The handle http://hdl.handle.net/1887/22985 holds various files of this Leiden University dissertation

Author: Klitsie, Liselotte Maria
Title: Tissue Doppler and speckle tracking strain echocardiography: from evaluation in healthy children to follow-up after surgery for a congenital heart defect
Issue Date: 2014-01-09
Review: Ventricular performance after surgery for a congenital heart defect as assessed using advanced echocardiography: from Doppler flow to 3D echocardiography and speckle-tracking strain imaging

Pediatr Cardiol 2013

Liselotte M. Klitsie
Arno A.W. Roest
Nico A. Blom
Arend D.J. ten Harkel
ABSTRACT
A varying degree of impairment of ventricular performance is observed over the long-term after surgery for a congenital heart defect (CHD). Impaired ventricular performance has been shown to be of prognostic value for increased risk of cardiovascular events in adult CHD patients. This emphasizes the importance of delineating the timing and cause of this postoperative impairment.

Impairment of ventricular performance could develop over time as a consequence of residua, sequelae and complications of the CHD or surgical procedure. Yet, impaired ventricular performance has also been observed immediately after surgery and can persist and/or worsen over time. This postoperative impairment of ventricular performance is the focus of this review.

This article provides an overview of echocardiographic techniques currently used to assess ventricular performance. Furthermore, we review current literature describing ventricular performance, as assessed using echocardiography, after correction of a CHD.

In general, a decrease in ventricular performance is observed directly after surgery for CHDs. Subsequent follow-up of ventricular performance is characterized by a varying degree of postoperative recovery. A consistent observation is the persistent impairment of right ventricular performance after repair in several different subgroups of CHD patients, ranging from ventricular septal defect repair to surgery for Tetralogy of Fallot.
INTRODUCTION

Many patients with a severe congenital heart defect (CHD) will not survive the first year of life without surgical intervention. Since the introduction of pediatric cardiac surgery during the early 1940s, survival started to increase with currently a survival rate into adulthood of 90% of patients born with a CHD.\(^1\) Although long-term survival seems promising, long-term follow-up of corrected CHD patients is still characterized by a varying degree of impairment of ventricular performance.\(^2,3\) This impairment may be the consequence of residua, sequelae, and complications, including persistent left-to-right or right-to-left shunting, stenosis, or insufficiency of heart valves and decreasing cardiac function over time.\(^4\) In addition, preoperative and perioperative factors may contribute to this impairment of ventricular performance after cardiac surgery.\(^5\)

Impaired ventricular performance has been shown to be of prognostic value for increased risk of cardiovascular events in adult CHD patients.\(^6\) Accordingly, to optimize therapeutic strategies it is important to delineate the timing and cause of this postoperative impairment of ventricular performance.

Currently several techniques can be used to assess ventricular performance after CHD surgery, including echocardiography and magnetic resonance imaging (MRI). MRI has an excellent spatial and temporal resolution, good reproducibility and enables assessment of cardiac dimensions without geometric assumptions. These characteristics allow accurate follow-up of ventricular performance, even in CHD patients with complex anatomy.\(^7,8\)

Echocardiographic techniques also provide a comprehensive and reproducible insight in both global and regional ventricular performance in CHD patients. An advantage of echocardiography compared with MRI in postoperative follow-up of ventricular performance is that echocardiography is almost always readily available. Furthermore, the relatively low cost and the absence of need for sedation in young children are advantages.\(^7,9\) Accordingly, particularly during the direct postoperative period, echocardiography is better equipped than MRI for assessment of changes in ventricular performance.

In the present review, we will focus on the echocardiographic assessment of ventricular performance after CHD correction. We will provide an overview of echocardiographic techniques currently used to assess ventricular performance. Furthermore, we will describe preoperative, perioperative and postoperative factors that may add to impairment of ventricular performance after CHD correction. Finally, we will review the current literature describing ventricular performance, as assessed using echocardiography, after correction of various types of CHDs.
ECHOCARDIOGRAPHIC TECHNIQUES

Conventional echocardiography

Left-ventricular (LV) fractional shortening (FS) and ejection fraction (EF), as assessed with M-mode and two-dimensional echocardiography, respectively, are valuable tools to describe LV systolic performance. Both FS and EF have shown to be easily applicable in clinical practice in both adults and children to assess LV performance.\(^\text{10}\) Furthermore, LV FS is reproducible and has a high temporal resolution, which strengthens its use.\(^\text{11}\) Yet, the geometric assumptions on which LV volume calculations are based can pose a problem in CHD patients, in whom geometry is often anomalous.

