042$ ), CPB ( $r = -0.53$ ,  $P = 0.009$ ), Tnl ( $r = -0.49$ ,  $P = 0.021$ ) and PICU stay ( $r = -0.42$ ,  $P = 0.046$ ), whilst LV IVV correlated only with CPB ( $r = -0.49$ ,  $P = 0.021$ ) and PICU stay ( $r = -0.51$ ,  $P = 0.015$ ).

Postoperative IVA in septum correlated with Tnl ( $r = -0.62$ ,  $P = 0.002$ ), ventilation ( $r = -0.41$ ,  $P = 0.054$ ), PICU stay ( $r = -0.47$ ,  $P = 0.024$ ). RV IVA did not demonstrate correlation with either myocardial injury or outcome and LV IVA cor-

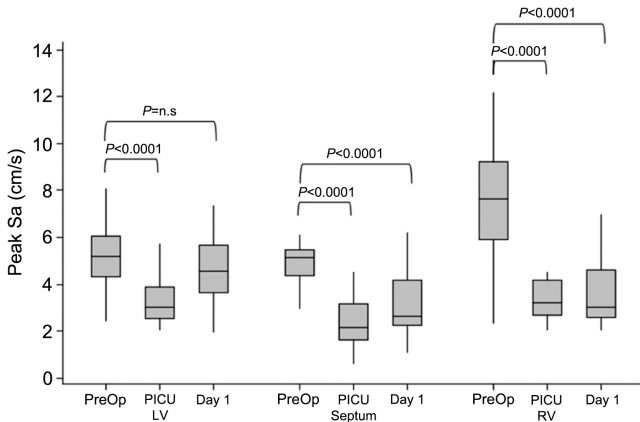


Fig. 2. Change in peak systolic myocardial velocity.

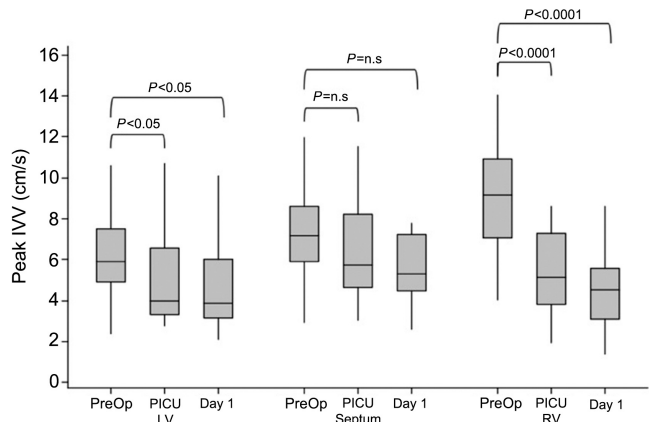


Fig. 4. Change in isovolumetric velocity.

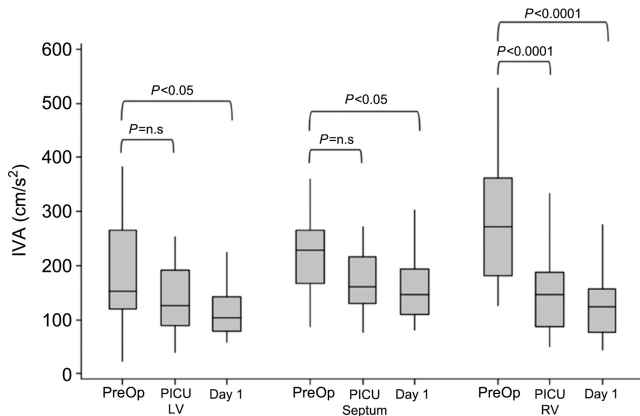


Fig. 3. Change in isovolumetric acceleration.

