ACCURACY AND FEASIBILITY OF REAL-TIME THREE DIMENSIONAL TRANSTHORACIC ECHOCARDIOGRAPHY IN VALVULAR PULMONARY STENOSIS PRE AND POST CATHETERIZATION

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Abstract

This is a prospective comparative clinical study including 30 patients previously diagnosed to have moderate to severe pulmonary valvular stenosis to compare between the pulmonary valve annulus by 2D echo , RT-3D echo versus Angiographic catheter calibration in the catheter laboratory before transvalvular balloon angioplasty dilatation. This detected the superiority of the RT-3D images in visualization of the thickened valvular cusps, its motility, fissuring of the commisures and calculation of the valve annulus and surface area regarding the choice of the appropriate size of the balloon for dilatation.

Key Words:

Congenital Heart Defect, Pulmonary Stenosis, Echocardiography, Pulmonary Artery Catheterization.

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List of abbreviation

- **2D:** Two Dimensional
- **2D-TTE:** Two Dimensional Trans Thoracic Echocardiography
- **3D:** Three Dimensional
- **3DE:** Three Dimensional Echocardiography
- **3D-TTE:**ThreeDimensional Trans Thoracic Echocardiography
- **ABG**: arterial blood gases
- **An:** Annulus
- **AS:** Aortic stenosis
- **ASD**: Atrial septal defect
- **AVSD**: Atrioventricular septal defect
- **B:** Balloon size
- **BMI:** Body Mass Index
- **BSA:** Body Surface Area
- **C:** Circumference
- **CHD**: congenital heart defect
- **CoA**: Coarctation of the aorta
- **CT:** Computerized Tomography
- **D1:** Diameter 1
- **D2:** Diameter 2
- **DILV**: Double inlet left ventricle

- **DORV**: Double outlet right ventricle
- **ECG:** Electro Cardio Gram
- **EF:** Ejection Fraction
- **F:** Female
- **HLHS**: Hypoplastic left heart syndrome
- **HRHS** Hypoplastic right heart syndrome
- **IAA**: Interrupted aortic arch
- **l-TGA**: levo-Transposition of the great arteries
- **d-TGA**: dextro-Transposition of the great arteries
- **M:** Male
- **MS:** Mitral stenosis
- **PA**: Pulmonary Artery
- **PAPVC**: Partial anomalous pulmonary venous connection
- **PDA**: Patent ductus arteriosus
- **PS:** Pulmonary stenosis
- **PV:** Pulmonary Valve
- **PVA:** Pulmonary Valve Area
- **PVS**: Pulmonic Valvular Stenosis
- **RT3D:** Real Time Three Dimensional
- **RT3D-TTE:** Real Time Three Dimensional Trans Thoracic Echocardiography
- **RVOT:** Right Ventricular Outflow Tract

- **SAX**-view: Short Axis –view
- **SD:** Standard Deviation
- **SS**: Scimitar syndrome
- **TAPVC**: Total anomalous pulmonary venous connection
- **TGA**: Transposition of the great vessels
- **ToF**: Tetralogy of Fallot
- **VEGF**: The vascular endothelial growth factor
- **VPS**: Valvular Pulmonary Stenosis
- **VSD**: Ventricular septal defect

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INTRODUCTION

Precise assessment of congenital heart lesions requires inferential evaluation from multiple Two-Dimensional Trans-Thoracic Echocardiography (2D-TTE). For many years it has been an important diagnostic tool for the evaluation of morphological and hemodynamic information of congenital heart diseases **(Castro et al; 2006).** Significant advances in ultrasound, such as the transition from M-mode to 2-Dimensional (2D) imaging, coupled with the addition of pulsed and continuous wave Doppler and color flow, have established echocardiography as one of the most clinically used diagnostic tools in daily cardiology practice **(Lang et al; 2006).** Although 2D echocardiography has impacted our ability to diagnose valvular and ischemic heart disease, the concept of 3-Dimensional (3D) imaging has been envisioned by numerous investigators as a natural evolution of this technology. Significant advances in ultrasound, electronic, and computer technology have thrust the field forward toward the development of a fully sampled matrix array transducer and on-line 3D display of rendered images, as well as software for post processing and quantification. The ease of data acquisition, the ability to image the entire heart nearly in real time, as well as the ability to focus on a specific structure in a single beat have brought 3D echocardiography closer to routine clinical use . Within several years of its inception, real-time 3D technology has sparked new endeavors in research and opened a glimpse into the future of echocardiography ***(Krenning et al; 2006).*** It has recently been shown that 3D color Doppler imaging can provide accurate

measurements of flow in the great vessels and ventricles by sampling the entire cross-sectional flow profile through the ventricular outflow tract, thus allowing the calculation of valvular flow volume, regurgitant volume, fraction, and orifice area ***(Chen et al; 2005).***

Aim of Work

The purpose of our clinical study is to evaluate the morphology of the pulmonary valve in Patients with valvular pulmonary stenosis assessed by real-time 3D echocardiogreaphy compared to 2D echocardiography, and measurement of the diameter of the pulmonary valve by 2D echo and compare it to measurement of perimeter and surface area by reconstructed images from 3D. Then followed by balloon dilatation through catherization of these Patients and re-evaluation later on by 2D & 3D echo & subsequent measurement of the pulmonary valve post dilatation.