In the right ventricle two-dimensional echocardiographic models to assess right-ventricular (RV) performance have been less successful. The complex shape of the right ventricle has prevented development of an appropriate echocardiographic model to assess RV EF. RV fractional area change (FAC) has been proven useful in adults to assess RV systolic performance. However, in children a weak correlation between MRI derived RV systolic performance variables and RV FAC has been observed, thus discouraging its use.\(^\text{12}\) An RV dimensional variable that has been proven useful as a simple and reproducible bedside tool in children is tricuspid annular plane systolic excursion (TAPSE).\(^\text{13}\) A disadvantage of TAPSE is its one-dimensional nature and its inability to detect regional alterations. In patients after correction of Tetralogy of Fallot (ToF), the inability of TAPSE to detect regional alterations was suggested to be the cause of the lack of correlation between MRI-derived RV EF and TAPSE.\(^\text{14}\)

Spectral Doppler inflow velocities across the mitral and tricuspid annulus, including peak early filling (E) and late diastolic filling (A) velocity, have been extensively used to describe biventricular diastolic performance.\(^\text{15}\) However the age, heart rate, and load-dependence of these techniques could render them suboptimal in evaluation of ventricular performance in pediatric CHD patients.\(^\text{15}\) Furthermore, observations of pseudonormalization of the mitral and tricuspid inflow pattern in some patients with abnormal relaxation complicates their interpretation.\(^\text{16}\)

Tissue Doppler imaging

Pulsed wave and colour tissue Doppler imaging (TDI) use the low-velocity, high-amplitude Doppler signals of myocardial tissue to assess the velocity of myocardial motion. This allows evaluation of both peak systolic (S’) and peak early (E’) and late (A’) diastolic velocities of the left and right ventricles.\(^\text{17}\) In children with a complex CHD, the relative geometry independence of TDI measurements is an important advantage compared with the previously described LV EF.\(^\text{18}\) Furthermore, more detailed evaluation of ventricular performance is possible due to its ability to evaluate regional performance.
Table 1. Characteristics of echocardiographic parameters

<table>
<thead>
<tr>
<th>Variable</th>
<th>References</th>
<th>Global performance</th>
<th>Regional performance</th>
<th>Systolic performance</th>
<th>Diastolic performance</th>
<th>Growth dependence</th>
<th>Heart rate dependence</th>
<th>Loading dependence</th>
<th>Geometric assumptions</th>
<th>Reproducibility</th>
</tr>
</thead>
<tbody>
<tr>
<td>TAPSE</td>
<td>14,115,116</td>
<td>+/-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Doppler flow</td>
<td>15</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>TDI Velocities</td>
<td>18,22,23,11,18</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+/-</td>
<td>+/-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>IVA</td>
<td>27,30,31</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+/-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>MPI</td>
<td>33,119</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+/-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Speckle-tracking Strain</td>
<td>37,120-122</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+/-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>SR</td>
<td>37,120,121</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+/-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>3DE EF</td>
<td>41,123</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
</tbody>
</table>

3DE, 3-dimensional echocardiography; EF, Ejection fraction; IVA, isovolumetric myocardial acceleration; MPI, myocardial performance index; SR, strain rate; TAPSE, tricuspid annular systolic excursion; TDI, tissue Doppler imaging.
A disadvantage of TDI is that no distinction can be made between active and passive motion.\textsuperscript{17} Furthermore, as with all Doppler-derived techniques, assessment of cardiac motion is only possible along the direction of the Doppler beam. This angle dependence allows only full segmental analysis in the longitudinal direction. For example, in patients with a univentricular heart, this is disadvantageous, because the decrease in longitudinal motion is suggested to be (partly) compensated by an increase in circumferential shortening.\textsuperscript{19} Omission of this circumferential compensation in TDI analysis could explain the lack of correlation between MRI-derived EF and TDI-derived velocities in patients with a univentricular heart.\textsuperscript{19,20} In addition, TDI measurements are often performed in basal segments, which may not always be representative for global ventricular performance. For example, in postoperative ToF patients, RV outflow tract (RVOT) dysfunction was not detected by basal TDI velocity measurements.\textsuperscript{21} Finally, the most important consideration when using TDI in CHD patients is that conflicting results exist concerning the effect of alterations in loading conditions on both systolic and diastolic TDI variables.\textsuperscript{22-24}\textsuperscript{23} Vignon et al.\textsuperscript{23} described systolic and diastolic TDI variables to be preload independent, whereas Drighil et al.\textsuperscript{22} reported preload dependence of these variables. Moreover, changes in TDI measurements after acute preload reduction, due to transcatheter atrial septal defect (ASD) closure, have been variable.\textsuperscript{24,25} Notwithstanding these considerations, TDI allows the detection of more subtle changes in ventricular performance compared with LV EF and LV FS.\textsuperscript{26} (Table 1)