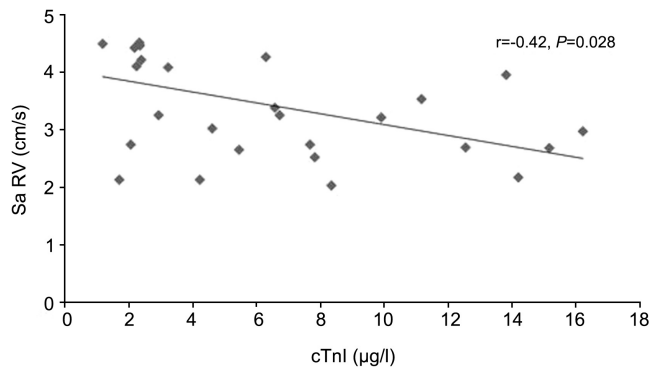


Fig. 5. RV systolic velocity (PICU) vs. cTnl.

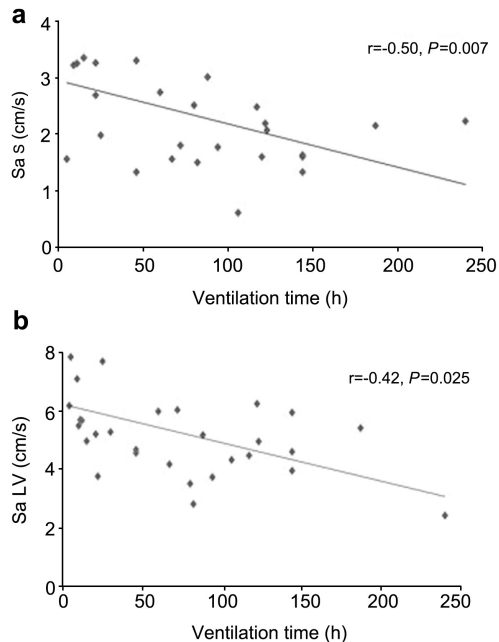


Fig. 6. (a) Septal systolic velocity (PICU) vs. ventilation time. (b) LV systolic velocity (PICU) vs. ventilation time.

related with CPB ( $r = -0.45$ ,  $P = 0.032$ ) and X-clamp ( $r = -0.49$ ,  $P = 0.019$ ).

#### 4. Discussion

The principal findings in this study were: (1) In patients with congenital heart lesions preoperative bi-ventricular systolic tissue Doppler parameters were reduced compared to normal controls; (2) Following cardiac surgery there was a reduction in TDI velocities and isovolumetric indices; (3) Reduced postoperative RV velocities were associated with a greater extent of myocardial injury as defined by Troponin-I release; (4) Reduced pre- and postoperative LV velocities were associated with longer ventilation and PICU times.

The assessment of ventricular function following paediatric cardiac surgery is complicated by the alteration to loading conditions caused by the elimination of intra-cardiac shunting and variation in postoperative pulmonary and systemic vascular resistances.

In this study contractility was quantified using absolute (peak systolic and IVV) and derived (IVA) tissue Doppler indices. Peak systolic myocardial velocities have been validated as an index of contractility in animal experiments of ischemia-reperfusion [7] and in studies of inotrope modulation, where velocities have correlated closely with fractional shortening measured by sonomicrometry and maximum elastance from pressure-volume data [2]. In clinical studies, systolic TDI velocities have proved to be reliable indicators of LV and RV function as well as predicting survival in patients with heterogeneous cardiac disease [8]. However, the magnitude of systolic myocardial velocities has been shown to be preload and afterload dependent [3, 9]. Recently, peak IVV and acceleration have been proposed and validated through animal studies as load independent contractile indices within the physiological

range expected postoperatively [3, 10]. The potential ability of TDI to assess ventricular function independent of load is particularly important when considering the RV which, in the context of congenital heart disease, is frequently characterised by pressure or volume loading [10].

