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## Ebstein's Anomaly of the Tricuspid Valve in the Neonate

By Duraisamy Balaguru, MD;  
P. Syamasundar Rao, MD

### Introduction

In the previous issues of *Neonatology Today*, the most common cyanotic congenital heart defects, the so called 5Ts, namely, Transposition of the Great Arteries,<sup>1</sup> Tetralogy of Fallot,<sup>2</sup> Tricuspid Atresia<sup>3</sup> and Total Anomalous Pulmonary Venous Connection,<sup>4</sup> and Truncus Arteriosus<sup>5</sup> as well as Hypoplastic Left Heart Syndrome<sup>6</sup> were discussed. In this issue of *Neonatology Today* we will address Ebstein's Anomaly of the tricuspid valve.

Ebstein's Anomaly of the tricuspid valve is characterized by downward displacement of the septal and posterior leaflets of tricuspid valve, leading to varying degrees of the tricuspid regurgitation and right atrial enlargement (Figure 1). This is a rare lesion that accounts for 0.3 to 0.6% of all congenital heart defects.<sup>7</sup>

### Etiology

No single gene defect has been consistently identified to be associated with Ebstein's Anomaly. The majority of cases are sporadic. Exposure to lithium during pregnancy has been reported as an etiologic factor.<sup>8,9</sup> However, some recent studies have challenged Lithium as an etiologic factor.<sup>10</sup> There is a higher incidence for recurrence in the offspring of women with Ebstein's Anomaly (6%) than seen in the offspring of men (0.6%).<sup>11</sup>

### Pathology

Displacement of septal and posterior leaflets of tricuspid valve is thought to be secondary to fail-

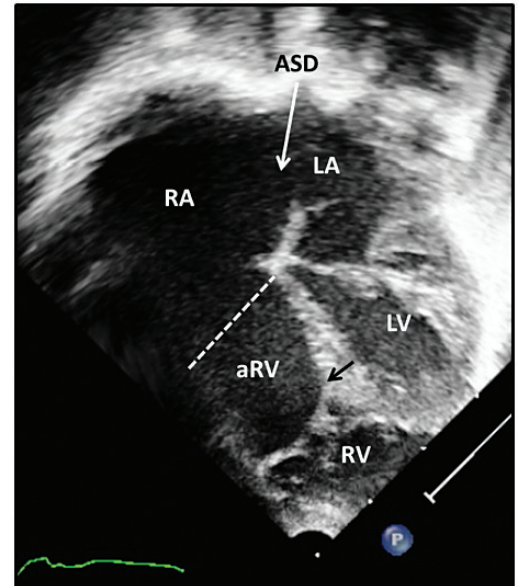


Figure 1. Selected video frame from apical, 4-chamber, two-dimensional echocardiographic view of 1-day old newborn shows apical displacement of the septal leaflet of the tricuspid valve (Black arrow). Dotted line denotes the level of true tricuspid valve annulus. Severe right atrial (RA) enlargement is noted. Atrial septum bows towards left-side (white arrow). aRV, "atrialized" portion of right ventricle; ASD, atrial septal defect (or Patent Foramen Ovale); LA, left atrium; LV, left ventricle; RV, right ventricle.

ure of delamination of the leaflets. The anterior leaflet is not usually affected.<sup>12</sup> In addition, the tricuspid valve leaflets are dysplastic and have abnormal chordal attachments. Rarely, tricuspid stenosis, and even atresia, may be present. The combination of these factors leads to incomplete closure of the tricuspid valve orifice and

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regurgitation. Tricuspid regurgitation starts in utero. The severity of tricuspid regurgitation varies with the severity of the anatomic defect.<sup>14</sup> Right atrium (RA) enlarges as a result of tricuspid regurgitation. The size of the RA at birth depends on the severity of tricuspid regurgitation in utero.

Displacement of the tricuspid leaflets results in a portion of the right ventricle (RV) becoming part of the right atrium (“atrialized” portion of RV; labelled “aRV” in Figure 1) and leaves the relatively smaller portion of the RV for pumping function. Furthermore, RV outflow tract obstruction<sup>15</sup> may be caused either by abnormal attachment of the tricuspid valve chordae, large anterior leaflet or associated pulmonary valve stenosis or atresia. Pulmonary valve atresia noted in newborns with Ebstein’s Anomaly may be either anatomic or “functional” (see below under “Clinical and Hemodynamic Implications in Newborn”).