To overcome the issue of load dependency several other TDI-based variables have been introduced, including isovolumetric myocardial acceleration (IVA) and the myocardial performance index (MPI). IVA is measured as the slope of the presystolic velocity curve (cm/s\textsuperscript{2}) and is minimally preload and afterload dependent. In animal studies, IVA was shown to be a good noninvasive index of contractility.\textsuperscript{27} Subsequent evaluation of IVA in clinical studies, including pediatric ToF patients, further supported its use by describing a correlation between IVA and RV invasive measurements.\textsuperscript{28} However, previously presented normal values of IVA in both adult and pediatric healthy subjects were highly variable\textsuperscript{29} and were shown to be heart rate dependent.\textsuperscript{30} Furthermore, low measurement reproducibility was shown.\textsuperscript{31} These observations limit the use of IVA as a clinical useful tool to assess systolic LV and RV performance.

Studies describing TDI-derived MPI, a ratio of the total time spent in isovolumic contraction and relaxation divided by the ejection time, have been more promising compared with IVA. MPI has been shown to be of predictive value in the detection of heart failure.\textsuperscript{32} In children, MPI has been shown to be reproducible\textsuperscript{33} and both LV and RV MPI were not clinically significantly correlated to age or heart rate.\textsuperscript{33} These characteristics facilitate the use of MPI in pediatric patients. A disadvantage of MPI is that the combination of systolic and diastolic performance in one variable renders the variable less specific. Furthermore, in ToF patients with significant pulmonary regurgitation (PR), isovolumic relaxation could be absent, thus complicating the
interpretation of MPI. This may explain the lack of correlation between RVEF and RV MPI in ToF patients. Hence, the value of MPI in ToF patients is disputable.

Finally, the calculation of strain rate (SR) and strain from TDI-derived velocity curves has further expanded the use of TDI to define ventricular performance. Strain, defined as the deformation of an object normalized to its original shape, can distinguish active from passive contraction. This is an important advantage of strain variables compared with previously described TDI variables. Yet, the TDI-derived systolic and diastolic strain and SR measurements are still angle dependent. Furthermore, the influence of small artefacts and drop-out is magnified in the calculation of strain and SR from TDI velocity curves.

**Speckle-tracking strain imaging**

Speckle-tracking strain imaging is an alternative technique to assess strain and SR. Using an automated tracking algorithm this technique assesses the frame-to-frame movement of so-called speckles (natural acoustic markers within the myocardium). The change in position of speckles relative to their original position is used to calculate strain. (Figure 1) The technique is angle independent and less influenced by drop-out than TDI derived strain measurements. Furthermore, speckle-tracking strain imaging allows multidirectional detailed assessment of LV and RV mechanics. For the LV this includes evaluation of longitudinal, radial and circumferential wall motion. Finally speckle-tracking strain imaging also allows for the evaluation of rotational motion, including twist and torsion, which adds an additional perspective.

Disadvantages of speckle-tracking strain imaging include the necessity of high-quality images to perform speckle-tracking strain imaging. In addition, the observed vendor dependence complicates its interpretation, and the technique may be considered time-consuming. Last, one of its present limitations is that the optimal frame rate is unclear, due to a trade-off between temporal and spatial resolution. Especially considering the high heart rates in children, too low frame rates will result in undersampling and less reliable results.

Despite these disadvantages, speckle-tracking strain imaging is a promising technique to evaluate biventricular performance. In both healthy adults and pediatric patients, reference values of speckle-tracking strain derived variables have been published. A relatively good feasibility and reproducibility of strain was described in these studies. In CHD patients, a good correlation was described between MRI-derived measurements and speckle-tracking strain imaging. Furthermore, the added value of multidirectional analysis in CHD patients was underlined in a study in patients with a univentricular heart. Finally, rotational mechanics have shown to be decreased in ToF patients compared with controls. Despite these encouraging results, future studies in CHD patients are necessary to define characteristics of speckle-tracking strain variables in CHD patients, including its prognostic value.
Panel A. The top left corner shows a grayscale harmonic image of the apical four-chamber view. The region of interest is set and divided into six segments. Time-strain curves of each segment (colored lines) and global time strain curves (dotted line) are shown on the right-hand side. Arrows indicate peak strain.

