

SBC2010-19* ' %

**ASSESSMENT OF ENERGY LOSS DUE TO PULMONARY VALVE
INSUFFICIENCY IN TETRALOGY OF FALLOT PHYSIOLOGY USING PATIENT SPECIFIC
GEOMETRY**

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ABSTRACT

Right ventricular (RV) enlargement and pulmonary valve insufficiency (PI) are well-known, unavoidable long term sequelae encountered by patients who undergo tetralogy of Fallot (TOF) surgery. Energy losses in the pulmonary artery (PA) of TOF and normal subjects was calculated using patient specific geometry and pressure and flow data to develop quantifiable clinical diagnostic parameters. The TOF subject shows 35% higher energy losses than our normal subject.

INTRODUCTION

Tetralogy of Fallot (TOF), the most common cause of "blue-baby syndrome" consists of four interrelated anatomical defects: 1) *Ventricular Septal Defect*: a hole between the right and the left ventricle; 2) *Pulmonary Stenosis*: constriction of the pulmonary outflow tract, 3) *RV Hypertrophy*: pressure overloading of RV, and 4) *Overriding Aorta*: Aorta overrides the RV [1]. TOF has now been successfully repaired in infants and small children for more than 50 years. Consequently, there are now an estimated 100,000 adult "repaired TOF" (rTOF) patients in the United States alone. The long-term sequelae of the disease, namely PI and RV enlargement, have become important. PI, when severe, abnormally alters the RV loading conditions, thereby triggering RV hypertrophy and dilatation. In turn, RV dilatation can evolve into irreversible RV myocardial contractile dysfunction, and has been related to sudden death in many rTOF patients [2].

Although PI and RV enlargement in rTOF patients can be quantified by cardiac imaging, such as MRI or echocardiography, neither of these modalities offer predictive diagnostic parameters to help optimize timing of surgical re-intervention in rTOF patients. Our previous research showed that energy based parameters such as RV

stroke work, i.e., energy transfer rate to the blood being pushed into PA, can discern performance of RV and PV between normal and rTOF patient, and thus offer more predictive functional indexes. A few researchers have used similar energy based parameters in assessment and therapies for other congenital cardiovascular pathophysiology [3, 4].

The objective of this study was to compute and compare *energy loss*, as well as inlet and outlet *energy transfer rates in the branch PA*, between an rTOF subject and a normal control, using *patient specific, MRI based, reconstructed PA geometry and cine-phase contrast flow/velocity measurement*. The outcome of this study will lead to quantifiable diagnostic parameters of RV inefficiency and deterioration in rTOF patients.

METHOD

For this study two subjects (age-, sex- and size-matched), one normal (Age: 4 years, Sex: male, Wt.: 20.3 Kg, BSA: 0.78 m², Stroke volume: 52 ml) and other rTOF (Age: 5 years, Sex: male, Wt.: 16.9 Kg, BSA: 0.72 m², Stroke volume: 56 ml) were selected.

PA Geometry: Patient specific PA geometry was reconstructed from the individual MRI images of an rTOF subject and a normal control. Contrast-enhanced 3D MRI images were loaded into a commercially available image processing software MIMICS (Materialise, Inc., Leuven, Belgium) to render geometry, by assigning appropriate grey scale values to the PA region. The surface of the PA region was then exported as stereo-lithographic (STL) mesh to create blood flow domain for subsequent mesh generation for blood flow calculations.

Pressure-Flow Measurements: The subjects underwent clinical cardiac catheterization, at which time pressure variation with time over at least one complete cardiac cycle was recorded in at least one PA

location. Flow measurements at the PA inlet and outlet were performed by PA *Flow Imaging* using retrospective ECG-gating, through-plane velocity-encoded phase contrast (PC) technique with 18-22 phases at each site. Catheterization and MRI procedures were performed within 3 weeks of one another for both subjects. The pressure versus time and flow versus time were co-registered using ECG tracings inherently present in both data sets.

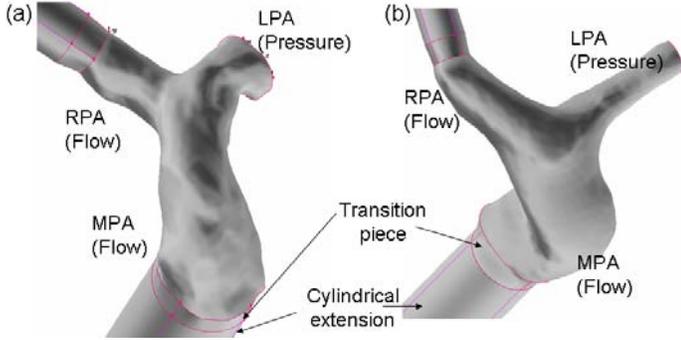


Figure 1: Individual branch PA geometry of (a) normal and (b) rTOF subject reconstructed using their respective MR images. For blood flow calculations, MPA and RPA ends are extruded into cylinder of equivalent area with the transition piece connecting the cylinder to the reconstructed cross-section. Locations of measurements of flow and pressure are shown. Womersley type velocity profiles were applied at the ends of the cylindrical extension as boundary conditions for CFD