### Associated Lesions

The presence of accessory conduction pathway causing Wolf-Parkinson-White (WPW) Syndrome is noted in nearly 20% of patients with Ebstein’s Anomaly.<sup>16</sup> Atrial Septal Defect or Patent Foramen Ovale (PFO) is commonly seen with Ebstein’s Anomaly, but other lesions are rare and include: Ventricular Septal Defect (VSD), Tetralogy of Fallot (TOF), Double-Outlet Right Ventricle Transposition of the Great Arteries (TGA) and Absent Pulmonary Valve Syndrome.<sup>17</sup>

### Clinical and Hemodynamic Implications in Newborn

Main determinants of hemodynamic abnormality and clinical presentation in the neonate include: (i) the degree of tricuspid regurgitation which, in turn, is dependent on degree of displacement of tricuspid valve leaflets, (ii) patency of RV outflow tract and (iii) pulmonary vascular resistance (PVR).<sup>1,18</sup>

Milder cases of Ebstein’s Anomaly may go undiagnosed unless an echocardiogram was performed for another reason. In moderate cases, tricuspid regurgitation leads to RA enlargement. Because there is elevated pulmonary artery pressures (pulmonary hypertension) in all babies during the postnatal transitional circulation, tricuspid valve regurgitation will present as an easily detectable heart murmur. There may be some degree of right-to-left shunting at the atrial level causing mild to moderate cyanosis. Such cyanosis will improve as the postnatal decrease in PVR occurs. Babies with mild and moderate severity are likely to be discharged home without any surgical intervention as a newborn. They may, however, need some surgical intervention later in life.

Severe tricuspid regurgitation is secondary to severe displacement of the tricuspid valve and associated abnormalities of chordal attachments, tricuspid valve leaflets and RV outflow

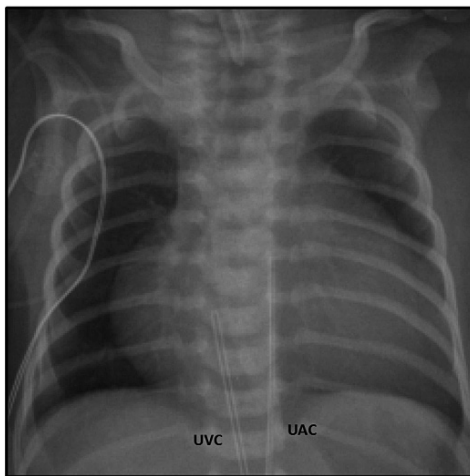


Figure 2. Chest X-ray of a newborn with severe Ebstein’s Anomaly showing severe cardiomegaly. Moderate pulmonary oligemia is present. Umbilical arterial (UAC) and venous (UVC) catheters are marked.

tract obstruction. Severe enlargement of the right atrium leads to the characteristic chest x-ray with severe cardiomegaly (Figure 2). With atrialization of a significant portion of RV cavity, there is inadequate myocardium available to generate adequate RV pressure to overcome PVR immediately after birth. Therefore, the RV is unable to open the pulmonary valve even though the pulmonary valve may be anatomically normal. This is described as “functional” pulmonary atresia. This situation is worsened by the presence of a Patent Ductus Arteriosus (PDA) either naturally occurring or secondary to prostaglandin infusion given for treatment for cyanosis. “Functional” pulmonary atresia should be differentiated from anatomic pulmonary atresia that may also occur with Ebstein’s Anomaly where there is fusion of pulmonary valve leaflets causing anatomic atresia. RV outflow tract obstruction may also occur in Ebstein’s Anomaly either from abnormal chordal attachments of the tricuspid valve or from the large, anterior tricuspid valve leaflet itself.

In moderate cases with mild or moderate cyanosis, cyanosis and clinical condition improves when the PVR decreases in the first few days to few weeks of age. Medical management of a baby with Ebstein’s Anomaly is largely focused on managing these hemodynamic interactions until resolution of pulmonary hypertension.

### Clinical Presentation

Clinical presentation is commensurate to severity of the lesion. Mild cases are usually diagnosed later in life, sometimes in adulthood when an echocardiogram is performed for a murmur, arrhythmia or unexplained heart failure. Cases that present in utero or as newborn are usually the severe forms.