Panel B and C. The top left corner shows a grayscale harmonic image of the short-axis view. The region of interest is divided into six segments, and the corresponding time-strain curves for radial (B) and circumferential (C) strain are derived.
Three-dimensional echocardiography
A major innovation in echocardiography has been the introduction of three-dimensional echocardiography (3DE). Especially in delineation of anatomy 3DE has been shown to be of additive value compared with previously described two-dimensional echocardiographic measurements.\textsuperscript{40} Furthermore, 3DE allows accurate and reproducible analysis of LV and RV volume and EF in both adults and children.\textsuperscript{41,42} The total absence of geometrical assumptions in 3DE is an important advantage, especially in CHD patients.

Challenges currently remain in the trade-off between temporal and spatial resolution.\textsuperscript{43} In addition, the necessity of breath hold during image acquisition complicates use of multiple-beat 3DE in children.\textsuperscript{43} Yet, in very young children breath hold may be less of a problem considering their high heart rate and relatively superficial breathing pattern. Furthermore, one-beat 3DE techniques are being introduced that overcome this breath hold difficulty.\textsuperscript{44} Hence, although challenges remain, we advocate the use of 3DE in pediatric CHD patients.

In addition to evaluation of global ventricular performance, speckle-tracking strain imaging in 3DE provides interesting potential for future evaluation. Strain imaging in 3DE images takes into account the previously described longitudinal, radial and circumferential contraction in one analysis. However, a frame rate > 40 frames/second is required to accurately evaluate strain, which is not possible with present 3DE techniques.\textsuperscript{35}

PREOPERATIVE, PERIOPERATIVE AND POSTOPERATIVE FACTORS
Previously described echocardiographic techniques have been used in several studies in CHD patients and describe a similar pattern of change after cardiac surgery with cardiopulmonary bypass (CPB). This pattern is characterized by immediate impairment of ventricular performance after surgery.\textsuperscript{5,45} (Figure 2) Several preoperative, perioperative and postoperative factors may add to this.

Preoperative
Before surgery volume and pressure loading in CHD patients induce a variable degree of remodeling of the heart.\textsuperscript{46,47} Corrective surgery and unloading will often induce reversed remodeling. Still, preoperative remodeling will affect ventricular performance immediately after surgery and was suggested to remain important during more long-term follow-up.\textsuperscript{47,48}
Another preoperative factor which has been described to influence postoperative ventricular performance is the degree of preoperative hypoxia. In CHD patients with a cyanotic heart defect, chronic preoperative hypoxia often leaves the heart more vulnerable for reoxygenation injury during surgery with CPB. Furthermore, preoperative hypoxia has been suggested to induce structural and functional alterations of the heart, which could result in cardiac dysfunction later in life.

**Figure 2 Box plots depicting TDI parameters in CHD patients and controls**

Box plots depicting mean and 95% confidence interval of the mean. Plots describe follow-up of ventricular performance in 141 patients undergoing surgery for a CHD and 40 age-matched controls. E', peak early diastolic TDI velocity; RV, right ventricle; S', peak systolic TDI velocity.

**Perioperative**

Perioperative factors that have been suggested to decrease postoperative ventricular performance include the following: direct surgical trauma, pericardiotomy, CPB, inadequate myocardial protection and hemodynamic changes.

The influence of CPB on the body has been extensively studied in both children and adults. Aortic cross-clamping and associated ischemia have been suggested to result in postoperative myocardial damage as well as inflammatory cascades initiated by CPB. In patients with transposition of the great arteries (TGA) who undergo corrective surgery, a strong association was shown between poor operative survival and aortic cross-clamp time and CPB. In addition, a correlation of both cross-clamp time and CPB time with peak systolic TDI velocities was previously described in several subtypes of CHD patients.
Inadequate myocardial protection, achieved with cold cardioplegia and external cooling, can also add to impairment of especially RV performance after cardiac surgery. Previous studies used temperature probes in the myocardium to assess the efficacy of external cooling and described differences of 19 °C between the left and right ventricle. This temperature difference was suggested to be caused by the anterior position of the right ventricle. This position renders it much more at risk of external heating than the left ventricle during surgery.