Pressure-Flow Energetics: At any inlet or outlet i (where $i = \text{MPA, LPA or RPA}$) a transient energy transfer rate based on pressure-flow energy is given by,

$$\dot{E}_i = \left(p_i + \frac{1}{2} \rho v_i^2 \right) Q_i \quad (1)$$

where, ρ , the density of blood was taken to be 1.035 gm/cc, Q_i , p_i and v_i are the blood flow rate, pressure and area averaged velocity respectively with v_i given by, $v_i = Q_i / A_i$, with A_i , being the cross-sectional area of the inlet, $i = \text{MPA, LPA or RPA}$. And the rate of energy loss in the branch PA, \dot{E}_{loss} was calculated using,

$$\dot{E}_{loss} = \dot{E}_{mpa} - (\dot{E}_{lpa} + \dot{E}_{rpa}) \quad (2)$$

Blood Flow Calculation in the Branch PA: Pressures and velocities involved in Eq. 1 and 2 were calculated using CFD. The blood was modeled as incompressible, shear thinning non-Newtonian fluid using a Carreau model [5]. Measured pulsatile pressure was applied at LPA. *Instead of simplistic boundary conditions such as spatially uniform or parabolic, Womersley type velocity profile calculated from flow-rates was applied at the extended inlet (MPA) and outlet (RPA)* [6]. The blood flow domain was meshed with tetrahedral elements with mesh size 0.5 mm to 0.75 mm, whereas the cylindrical extensions were meshed with triangular prisms. Numerical calculations were performed using finite volume solver (FLUENT, ANSYS Inc., Canonsburg).

The energy transfer rate at the MPA, LPA and RPA were calculated using Eq. 1. The rate of energy transferred by the RV to the blood being pushed into MPA is not uniform over the cardiac cycle. Equation 1 represents the sum total of energy blood has at any given section. Energy loss between inlet and outlet is computed using Eq. 2 which is dissipated in viscous losses.

RESULTS AND DISCUSSION

The energy losses computed using Eq 2 are plotted (Fig. 2) over one cardiac cycle starting with the beginning of systole (defined as the beginning of the ECG R wave). The cardiac period of the normal subject was 0.74 sec and that of the rTOF subject was 0.58 sec. The peak loss happens for both the subjects during systole. Peak energy loss was 0.41 J/sec for the rTOFsubject, 120% higher than the peak loss of 0.18 J/sec for the normal subject (a). The average rate of energy loss over one complete cardiac cycle for the rTOF subject was 0.038 J/sec, 70% greater than the normal subject (0.065 J/sec).

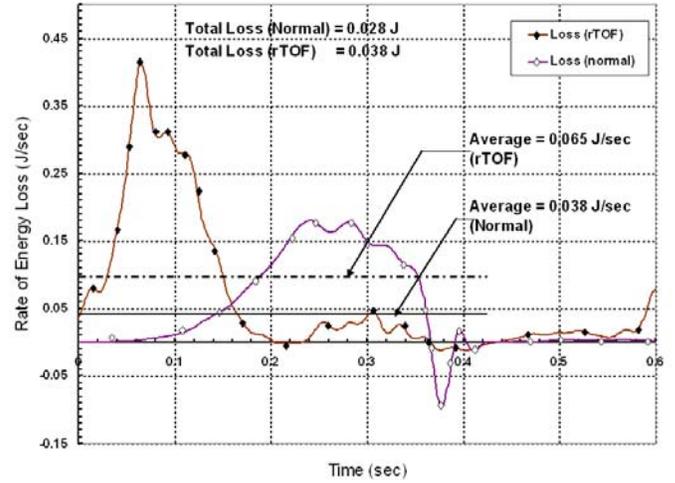


Figure 2: Variation of the rate energy loss (Eq. 2) over one cardiac cycle for a normal and rTOF subjects of comparable physiology. rTOF subject shows significantly higher total as well as average loss.

Figure 2 also demonstrates a phase difference for peak energy loss between the normal and the rTOF subject. The variation of energy transfer rates with time at the MPA also shows a similar phase shift. Our conclusion from this observation is that the transfer rate more quickly peaks for the rTOF subject, because very little effort is needed by the RV to push the blood across the PV region, since there is no valve to restrict flow. Additional energy losses due to wall motion are not accounted for by the technique described, so are the subject of a future study using variable compliance PA wall modeling.

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