### Fetus

Fetal presentation may manifest as hydrops or fetal arrhythmia. Ebstein’s Anomaly is also

recognized in routine antenatal ultrasonographic screening as well as when causes of asymptomatic cardiomegaly, right atrial enlargement or tricuspid regurgitation are investigated. There may be associated lung hypoplasia, largely related to the size of the heart. Severe forms of fetal Ebstein’s Anomaly have a high incidence of fetal loss.<sup>14</sup>

### Neonate

Milder cases are largely asymptomatic, and therefore, may have no abnormal findings except for, perhaps, a transient heart murmur from tricuspid regurgitation and oxygen saturations that are within normal range. Severe cases present with cyanosis which is secondary to combination of right to left shunt across PFO and diminished pulmonary blood flow. Severe cyanosis leads to metabolic acidosis and consequent decrease in myocardial contractility. Cyanosis is present in 50% of newborns with Ebstein’s Anomaly. Murmur, supraventricular tachycardia (SVT), heart failure and/or cardiomegaly on chest x-ray are other modes of presentation in newborns.<sup>1,18</sup>

### Older Children and Adults

Asymptomatic murmur, cardiomegaly on chest x-ray, heart failure and arrhythmias are presenting signs in older children and adults with Ebstein’s Anomaly.

### Physical Examination

Cyanosis is a common feature except in mild cases. Cardiac sounds (S3, S4 or both).<sup>18</sup> Holosystolic murmur of tricuspid regurgitation secondary to intrinsic valve abnormality and/or pulmonary hypertension is heard. When pulmonary hypertension resolves, holosystolic murmur becomes a shorter, systolic murmur with comparatively lower frequency due to lower RV systolic pressure and higher RA pressure. Low frequency, mid-diastolic murmur secondary to tricuspid valve stenosis (relative or true) may also be heard. Hyperdynamic precordium, presence of a thrill in the left lower sternal border, and liver enlargement are additional findings on examination, particularly in severe cases.

### Investigations

#### Chest X-ray

Ebstein’s Anomaly is one of the few causes of large cardiac silhouette. Cardiac enlargement on chest x-ray is mostly due to right atrial enlargement which is commensurate with the severity of tricuspid regurgitation (Figure 2). Oligemic lung fields are typical, but normal vascular markings may also be seen.

#### Electrocardiogram

Tall and peaked P waves indicating right atrial enlargement, relatively-low QRS voltages and right bundle branch block pattern are

typical findings in Ebstein's Anomaly (Figure 3). Prolongation of PR interval is noted in 2/3<sup>rd</sup>s of the neonates.<sup>19</sup> Features of WPW Syndrome with short PR interval with delta wave may be present.

### Echocardiogram and Doppler

Echocardiography is the modality of choice to obtain a complete diagnosis both in utero and in neonates. Neonatal echocardiography is performed after birth, regardless of fetal diagnosis, to assess the anatomy and the current physiologic status – estimating severity of tricuspid valve displacement, tricuspid regurgitation and right atrial size. An assessment of valve leaflets and their chordal attachments, the status of the right ventricle and its function and pulmonary artery pressure estimation and the status of PDA is made. The Celemajer Index, described below, provides prognostic evaluation from neonatal echocardiography as well.<sup>20</sup> Three-dimensional echocardiography adds to the understanding of tricuspid valve and right ventricular anatomy in Ebstein's Anomaly.

Fetal echocardiography provides diagnosis, information for in-utero management and prognosis for fetus with Ebstein's Anomaly.<sup>14,20,21</sup> Celemajer index and SAS score are frequently used in fetal echocardiogram to assess prognosis.

**Celemajer Index.** The apical 4-chamber view in fetal or neonatal echocardiogram (Figure 4) is used to derive Celemajer Index. Ratio of area of RA + atrialized portion of RV (RA+aRV in Figure 4) to the combined area of RV, left ventricle (LV) and left atrium (LA) are used for grading: Grade 1 (ratio < 0.5) had a mortality of 0%, Grade 2 (ratio 0.5 – 0.99) had a mortality of 10%, Grade 3 (ratio 1.0 – 1.49) had a mortality of 44% and Grade 4 (ratio > 1.5) had 100% mortality;<sup>20</sup> the higher the grade, the greater the mortality.