Congenital Heart Defect

A **congenital heart defect** (**CHD**) is a defect in the structure of the heart and great vessels which is present at birth. Many types of heart defects exist, most of which either obstruct blood flow in the heart or vessels near it, or cause blood to flow through the heart in an abnormal pattern. Other defects, such as long QT syndrome, affect the heart's rhythm. Heart defects are among the most common birth defects and are the leading cause of birth defect-related deaths. Approximately 9 people in 1000 are born with a congenital heart defect. Many defects don't need treatment, but some complex congenital heart defects require medication or surgery.(Hoffman et al; 2002)

Embryology (Heart development):

There is a complex sequence of events that result in a well formed heart at birth and disruption of any portion may result in a defect. **(Mitchell et al; 2010**) The orderly timing of cell growth, cell migration, and programmed cell death ("apoptosis") has been studied extensively and the genes that control the process are being elucidated. Around day 15 of development, the cells that will become the heart exist in two horseshoe shaped bands of the middle tissue layer (mesoderm), **(Srivastava, 2006)** and some cells migrate from portion of the outer layer (ectoderm), the neural crest which is the source of a variety of cells found throughout the body. On day 19 of development, a pair of vascular elements, the "endocardial tubes", form. The tubes fuse when cells between then undergo programmed death and cells from the first heart field migrate to the tube, and

form a ring of heart cells (myocytes) around it by day 21. On day 22, the heart begins to beat and by day 24, blood is circulating. **(Tidyman et al; 2009)**

At day 22, the circulatory system is bilaterally symmetrical with paired vessels on each side and the heart consisting of a simple tube located in the midline of the body layout. The portion that will become the atria and will be located closest to the head are the most distant from the head. From days 23 through 28, the heart tube folds and twists, with the future ventricles moving left of center (the ultimate location of the heart) and the atria moving towards the head. **(Tidyman et al; 2009)**

On day 28, areas of tissue in the heart tube begin to expand inwards; after about two weeks, these expansions, the membranous "septum primum" and the muscular "endocardial cushions", fuse to form the four heart chambers of the heart. A failure to fuse properly will result in a defect that may allow blood to leak between chambers. After this happens, cells which have migrated from the neural crest begin to divide the bulbus cordis, the main outflow tract is divided in two by the growth a spiraling septum, becoming the great vessels—the ascending segment of the aorta and the pulmonary trunk. If the separation is incomplete, the result is a "persistent truncus arteriosis". The vessels may be reversed ("transposition of the great vessels"). The two halves of the split tract must migrate into the correct positions over the appropriate ventricles. A failure may result in some blood flowing into the wrong vessel (*e.g.* overriding aorta). The four chambered heart and the great vessels

have features required for fetal growth. The lungs are unexpanded and cannot accommodate the full circulatory volume. Two structures exist to shunt blood flow away from the lungs. Cells in part of the septum primum die creating a hole while muscle cells, the "septum secundum", grow along the right atrial side the septum primum, except for one region, leaving a gap through which blood can pass from the right artium to the left atrium, the foramen ovale. A small vessel, the ductus arteriosus allows blood from the pulmonary artery to pass to the aorta. **(Larsen 1993)**

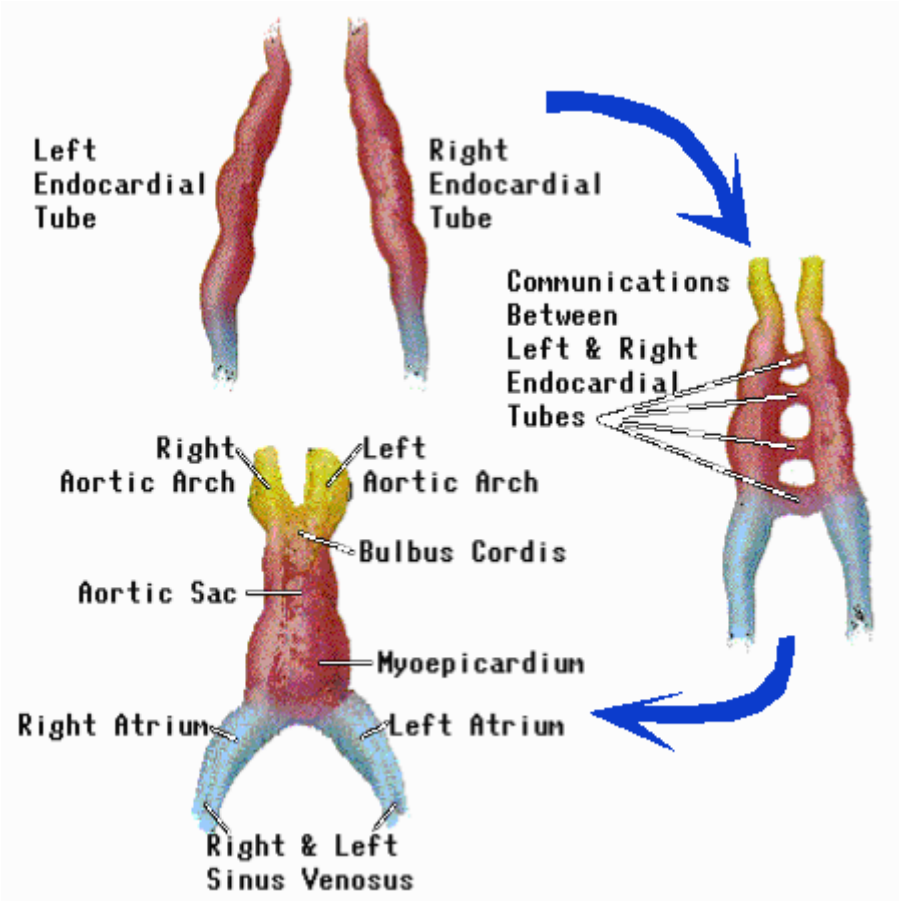

Fig .1. Showing the embryology and development of the heart. **(Hoffman 2005)**