Finally, surgery for a CHD is frequently associated with hemodynamic changes, including changes in preload and/or afterload conditions. According to the Frank-Starling mechanism, loading alterations influence ventricular performance. In addition, loading alterations may influence echocardiographic measurements.

Postoperative
Direct postoperative factors, including inotropes and mechanical ventilation, can also contribute to impairment of ventricular performance after CHD correction.

Inotropes are often vital in perioperative and postoperative care to maintain a steady-state condition during surgery and its direct postoperative period. Traditionally, catecholamines are used. However, despite their positive inotropic effect, which induces an increased force of myocardial contraction, catecholamines can also adversely affect ventricular performance. For example the use of high-dose epinephrine may increase systemic vascular resistance, which increases afterload and decreases LV performance. Phosphodiesterase inhibitors including Milrinone, have shown to be effective in pediatric patients and are most commonly used in the direct postoperative period.

Similar to inotropes, mechanical ventilation has been described to both positively and negatively affect postoperative ventricular performance depending on the type of ventilation and CHD. For example in Fontan and ToF patients after repair, positive-pressure ventilation may have a deleterious effect on cardiac output, whereas negative-pressure ventilation has been shown to increase cardiac output. Yet, in patients with LV dysfunction, cardiac output could increase in response to positive-pressure ventilation. Considering these differential effects, ventilation settings remain an important topic in the postoperative care of CHD patients.

FOLLOW-UP OF CHD PATIENTS
Subgroups of CHD patients described in the present review include patients with an ASD, ventricular septal defect (VSD), ToF, TGA, and univentricular heart. During more long-term follow-up, each subgroup is thought to have its own unique follow-up pattern of ventricular performance.
Atrial septal defect

The presence of an ASD results in left-to-right shunting, which often results in right atrial and RV volume overload. The size of shunting can be quantified using echocardiography by the Doppler-derived Qp:Qs ratio. Furthermore, assessment of right atrial and RV volume is often used to assess the size of shunting and consequent RV overload. Both right atrial and RV volume can be accurately quantified by 3DE by way of volume measurements. In pediatric patients, this RV overload translates to an increase in RV systolic performance variables before surgery. However, if untreated, long-term RV overload has a deleterious effect on RV performance as assessed using 3DE-derived RV EF and RV MPI. In addition to RV overload, the left-to-right shunt in ASD patients may induce a decrease in LV preload. This could be the cause of the observed decrease in LV size and LV E’ before surgery. This subtle impairment was not observed using the load-dependent Doppler mitral E and A measurements in this study.

After closure of an ASD in children, hemodynamics and ventricular volumes rapidly normalize and closure is thought to induce reverse remodeling. However, the method of closure, surgical or transcatheater, has been shown to be associated with differential postoperative follow-up.

Directly after surgical closure of an ASD using CPB, a decrease of LV and especially RV systolic and diastolic performance has been described as assessed using LV and RV S’, E’, A’ and MPI. Studies describing more long-term follow-up, using the same TDI velocities and MPI, generally report complete recovery of LV performance to normal values. Only one study, which used TDI-derived LV strain and SR measurements, described regional impairment in LV longitudinal performance still present after the first postoperative year. In contrast, RV systolic and diastolic performance, as assessed using both TDI and strain imaging, was still impaired several years after surgical ASD closure.

In contrast to surgical closure, within the first 24 hours after transcatheater ASD closure in infants and children, little or no impairment of biventricular performance was described using either TDI or strain measurements. In addition during more long-term follow-up of these patients, RV and LV performance remained preserved. Interestingly, in adults undergoing transcatheter closure, a gradual increase of especially RV performance has been described after correction as assessed with RV MPI. This can be the result of more pronounced preoperative impairment of RV performance as a result of long-term volume overload in adults.

These contrasting results of differential techniques for ASD closure in children suggest an important influence of perioperative factors, including CPB and exposure of the myocardium to varying temperatures, on RV performance.
**Ventricular septal defect**

In VSD patients, the left-to-right shunt during systole often leads to increased LV preload before surgery. Considering the load-dependence of Doppler flow velocities, the increased preload may be the cause of the high LV E and A velocities observed before surgery. This suggestion is supported by the lack of differences in LV E’ and A’ between patients and controls before surgery.

Similar to ASD patients, decreased RV performance was observed in VSD patients directly after surgery. Subsequent follow-up of these patients using RV MPI has suggested a recovery to normal values within the first postoperative month. These results contrast with results of another study in VSD patients, that described significantly lower TAPSE and RV S’ measurements in patients versus controls up to 20 months postoperatively.