**Simpson-Andrews-Sharland Score (SAS Score).** SAS Score is based on observations at the first prenatal echocardiogram and includes: (i) Cardiothoracic ratio in fetal echocardiogram, (ii) Celemajer Index, (iii) RV-LV ratio, (iv) Reduced/absent pulmonary valve flow and (v) Retrograde ductus arteriosus flow.<sup>21</sup> This score predicts survivors vs. non-survivors and is useful in counseling during pregnancy. Possible scores range from 0 to 10. Similar to the Celemajer Index, higher scores correlate with higher mortality. When the score was ≤ 3, survival was 91%. There were no survivors when the score was ≥ 5 in their original study.<sup>21</sup>

A recent single-center study from Boston Children's Hospital suggested that (i) presence of severe tricuspid regurgitation, Celemajer Index > 1.0 and absence of forward flow through pulmonary valve were predictive of poor outcome.<sup>22</sup>

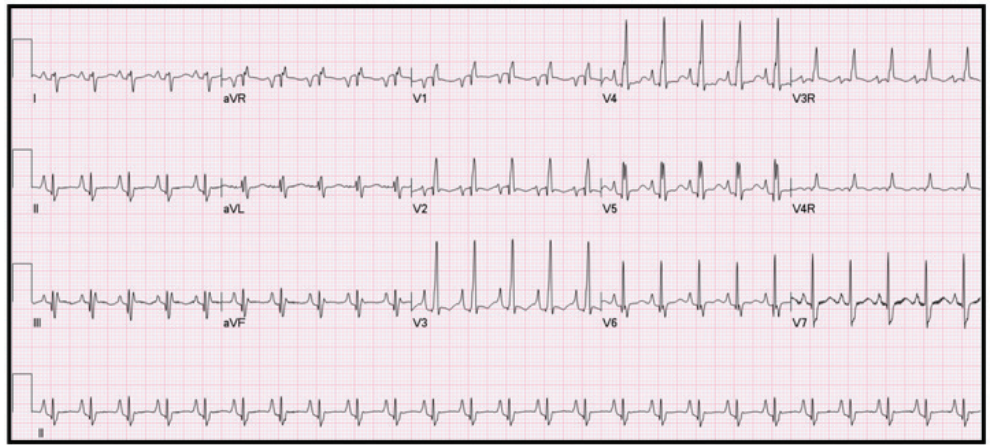


Figure 3. Electrocardiogram of a newborn with Ebstein's Anomaly. Note the tall, peaked P waves representing right atrial enlargement. Low voltage QRS complexes are noted in limb leads. rSR' pattern is present in leads V1 and V2.

### Cardiac Computed Tomography and Magnetic Resonance Imaging

These modalities usually are not necessary in a newborn with Ebstein's Anomaly.

### Cardiac Catheterization

There is no specific indication for cardiac catheterization for diagnostic purposes in a newborn with Ebstein's Anomaly with rare exceptions, perhaps, when pulmonary valve stenosis or atresia could not be confirmed by echocardiography or to evaluate an associated lesion in rare instances. Balloon pulmonary valvuloplasty may be helpful in carefully selected, rare patients with pulmonary valve stenosis or atresia. However, there is a potential for inducing a "central, circular shunt" if severe pulmonary regurgitation is induced

by the balloon procedure and the patient requires an aorto-pulmonary shunt at a later date. A central, circular shunt is a situation where there is severe pulmonary regurgitation, severe tricuspid regurgitation and a right-to-left shunt across PFO/ASD. A significant part of the aorto-pulmonary shunt flow may circulate "backwards" via the pulmonary valve, tricuspid valve and PFO/ASD into the left heart and flow into systemic circulation. In this situation, cyanosis is unrelieved or only partially relieved by an aorto-pulmonary shunt and the left heart has volume overload leading to heart failure. The benefit of balloon pulmonary valvuloplasty should be carefully weighed against the risk of inducing such a "circular" shunt.

### Differential Diagnosis

Severe cardiomegaly with pulmonary oligemia in a cyanotic newborn is highly suggestive of Ebstein's Anomaly; however, the following conditions should also be considered in such situations: critical pulmonary stenosis or pulmonary atresia with intact ventricular septum and "functional" pulmonary atresia. Other lesions such as tricuspid atresia, TGA and TOF may rarely mimic Ebstein's Anomaly. But, their clinical features are distinctive and echocardiography is used as confirmation to clearly differentiate these conditions from Ebstein's Anomaly.