From the preoperative data it was found that, not only were the patients with cardiac lesions observed to have reduced velocities compared to controls, the extent of velocity reduction was greater with increasing cardiac complexity. By contrast, the variation observed in isovolumetric indices, particularly IVV, was much less across the disease spectrum consistent with the load-independent character of these parameters. Of interest, IVA appeared to be most reduced in the specific ventricle likely to be volume loaded by the cardiac lesion, i.e. RV with ASD and LV with VSD/AVSD. The study also identified that reduced preoperative LV TDI indices were associated with longer ventilation/PICU times, confirming a clinical impression that patients in preoperative heart failure with VSD lesions, with large Qp:Qs and volume loaded LV, are likely to require longer postoperative recovery. Previous adult studies have also shown LV systolic velocities may predict survival in patients with congestive heart failure [11]. Ultimately it may be that LV systolic TDI parameters will have a predictive value in determining postoperative course and can be factored into optimising the timing of surgery.

In the current study, the finding of a reduction in all systolic TDI parameters measured at the septum, mitral and tricuspid annuli suggests an overall global impairment of contractility in the early postoperative period. Of particular interest was the disparity seen between RV and LV TDI parameters postoperatively. LV peak systolic velocities recovered towards baseline preoperative values by 15 h postoperatively and this pattern of change may reflect the nadir in cardiac index described by Wernovsky [12]. However, peak systolic velocities are an ejection-phase index and therefore, load-dependent and the initial fall in LV velocities may in part result from diminished LV pre-load following VSD closure [9]. By contrast, RV and septal systolic velocities remained reduced with no significant recovery between the two postoperative time points. Potentially these velocities could also be reduced as a consequence of increased RV afterload by elevations in pulmonary arterial pressure common in postoperative patients with repaired VSD or canal defects. The greater and more significant reduction in RV isovolumic acceleration seen postoperatively supports the view that an intrinsic RV contractile impairment existed. Ultimately, however, there exists a complex interplay of changes in loading conditions and myocardial contractility that are often interrelated and therefore, not possible to definitively isolate.

Troponin-I has been shown to correlate well with the extent of peri-operative myocardial damage and is predictive of postoperative outcome in the paediatric population [13]. In this patient group although direct retraction trauma cannot be avoided, no ventricular incisions or myocardial resection were performed and therefore, myocardial injury and cTnI release should relate to the duration of ischemia-reperfusion. This was strongly evident in our study with clear correlations between cross-clamp and bypass times and cTnI levels. The significant correlations between cTnI

levels and RV and septal TDI parameters would implicate myocardial injury as the principal mechanism behind the reduced contractility. Of interest, although both ventricles were exposed to the same ischemic-reperfusion insult, no significant correlations between Troponin-I and postoperative LV TDI parameters were found. This may reflect a vulnerability of the RV to myocardial injury in this patient group. The RV is vulnerable to direct surgical trauma and contusion resulting from retraction and VSD suture placement. Postoperative conduction abnormalities, including right bundle branch block may have a more pronounced effect on RV contraction although no conduction defects were seen in these patients.

In our acute postoperative setting, TDI had a predictive value for early outcome with reduced LV and septal TDI significantly correlating with longer ventilation time and PICU stay. By contrast RV TDI, the ventricle with the most marked postoperative TDI reduction, did not demonstrate a correlation with outcome.

#### 4.1. Clinical implications and future considerations

Preoperative TDI quantification of LV systolic function may prove to be a useful adjunct in determining the timing of corrective surgery. TDI also has the ability to assess contractile dysfunction in the PICU setting, allowing early and ongoing assessment of ventricular performance following cardiac surgery. This may enable appropriate therapeutic intervention prior to the onset of the systemic manifestations of low cardiac output. Due to its particular sensitivity for RV contraction, TDI may provide better evaluation of RV–pulmonary interaction post cardiac surgery.

#### 4.2. Study limitations

We did not analyse TDI beyond the first postoperative day and it would have been interesting to know if the RV and septal parameters returned to normal during the early postoperative period. In a previous study of postoperative Fallot's patients a reduction in RV myocardial acceleration persisted late after repair [10]. However, this may relate to ongoing haemodynamic lesions including chronic pulmonary regurgitation rather than a permanent myocardial injury sustained at the time of surgical repair.

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