Considering the preoperative LV overload in VSD patients, postoperative impairment of LV rather than RV performance might be expected. However, despite a direct postoperative decrease in LV systolic and diastolic performance, subsequent follow-up of LV performance using MPI revealed a gradual recovery to normal values within the first postoperative month. Amongst other factors, the decrease in LV performance immediately after VSD closure was suggested to be the result of the preload reduction. This induces a decrease of LV contractility according to the Frank-Starling principle. Studies describing more long-term follow-up of ventricular performance in VSD patients are limited. One study described decreased LV S’ and E’ 11 years after surgery. However, most other studies reported normal LV performance during follow-up, as assessed using LV TDI velocities and 3DE-derived LV EF.

**Transposition of the great arteries**

Before correction, the right ventricle functions as the systemic ventricle in TGA patients, whereas the left ventricle supports the pulmonary circulation. Although pulmonary pressure is high directly after birth, this pressure decreases over time. This may result in LV remodeling and the need for LV retraining before arterial switch operation (ASO) is possible. Accordingly, present management strategy is to perform ASO in the early neonatal period.

Directly after ASO, a significant decrease in LV FS, S’ and IVA was observed. This decrease in LV performance was related to the duration of aortic cross-clamping. In addition, loading alterations, direct surgical trauma, and reoxygenation injury may add to this immediate postoperative decrease in LV performance. TGA patients are often particularly vulnerable for reoxygenation injury during surgery due to preoperative hypoxia.
Studies describing subsequent follow-up of LV performance have yielded contrasting results. Colan et al.\textsuperscript{85} described normal LV FS 6 months to 10 years after surgery, whereas Hui et al.\textsuperscript{84} described decreased LV FS and EF in patients versus controls 10 years after surgery. A more recent study by Pettersen et al.\textsuperscript{87} used speckle-tracking strain imaging and TDI in addition to LV EF to describe LV performance 12 years after surgery. They reported normal LV EF, TDI, and circumferential strain measurements in patients. However, a slight impairment of regional LV longitudinal strain and LV torsion was observed in TGA patients versus controls. These results suggest speckle-tracking strain imaging to be more suitable in postoperative TGA patients to detect regional impairment of LV systolic performance.

Follow-up of RV performance in patients after ASO has been limited thus far. Yet, the introduction of speckle-tracking strain imaging and TDI have amplified the possibilities. One of the first studies to use these techniques in TGA patients reported decreased RV longitudinal strain, whereas RV circumferential strain and TDI were normal 12 years after surgery.\textsuperscript{87} The cause of this persistent impairment of RV performance is probably multifactorial and most likely includes previously
described preoperative hypoxia, CPB associated damage, and direct surgical trauma. In addition, postoperative residua and complications of ASO, including pulmonary artery obstruction and coronary artery injury, can negatively affect RV performance long after surgery. These results encourage further follow-up of RV performance using TDI and speckle-tracking strain imaging.

Tetralogy of Fallot

ToF is characterized by pulmonary stenosis, VSD, overriding of the aorta and RV hypertrophy. Clinical presentation depends mainly on the degree of pulmonary stenosis, which may induce RV pressure overload and/or clinical cyanosis. Both have been suggested to add to the impairment of RV performance observed before surgery in ToF patients. Accordingly, to limit these detrimental effects surgical correction of ToF is recommended before the age of 1 year.

Postoperative studies have mostly focused on RV performance. Of these studies, the only one that described follow-up immediately after surgery described a significant decrease in RV systolic and diastolic TDI velocities and IVA. Subsequently, no recovery to preoperative levels was observed within the first 3 months. Most other studies in corrected ToF patients were cross-sectional studies performed after the first postoperative year. In these studies, impaired RV systolic and diastolic performance was described even in asymptomatic patients versus controls. Accordingly, similar to results in ASD, VSD and TGA patients, a persistent impairment of RV performance is present in ToF patients after surgical correction.

Several factors have been suggested to add to this postoperative impairment of RV systolic and diastolic performance. Decreased RV diastolic compliance, which is recognized by restrictive RV physiology, was suggested to induce decreased RV diastolic performance directly after surgery. Yet, during more long-term follow-up, the decreased diastolic compliance is probably protective because it limits PR, and associated RV dilatation, is thought to be one of the most important causes of RV dysfunction during more long-term follow-up. Yet, decreased RV performance, as assessed by RV S’ and E’, was also observed in patients without significant PR. Accordingly CPB, ventriculotomy, inadequate myocardial protection, RVOT stenosis, and patch implantation may further add to the postoperative impairment of RV performance. Especially RVOT dysfunction was suggested to have an important role in inducing decreased RV performance.