In rare instances, tricuspid valve lesions other than Ebstein's Anomaly<sup>23</sup> may cause severe tricuspid regurgitation in a newborn. Such lesions include tricuspid valve dysplasia without displacement of its attachments, tricuspid valve prolapse, trauma, RV dysplasia, endocarditis and annular dilatation secondary to free pulmonary regurgitation. Therefore, it is important to ascertain the presence of the two cardinal findings of Ebstein's Anomaly, namely, (i) apical displacement of the septal leaflet of tricuspid valve (> 8 mm/m<sup>2</sup> of body weight) and (ii) the presence of a redundant, elongated anterior tricuspid valve leaflet.

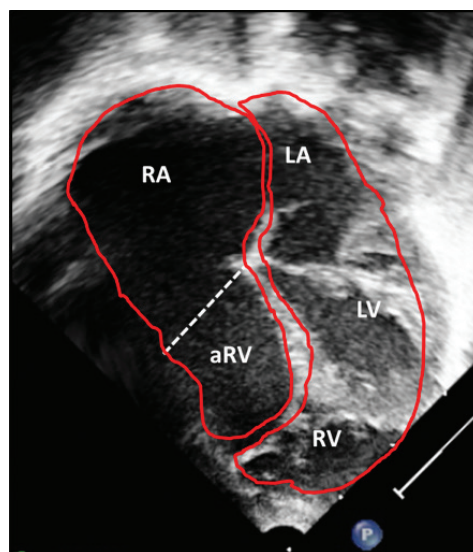


Figure 4. Same image as in Figure 1 is provided with red lines drawn to indicate the method of measurements for Celemajer Index calculation. Ratio of (area of RA+aRV)/(area of RV+LV+LA) is used to predict outcome in fetus or newborn with Ebstein's Anomaly. See text for details. Abbreviations are same as in Figure 1.

## Natural and “Unnatural” History

An early study from Boston Children’s Hospital (1971) reviewed the outcome of patients with Ebstein’s Anomaly and Isolated Ebstein’s Anomaly<sup>24</sup> – 70% survived up to 2 years and 50% survived up to 13 years. Ebstein’s Anomaly patients with associated anomalies, however, had a 15% survival at 2 years. A more recent study from Belgium (2010) in a cohort of 49 patients who were more than 16 years old reports the following.<sup>25</sup> The mean follow-up period was 11.4 years (1 – 32 years). Half of them (51%) had undergone tricuspid valve surgery. Eight patients required reoperation to repeat tricuspid valve repair. Twenty-six (51%) patients had SVT. Typical WPW Syndrome was noted in 15 (31%) and ablation was performed in 17 (34%). A pacemaker was implanted in 5 (10%).<sup>25</sup>

## Management

### Medical Management of Newborn

The management depends on severity of the symptoms and age at presentation and is discussed in detail in pediatric cardiology text books.<sup>7,26,27</sup> Asymptomatic cyanotic newborn do not need any active treatment unless cyanosis is severe. Cyanosis will resolve to a variable extent when PVR decreases with time. Treatment for severe cyanosis at birth consists of temporarily keeping the PDA open using PGE<sub>1</sub> infusion (0.05 – 0.1 mcg/kg/min) until pulmonary resistance drops. Occasionally, use of inhaled nitric oxide (iNO) to reduce PVR has helped to improve pulmonary blood flow and, hence, systemic oxygenation. Such therapy is usually needed for only a few days after which the patient can be safely weaned. Intubation and positive pressure ventilation may help to manage pulmonary hypertension more effectively. Deep sedation and muscle relaxant may be necessary for a few days to manage pulmonary hypertension. Correction of metabolic acidosis with bicarbonate infusions and inotropic infusions for low cardiac output may be needed.<sup>28</sup> Few neonates may require a surgical systemic-pulmonary shunt to maintain adequate pulmonary blood flow and thus, maintain adequate systemic oxygen saturation.

Features of heart failure secondary to severe tricuspid regurgitation may be treated with anti-failure medications such as Furosemide and Digoxin.

SVT related to accessory pathways or atrial flutter from enlarged RA should be controlled using appropriate anti-arrhythmic medications; Adenosine for acute control of SVT and suitable anti-arrhythmic medication such as Propranolol, Flecainide and Amiodarone for the long term control. Ablation of the accessory pathway causing SVT is reserved for older children and adults.

