

CONGENITAL CARDIOLOGY TODAY

Timely News and Information for BC/BE Congenital/Structural Cardiologists and Surgeons

May 2014; Volume 12; Issue 5
International Edition

IN THIS ISSUE

Ruptured Non-Coronary Sinus of Valsalva Aneurysm Causing Right Heart Failure

By Nicole Brown, MD; Gruschen Veldtman, MBChB, FRCP; David Luis Simón Morales, MD; Christopher Learn, MD
~Page 1

Placement of an Implantable Cardioverter Defibrillator in a Pediatric Patient with Heparin-Induced Thrombocytopenia with Thrombosis, Bridging with Argatroban

By Joshua A. Daily, MD; Cristina Tarango, MD; Richard J. Ciosek, MD; Jeffrey B. Anderson, MD; David S. Spar, MD
~Page 8

Does Renal Denervation Have a Future? TRENDS Conference Gives a Resounding "Yes"

By Sameer Gafoor, MD
~Page 12

DEPARTMENT

Medical News Products & Information

~Page 13

June Medical Meeting Focus

~Page 14

Upcoming Medical Meetings

SCAI 2014 Scientific Sessions
May 28, 2014; Las Vegas, NV USA
www.scai.org

Basic & Advanced Fetal Cardiology Symposium Workshop
Jun 5-6, 2014; Chicago, IL USA
<http://fetalcardiacsymposium.com/>

PICS-AICS
(includes: Cardiac Imaging for Structural Heart Disease - A Special One Day Symposium at PICS)
Jun. 7-10, 2014; Chicago, IL USA
www.picsymposium.com

CONGENITAL CARDIOLOGY TODAY

Editorial and Subscription Offices
16 Cove Rd, Ste. 200
Westerly, RI 02891 USA
www.CongenitalCardiologyToday.com

Congenital Cardiology Today is the official publication of the CHiP Network (www.chipnetwork.org)

© 2003-2014 by Congenital Cardiology Today
ISSN: 1544-7787 (print); 1544-0499 (online).
Published monthly. All rights reserved.

Ruptured Non-Coronary Sinus of Valsalva Aneurysm Causing Right Heart Failure

By Nicole Brown, MD; Gruschen Veldtman, MBChB, FRCP; David Luis Simón Morales, MD; Christopher Learn, MD

Introduction

Sinus of Valsalva Aneurysms (SVAs) are rare cardiac defects that can be either congenital or acquired. The greatest concern is the risk of rupture. Even if unruptured, they can obstruct adjacent structures such as the coronary artery ostium or the right ventricular outflow tract. SVA are also associated with malignant arrhythmias and endocarditis,¹ and there is 3:1 male predominance. Although still rare, SVAs are both more common and up to five times more likely to rupture among Asian populations compared to Western populations.^{2,3} Rupture most commonly occurs in patients between 20-30 years of age.¹

The aortic sinuses are named after Antonio Valsalva, an Italian anatomist and physician. They extend from the aortic annulus to the sinotubular junction. The function of the sinuses of Valsalva remains incompletely understood and a topic of ongoing research. They provide space between the open aortic valve leaflets and the coronary ostia. The vortices present in the sinuses are thought to facilitate coronary blood flow and reduce stress on the aortic leaflets during closure.^{4, 5} Comparative models of different aortic grafts showed higher stress in the cylindrical model

compared to the bulbous sinus design.⁴ Preservation or reconstruction of the sinuses during valve-sparing aortic root surgery leads to re-creation of the vortices in the sinuses. This is believed to support normal leaflet movement, coaptation height, and valve durability. Despite abnormal leaflet motion seen with root reimplantation without aortic sinus re-creation, there is no definitive proof of adverse effects on valve durability.⁵

Abnormalities of sinus anatomy, such as the presence of an aneurysm, may profoundly alter the distribution of forces at the base of the aorta and disrupt aortic valve function. These altered forces may in turn worsen inherent medial weakness within an already compromised sinus. We report the case of a young man with a known non-coronary sinus of Valsalva aneurysm who presented following rupture of the aneurysm into the right atrium (RA).

"Sinus of Valsalva Aneurysms (SVAs) are rare cardiac defects that can be either congenital or acquired. The greatest concern is the risk of rupture."

CONGENITAL CARDIOLOGY TODAY

CALL FOR CASES AND OTHER ORIGINAL ARTICLES

Do you have interesting research results, observations, human interest stories, reports of meetings, etc. to share?

Submit your manuscript to: RichardK@CCT.bz

When Size is the Heart of the Matter

Tyshak NuCLEUS™

Percutaneous Transluminal Valvuloplasty Catheters



PTS-X™

Sizing Balloon

Catheters For Determining Congenital Defects



B|BRAUN

Interventional
Systems

Manufactured for:

B. Braun Interventional Systems Inc.

824 Twelfth Avenue

Bethlehem, PA 18018 USA

Tel: 1-877-VENA CAV (836-2228) (USA)

Fax: 1-610-849-1334

www.bisusa.org

Case Report

A 26-year-old male with partial trisomy 15 and severe global developmental delay suffered a right-sided embolic stroke at 14 years of age. This led to a diagnosis of a non-coronary sinus of Valsalva aneurysm. Surgical intervention was not pursued because of his multiple developmental and medical issues, including severe scoliosis and a non-functional dysplastic right kidney. He was instead started on warfarin. He was followed for over 10 years by pediatric cardiology with no new symptoms, a relatively stable international normalized ratio (INR), and no significant change by echocardiography.