Of note is that although the existence of RV dysfunction in postoperative ToF patients is not disputed, the best echocardiographic technique to quantify this has not yet been established. Both RVOT dysfunction and postoperative PR are factors complicating this. Accordingly, MRI currently remains the standard follow-up method.
Regarding LV performance, LV S', E' and A' remained comparable with preoperative levels within the first month after ToF surgery. Although some studies have described normal LV performance, during more long-term follow-up, impaired LV systolic and diastolic performance, as assessed using TDI and speckle-tracking strain imaging, has been observed to follow ToF repair as well. The significance of this LV impairment was highlighted by recent studies, which described LV performance variables to be of prognostic value in the assessment of increased risk of life-threatening arrhythmia and sudden cardiac death.

Impaired LV performance in postoperative ToF patients can be the result of preoperative hypoxia, myocardial fibrosis, paradoxical septal motion, and ventricular-ventricular interaction. The presence of ventricular-ventricular interaction in postoperative ToF patients is supported by the correlation between several LV and RV ventricular performance indices. This adverse ventricular-ventricular interaction has been suggested to be the result of RV enlargement.

Univentricular heart

Before surgery ventricular dysfunction is often present in patients with a univentricular heart. Volume and pressure overload, as well as preoperative cyanosis, may contribute to this impairment. Subsequent repair is often staged in three phases to finally end up with a Fontan circulation.

Echocardiographic follow-up of ventricular performance in patients who undergo single-ventricle repair is challenging due to the heterogeneity in underlying anatomy and abnormal ventricular geometry. Yet, the good correlation between MRI-derived measurements and both speckle-tracking strain imaging and 3DE-derived measurements in patients with a univentricular heart promotes the use of these techniques.

The goal of the first stage of repair is to create a balance between the systemic and pulmonary circulation if it is not already present. This can be achieved by pulmonary artery banding or introduction of a Blalock-Taussig shunt (aortic-to-pulmonary artery connection) or Sano shunt (right ventricle-to-pulmonary artery connection). Extensive research of the consequences of this first stage of repair has been performed in patients with hypoplastic left heart syndrome. In these patients, shunt introduction is often combined with construction of a neoaorta; together they constitute the Norwood procedure. After the Norwood procedure, variables describing longitudinal ventricular performance, including longitudinal strain and SR, were decreased. Khoo et al. observed no decrease in RV circumferential strain after the Norwood procedure and suggested the decrease in longitudinal performance variables to be an adaptive mechanism of the right ventricle to a left ventricle-like contraction pattern. Yet, the decrease in variables describing
Ventricular performance after the Norwood procedure has also been suggested to be the result of loading alterations, CPB, or surgery itself.\textsuperscript{104} The suggestion of shunt type influencing postoperative ventricular performance in patients with hypoplastic left heart syndrome\textsuperscript{105} was recently invalidated by the results of a large randomized controlled trial.\textsuperscript{106} No differences were observed in Doppler flow or TDI variables between patients with a Blalock-Taussig or Sano shunt up to 14 months postoperatively in this study.\textsuperscript{106}

The second stage of repair, characterized by creation of a bidirectional Glenn shunt (vena cava superior-to-pulmonary artery connection), results in a reduction of volume loading of the ventricle. Longitudinal follow-up of ventricular performance after this stage of repair has yielded contrasting results. In patients with hypoplastic left heart syndrome, a decrease in RV S’ and 3DE-derived RV EF has been described.\textsuperscript{103,107} This deterioration of RV function was suggested to be the result of chronic pressure overload, surgical intervention, and ventricular dilatation due to tricuspid regurgitation.\textsuperscript{103} In contrast, a decrease in MPI, suggesting improvement of ventricular performance, was reported in a heterogeneous group of single-ventricle patients who underwent bidirectional Glenn.\textsuperscript{108}