Restrictive PFO/ASD is rare and do not usually require balloon atrial septostomy. While

balloon atrial septostomy may relieve systemic venous congestion, one should be cognizant of the increase in cyanosis that may occur due to increased right to left shunting at the atrial level after the septostomy.

RV outflow tract obstruction is commonly secondary to anterior leaflet attachments. Therefore, balloon pulmonary valvuloplasty is unlikely to help unless valvar stenosis is a significant part of RV outflow tract obstruction.

## Surgical Management

Neonates who require surgical repair present a great challenge. Management objectives in a neonate are initially focused on avoiding surgical intervention, allowing adequate time for medical management to work. However, if the baby has significant cyanosis and heart failure, with or without RV outflow tract obstruction (anatomic or functional), surgical treatment will become unavoidable.<sup>29</sup>

Multiple surgical approaches have been described for treatment of Ebstein’s Anomaly. Most of these surgeries are better avoided in a newborn as much as possible and only performed when medical management is ineffective. The surgical options include various methods described by different surgeons. In essence, “Ebstein’s repair” would consist of repair of the tricuspid valve, plication of the atrialized portion of right ventricle or re-attaching the leaflets at the annulus-level and sometimes, placing a prosthetic valve. These repairs result in two-ventricle system and are better performed later in life.

Starnes procedure<sup>30</sup> is used when the newborn is too sick to wait for a later surgery and in principle, consists of excluding the right ventricle from circulation by closing the tricuspid valve with a patch and placing an aorto-pulmonary shunt for providing pulmonary blood flow. Later, the patient will undergo Glenn and Fontan operations at approximately 6 months and 3-4 years of age, respectively.<sup>30</sup> This latter option leads to a single-ventricle system. An intermediate option is a “One and a half ventricle repair” where the traditional Ebstein’s repair is performed, but a Glenn anastomosis is added so that the volume load for right ventricle is reduced.<sup>31</sup>

## Summary and Conclusion

Ebstein’s Anomaly is a rare congenital heart disease. Clinical manifestations vary depending upon the severity of the lesion. Mild forms may be asymptomatic and may not need any treatment. Moderate forms may be managed with relative ease. Severe forms of the disease are a challenge to manage. Prognosis depends on the severity of the lesion, age at presentation and type of surgical repair. Surgical outcomes have improved over time, but an early presentation as a fetus or newborn is associate with a poor prognosis.

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# Highlights from the Fetal Cardiac Symposium at Rush University in Chicago- June 5<sup>th</sup>-6<sup>th</sup>, 2014

By Karim Diab, MD, FACC, FASE

The Rush Center for Congenital Heart Disease launched its first national conference focusing on fetal cardiology on June 5<sup>th</sup>-6<sup>th</sup> on campus at Rush University Medical Center in Chicago. With its vibrant culture and warm summer, Chicago was a perfect location for bringing together experts in the field of Fetal Cardiology to the Midwest! With congenital heart defects being the most common birth defects in humans and with the low national prenatal detection rate of cardiac defects despite universal screening during pregnancy, the main goal of the conference was to help improve the status of prenatal diagnosis of Congenital Heart Disease both at the local and national levels by improving the technical skills in scanning the fetal heart. The event was also unique in the Chicago area because no such specialized meeting has been held recently in the Midwest.

The symposium, in its first year of launching, was a tremendous success and was sold out, with an audience of 160 registrants who came from 32 different countries and states within the USA. Seventeen percent of the registrants came from overseas, including countries such as Canada, Brazil, Egypt, Costa Rica, South Africa, India and Saudi Arabia. Most of the attendees (~60%), however, came from the Midwest states, highlighting the need for such a conference focusing on the fetal heart in this region. Although more than 50% of the attendees were physicians, there were about 40% sonographers attending the meeting, likely reflecting the attractiveness of hands-on workshops that provided the attendees with practical scanning opportunities rather than only didactic lectures. The attendees came from various specialties including Pediatric Cardiology, OB and MFM as well as other specialties such as Neonatology and Radiology.

