# CHANGES IN REGIONAL WALL STRESS IMMEDIATELY FOLLOWING VENTRICULAR SEPTAL DEFECT CLOSURE

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# INTRODUCTION

We recently reported that after surgical correction for ventricular septal defect (VSD) depression of left ventricular (LV) short-axis fractional shortening (FS) was localized to the mid-septal and midlateral walls [1]. We reasoned that regional changes in LV FS after VSD closure were due to regional changes in short-axis cross-sectional shape and wall motion, due to changes in some combination of LV preload, afterload and contractility. Until now, we have considered preload and afterload to have global effects on FS and assumed that the regionally heterogeneous change in FS reflected a regional change in contractility due to localized myocardial damage associated with the surgery itself. However, with changes in LV geometry, wall thickness and pressure after VSD closure, there could have been local changes in myocardial wall stress, imposing regional variations in preload and afterload on the myocardium. To test whether changes in myocardial loading may have been involved in causing the regionally heterogeneous change in FS after VSD closure, we examined estimates of local wall stress in the LV short-axis immediately before and after surgical VSD repair.

### **METHODS**

### Intraoperative Data Acquisition

Pediatric patients undergoing surgical correction of VSD by Gore-Tex patch closure were studied using two-dimensional transesophageal echocardiography (TEE). Intraoperative TEE shortaxis cross-sections at the level of the papillary muscles were obtained immediately before and after cardiopulmonary bypass (CPB). LV pressure (LVP) was measured using a micromanometer inserted into the LV.

## **Data Analysis**

Three cardiac cycles from before and after defect closure were chosen for each patient. For each beat, end-diastole (ED) was chosen as the frame with the largest LV cross-sectional area and end-systole (ES) as the frame with the smallest. These frames were then digitized for analysis on a personal computer. Using routines developed in Matlab, ED area (EDA) and ES area (ESA) were measured by manual planimetry of the endocardial border, excluding the papillary muscles. Stroke area (SA) was calculated as the difference between EDA and ESA and area ejection fraction ( $EF_a$ ) as the fractional change in area from ED to ES. For each patient, EDA, ESA and SA were normalized by dividing by body surface area and the three beats averaged. The results were then averaged over the entire patient group.

Segmental wall motion analysis to measure FS was performed in Matlab using routines developed in this laboratory. One hundred evenly spaced radial chords were generated from the centroids of the traced borders and numbered clockwise so that chord 1 represented the mid-posterior wall, 25 the mid-lateral wall, 50 the mid-anterior wall and 75 the mid-septum. The ED and ES borders for each cardiac cycle were then superimposed on each other by aligning their centroids (floating centroid method). Fractional shortening of each chord from ED to ES was calculated by dividing the change in length of the chord by its ED length. Again, the three beats were averaged for each patient and the results averaged over the entire patient group.

Local circumferential wall stress was estimated at these points using the Laplace equation for a thick walled cylinder as described by Janz [2]:

$$\sigma = \Pr/h \tag{3}$$

where  $\sigma$  is the circumferential wall stress, P is pressure, r is the local circumferential radius of curvature at the endocardium and h is the local wall thickness. ED pressure (EDP) was taken at the beginning of the rapid upstroke and ES pressure (ESP) at the beginning of the rapid downstroke in LVP. Endocardial local radius of curvature was calculated as the reciprocal of local curvature, found using a method described by Matas et al. [3] and filtered using a 5-point moving average filter. Local wall thickness was calculated as the difference between the lengths of the epicardial and endocardial radial chords. Average values of these parameters were calculated for each patient from the three cardiac cycles and an average stress was computed from these, with the results averaged over the entire patient group.

#### RESULTS

As a preliminary study, 3 patients were analyzed. Immediately following VSD closure they showed a decrease in EDA, SA and  $EF_a$ , as well as depression of FS localized to the septal and lateral wall segments. This was consistent with previous results.

A plot of estimated regional ES wall stress (mm Hg) before (PRE) and after (POST) surgery averaged over the 3 patients is shown in Figure 1, where chords 1, 25, 50 and 75 represent the mid-points of the posterior (post), lateral (lat), anterior (ant) and septal (sept) walls, respectively.



Figure 1. Estimated ES stress before and after VSD closure

Here we see a large decrease in ES wall stress after VSD repair isolated to segments near the mid-lateral and mid-septal walls, with slight increases in stress elsewhere. The same pattern was seen in changes of LV radius of curvature. Also with defect closure, there was a small regionally homogeneous increase in wall thickness and an increase in ESP. There were no obvious changes in ED wall stress.

#### DISCUSSION

Preload is the load imposed on the myocardium at ED and afterload the load at ES. With all other myocardial properties held constant, a change in preload or afterload will mean a change in the amount of myocardial contraction. So, regionally heterogeneous changes in preload or afterload in the LV could explain heterogeneous changes in LV contraction. If acute changes in either LV geometry, wall thickness or pressure immediately following VSD repair are regionally heterogeneous, then changes in myocardial wall stress will be so as well.

Here we have shown regionally heterogeneous changes in local LV afterload (ES  $\sigma$ ) due to acute local changes in ES radius of curvature and wall thickness. After surgery, ES radius of curvature decreased in the lateral wall and septum. At the same time, there was a regionally homogeneous increase in wall thickness and an increase in ESP. An increase in ESP would tend to increase myocardial wall stress, however the large decrease in radius of curvature in the lateral wall and septum combined with the uniform increase in wall thickness meant an overall decrease in afterload in these segments.

In previous work, we speculated that acute functional depression following VSD repair was due to localized impairment of septal function. It has been shown that when assessing regional wall motion with hypokinesia of one region, a floating centroid is drawn toward the abnormal segment, diminishing the extent of abnormal motion and projecting it to the contralateral wall [4]. Thus we suggested that the observed decrease in FS in the mid-septum and mid-lateral wall may in fact mostly have occurred in the septum and the decrease in lateral wall FS was simply an artifact of the use of a floating centroid. The results shown here suggest that real changes in loading may occur both in the septum and lateral wall.

While the results from this analysis are promising, instability in the measurement of local radius of curvature must be resolved before further work can be done. Local radius of curvature was found by taking the reciprocal of local curvature, a method that proved to be very sensitive to noise in the traced borders at low curvature, causing large spikes in the estimated stress. This meant that when stress was averaged over multiple patients it was often dominated in certain segments by the values from a single patient.

To properly optimize a method to measure local curvature, we must determine the best number of neighboring points to be used in the fit. By calculating the sum of squared errors (SSE) between local curvature measured from a manual tracing of an ellipse to the exact analytically determined values of local curvature we can determine the best number of points to use when measuring local curvature, as seen in Figure 2.



Figure 2. SSE of measured local curvature for a varying number of fitting points

Here the lowest SSE occurs when 9 points are used in the fit of local curvature. By this method we can improve our determination of local curvature and improve our estimates of regional stress. However, the fact remains that our analysis uses a simple, isotropic cylindrical model of the LV to estimate local stress. Because of the complex 3D geometry of the LV, the highly nonlinear material properties of the myocardium and the effect of the pressure in the right ventricle on the stress seen in the interventricular septum, this may be a poor first approximation of the actual regional circumferential stress in the LV. Future studies will explore the impact of using a geometrically realistic finite element model.

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