The final stage of Fontan repair includes construction of a vena cava inferior-to-pulmonary artery connection. Follow-up in patients shortly after this last stage portrayed no significant difference in Doppler flow-derived velocity or MPI before surgery versus after surgery.\textsuperscript{109,110} Subsequent cross-sectional studies of Fontan patients, ranging from 2 weeks to 18 years after Fontan, have been performed using a multitude of echocardiographic techniques and reported impaired ventricular systolic and diastolic performance in patients versus controls.\textsuperscript{111-113} This appeared to be even worse in patients with a single right ventricle compared with a single left ventricle.\textsuperscript{113} The observed impairment of diastolic ventricular performance was suggested to be the result of decreased compliance of the ventricle. Decreased compliance may be induced by the preoperative volume load and associated hypertrophy, myocardial fibrosis, or decreased filling of the ventricle after Fontan repair.\textsuperscript{111} Decreased strain and SR after Fontan repair were attributed to absence of ventricular-ventricular interaction, pre-Fontan chronic pressure and volume overload, abnormal myofiber orientation, uncoordinated ventricular contraction and relaxation, and increased afterload.\textsuperscript{112} Finally, chronic preoperative hypoxia, CPB, and postoperative residua and complications have been suggested to add to the persistent impairment of ventricular performance in Fontan patients.\textsuperscript{114}
CONCLUSIONS AND FUTURE PERSPECTIVES

In recent years, a multitude of echocardiographic techniques have been introduced that have brought about an expansion of knowledge of ventricular performance after surgery for a CHD. Due to influences of preoperative, perioperative and postoperative factors, a decrease in ventricular performance is often observed directly after surgery. Subsequent follow-up of ventricular performance is characterized by a varying degree of postoperative recovery. A remarkable observation is the persistent impairment of RV performance after repair in several different subgroups of CHD patients, ranging from VSD repair to surgery for ToF.

Still, challenges remain in the echocardiographic follow-up of CHD patients after surgery. Further validation studies and reproducibility analyses of TDI and speckle-tracking strain imaging as well as 3DE in CHD patients are necessary. In addition long-term longitudinal follow-up studies, starting shortly after surgery, are desirable to discover the cause of the observed persistent impairment in ventricular performance. Finally, correlation between ventricular performance and clinical follow-up is essential to put changes in echocardiographic variables into clinical perspective.
REFERENCES


61. with a low to negative pressure ventilation after tetralogy of fallot repair: a hemodynamic tool for patients
Shekerdemian LS, Bush A, Shore DF, Lincoln C, Redington AN. Cardiorespiratory responses
Pediatr Cardiol
Latifi S, Lidsky K, Blumer JL. Pharmacology of inotropic agents in infants and children. Prog
disease. Circulation

56. Bryant RM, Shirley RL, Ott DA, Feltes TF. Left ventricular performance following the arterial
switch operation: use of noninvasive wall stress analysis in the postoperative period. Crit

55. Hoffman TM, Wernovsky G, Atz AM et al. Efficacy and safety of milrinone in preventing low
switch operation: use of noninvasive wall stress analysis in the postoperative period. Crit

54. Asimakopoulos G. Systemic inflammation and cardiac surgery: an update. Perfusion

53. Kozik DJ, Tweddell JS. Characterizing the inflammatory response to cardiopulmonary bypass

52. Prifti E, Crican A, Bonacchi M et al. Early and long term outcome of the arterial switch
operation for transposition of the great arteries: predictors and functional evaluation. Eur J

51. Vassalos A, Lilley S, Young D et al. Tissue Doppler imaging following paediatric cardiac
surgery: early patterns of change and relationship to outcome. Interact Cardiovasc Thorac

50. Fisk RL, Ghaswalla D, Guilbeau EJ. Asymmetrical myocardial hypothermia during

49. Bryant RM, Shirley RL, Ott DA, Feltes TF. Left ventricular performance following the arterial
switch operation: use of noninvasive wall stress analysis in the postoperative period. Crit

cardiac output syndrome in infants and children after corrective surgery for congenital heart

47. Latifi S, Lisicky K, Blumer JL. Pharmacology of inotropic agents in infants and children. Prog

46. Shekerdemian LS, Bush A, Shore DF, Lincoln C, Redington AN. Cardiorespiratory responses
to negative pressure ventilation after tetralogy of fallot repair: a hemodynamic tool for patients
96. Davlouros PA, Kilner PJ, Hornung TS et al. Right ventricular function in adults with repaired tetralogy of Fallot assessed with cardiovascular magnetic resonance imaging: detrimental role of right ventricular outflow aneurysms or akinesia and adverse right-to-left ventricular interaction. J Am Coll Cardiol 2002;40:2044-2052.


114. Rossano JW, Chang AC. Perioperative management of patients with poorly functioning ventricles in the setting of the functionally univentricular heart. Cardiol Young 2006;16 Suppl 1:47-54.