The conference featured a two-day meeting that offered thorough and updated presentations on scanning the fetal heart and diagnosing and managing various common fetal congenital heart disease malformations. The activity was designated for a maximum of 15 AMA PRA Category 1 continuing medical education credits, 15 Continuing Medical Education (CME) credits, and 12.75 CME credits in Medical Sonography (SDMS). Lectures, given by an internationally acclaimed faculty in pediatric cardiology and Maternal-Fetal Medicine specialists, emphasized the basics of fetal cardiac scanning coupled with live case demonstrations and tips for diagnosing various anomalies.

There was intensive focus on anomalies of the four-chamber and outflow-tracts views, reflecting the recently published guidelines for screening for fetal heart disease. In addition, the symposium featured unique two-hour workshops on both days of the meeting which gave the attendees a unique opportunity to scan pregnant volunteers with both normal hearts and cardiac pathology. This provided an excellent opportunity for becoming more familiar with the required cardiac views including the 4-chamber, the outflow tracts and the three-vessel views. It also allowed participants to experience scanning using various technological instruments and machines that are currently on the market. All this was done under the supervision of expert faculty in the field of Fetal Cardiology and Maternal-Fetal Medicine.

The symposium started with an overview on basic fetal cardiac views and red flags from an obstetrician's perspective followed by a live scanning demonstration of a complete fetal echocardiographic study. It then sequentially focused on the essential screening views of the fetal heart including the four-chamber and the outflow tract views, as well as the three-vessel view. This demonstrated the normal findings as well as typical cardiac lesions diagnosed with the particular view which helped give the audience practical tips for scanning and diagnosing various cardiac malformations. Additional lectures focused on topics and lesions such as vascular rings, coarctation, heterotaxy syndrome, abnormalities of the PDA, borderline common cardiac findings. There was also a session on interesting audience cases which gave the audience ample opportunity to present challenging fetal cardiac cases and discuss them with the faculty.

The second day of the symposium started with a session focusing on cardiovascular physiology in the fetus with a normal heart and the fetus with specific cardiac lesions as well as fetal tachy-arrhythmias. The highlight of the second day, however, was an extensive session focusing on fetal cardiac and non-cardiac interventions. Drs. Simone and Carlos Pedra presented their recent data on fetal cardiac intervention including balloon aortic valvuloplasty for critical AS with impending HLHS as well as fetal atrial septostomy for restrictive or intact atrial septum. Dr. Jaeggi presented an interesting case of tricuspid atresia with restrictive atrial septum that needed stenting of the atrial septum!

Dr. Johnson also presented updates on Twin-Twin Transfusion Syndrome and current in utero therapies; a handout on the topic was also provided for the attendees.

Two families with babies with critical CHD presented their personal experience with one family "taken by surprise" as the baby was not prenatally diagnosed and the other had a prenatal diagnosis.

The second day continued with another hands-on workshop which provided more time for attendees to practice obtaining the appropriate fetal cardiac views and helped demonstrate the concepts presented during the didactic lectures.

Additional sessions focused on: the evaluation of fetal cardiac function, the use of and its future, and family counseling in fetal CHD. An interesting talk on stem cell tissue engineering for repair of CHD was also included.

Overall, the conference was well received by attendees and the average ratings (based on a Likert 5 point scale) for general satisfaction with the program were high, with an average rating of 4.62. In the area of presentation content and presentation effectiveness, the activity received a high rating as well indicating a high level of satisfaction with the presentations' content and the effectiveness of faculty.

Given the recent updates and revisions to the North American guidelines for a fetal anatomic ultrasound screen during the second trimester and their focus on cardiac screening, the need for such annual fetal symposia in different regions is a must without any doubt!

The directors of the meeting would like to thank all those who helped make the first symposium an enjoyable experience! This year, the symposium featured a longer meeting, 2 ½ days, with two hands-on sessions and more out-of-state speakers. Keep an eye out for the 2015 meeting notice as the registration sold out more than a month in advance for the 2014 meeting. For more information, visit the meeting website at: [www.FetalCardiacSymposium.com](http://www.FetalCardiacSymposium.com)

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# Highlights of the 2<sup>nd</sup> Bangkok International Fetal Echocardiography Conference

Mark Sklansky, MD; Alisa Limsuwan, MD

The 2<sup>nd</sup> Bangkok International Fetal Echocardiography Symposium, held January 14-16, 2015, proved to be even more successful than the inaugural symposium held in 2014. Like the first symposium, this conference was held at the majestic Shangri-La Hotel, located on Bangkok's famous Chao Phraya River. Both years, this pioneering international fetal echocardiography symposium in Thailand has attracted a truly international group of attendees, composed predominantly of physicians (pediatric cardiologists, maternal-fetal-medicine subspecialists, and trainees), but also included sonographers and representatives from industry.

The symposium, directed by Drs. Alisa Limsuwan and Suthep Wanitkun, and with organizational support from Drs. Poomiporn Katunyuwong, Patama Promsonthi, and Boonsri Chanrachakul, featured international speakers (Dr. Mark Sklansky—pediatric cardiologist at UCLA, Dr. Giuseppe Rizzo—maternal-fetal-medicine specialist from Rome, and Dr. Tze Kin Lau—Maternal-Fetal Medicine specialist from Hong Kong), as well as widely respected pediatric cardiology, maternal-fetal-medicine and pathology experts from Thailand. The symposium provided a comprehensive series of didactic lectures from experts in pathology, pediatric cardiology, Maternal-Fetal Medicine, and radiology. Woven seamlessly into the didactic schedule were clinically compelling case presenta-



tions, a series of hands-on opportunities for registrants to scan actual patients with guidance from Drs. Sklansky and Rizzo, and live scanning by Dr. Sklansky of a fetal patient with heterotaxy.

The symposium's didactic line-up began with formal presentations on fetal cardiac pathology, genetics, and physiology, followed by a discussion of first trimester evaluation and the role of nuchal translucency thickness evaluation. Next, speakers presented a series of talks on current guidelines for fetal cardiac screening, and basic and more advanced techniques for fetal cardiac evaluation. Following these background discussions, experts presented a broad series of detailed, clinically-oriented lectures on abnormalities of the four-chamber view and outflow tracts. The third and final day of the symposium included formal presentations and case presentations of fetal arrhythmias and of fetal 3D/4D cardiac imaging, an overview of fetal cardiac tumors and, finally, a discussion and summary of take-home pearls for all those involved with fetal cardiac imaging.

Throughout the conference, registrants enjoyed the incredible beauty and cuisine of the lavish Shangri-La Hotel, with regular breaks



and daily lunch at Shangri-La's world-class Next2 Cafe restaurant. During additional breaks, registrants enjoyed interacting with representatives from Philips, Life Vision/GE, and Berli Jucker (Aloka), who demonstrated their latest equipment and software.

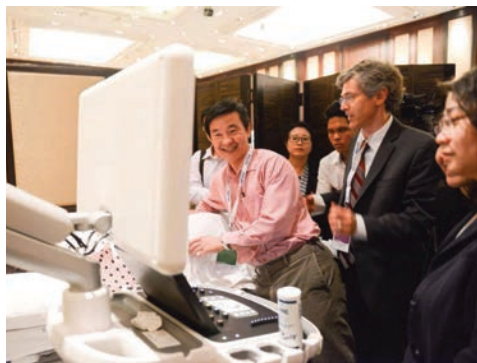
Given the tremendous success of this second international symposium, plans are already underway for the 3<sup>rd</sup> Bangkok International Fetal Echocardiography Symposium. Dates will be announced soon; please contact Dr. Alisa Limsuwan for additional information at: [alimsuwan@yahoo.com](mailto:alimsuwan@yahoo.com) or [bkkfetalecho.com](http://bkkfetalecho.com).

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St. Luke's Neonatology in Idaho is seeking a BC/BE Neonatologist and a Neonatal Nurse Practitioner to join 10 BC Neonatologists and 8 NNPs to assist with coverage of our three St. Luke's NICUs. The positions are primarily based at the NICU in Twin Falls, Idaho, which is in the process of expanding its scope of coverage to Level III status.

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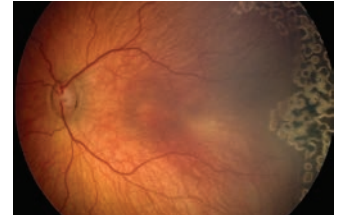


Image of Retinopathy of Prematurity

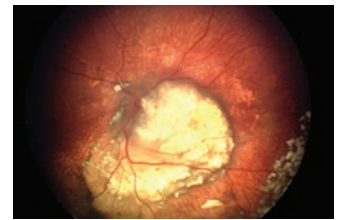


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