At the time of his initial visit with the Cincinnati Children's Adolescent and Adult Congenital Heart Disease (AACHD) team, he was clinically stable with no cardiovascular symptoms, no murmurs, and stable electrocardiogram (ECG) findings. His echocardiogram showed a large non-coronary sinus of Valsalva aneurysm with stable dimensions (3.34 x 1.17 cm) that protruded into the right atrium at the level of the right ventricular inlet. In contrast to prior studies, an 8 mmHg mean inflow gradient across the tricuspid valve was noted. Right atrial dimensions remained normal despite the gradient. Elective repair and the risk of rupture were discussed with the parents, who opted for continued conservative management.

Nine months later, he presented to an outside emergency department with parental concerns of several weeks of changes in behavior, worsening oral intake, dry cough, and gradual onset of lower extremity edema. Doppler interrogation was negative for deep vein thrombosis. Labs revealed elevated blood urea nitrogen (BUN), uncharacteristically high INR, slightly elevated liver transaminases, mild thrombocytopenia, and proteinuria with hypoalbuminemia. No precipitating cause was found. He was re-evaluated by his primary care physician several days later. A renal ultrasound showed stable kidney findings, but new ascites and dilated hepatic veins. He was transferred directly to the AACHD clinic for further evaluation.

Vital signs were as follows: weight 46.3 kg (baseline 38.6 kg), blood pressure 113/71 mmHg, heart rate 100 bpm, respiratory rate 20 per minute, saturation 98% on room air. The exam showed very thin habitus, mild lethargy and injected sclera. His scoliosis, pectus excavatum, left-sided hemiplegia and contractures, and developmental delay were unchanged. His lungs were clear with normal work of breathing.

Cardiac exam revealed striking changes compared to his baseline exam. His precordium was now hyperdynamic with a right ventricular lift. On auscultation, he had a regular tachycardia, normal S1 and S2, a prominent S3/S4 gallop at the left lower sternal border, and a new harsh III/VI continuous murmur, loudest at the right upper sternal border with radiation throughout the precordium. Pulses were 2+ and symmetric with no radio-femoral delay, and capillary refill was normal with warm, well-perfused extremities. Mild 1+ edema was present in his lower extremities (left, slightly worse than right). Abdominal exam revealed ascites and an enlarged, pulsatile liver.

ECG showed normal sinus rhythm with right atrial enlargement and non-specific intra-ventricular conduction delay, and mildly prolonged QTc (Figure 1).

An echocardiogram confirmed rupture of the SVA through several small fenestrated defects of the non-coronary sinus with blood shunting left-to-right into the right atrium. Peak velocity across the defect was 4-5 m/sec. The aneurysm had increased in size (3.1cm x 2.8cm) and had an oblong shape. It protruded into the superior and medial right atrium and caused severe obstruction of RV inflow (Figure 2). The tricuspid valve mean gradient had increased to 14-15 mmHg, which may have been at least partially related to increased flow across the valve. There was no evidence of thrombus within the aneurysm. The aortic valve demonstrated only trivial regurgitation and no stenosis. The right atrium and ventricle were both dilated with prominent flow reversal in the hepatic veins. Biventricular systolic function was normal. A small-moderate pericardial effusion and abdominal ascites were also noted.

The patient was transferred to the cardiac intensive care unit and taken to the operating room the following morning. Routine pre-operative transesophageal echocardiogram (TEE) (Figure 3) confirmed findings on the transthoracic echocardiogram (TTE). Multiple fenestrations were seen in the non-coronary leaflet near the commissure with the right coronary leaflet. One jet was directed between the anterior and septal tricuspid valve leaflets and another larger jet was directed toward the right atrial appendage. The surgeon encountered a large effusion upon entering the pericardium. The right coronary artery was incidentally found to arise from the left sinus of Valsalva. There was a large amount of aneurysmal tissue present in the right atrium with adherence to the septal leaflet of the tricuspid valve. This caused poor tricuspid valve leaflet coaptation between the anterior and septal leaflets.

The surgeon resected some of the aneurysmal tissue within the RA. A trimmed patch of CorMatrix was used to close the defect from the aortic side, and the aneurysm tissue was also closed from the RA side to achieve a two-level closure. The aortic valve was repaired by reattaching some of the leaflet to the aortic wall, and it functioned well. The tricuspid valve was repaired by placing an Alfieri stitch between the anterior and septal leaflets.

Post-operative imaging showed no residual shunting and good function of the tricuspid and aortic valves. The patient did very well after surgery and had good spontaneous diuresis. He was discharged on the sixth post-operative day. We were able to stop his warfarin since the aneurysm and source of his prior embolic stroke had been removed. He was continued on aspirin for stroke prophylaxis. Within two weeks of his repair, he was back to his dry weight and had resolution of all signs of right heart failure.

Discussion

This case represents a subacute presentation of a ruptured non-coronary SVA. About 50-70% of patients have a gradual onset of fatigue, dyspnea, and other symptoms of congestive heart failure.¹ Similarly, our patient was asymptomatic until after his aneurysm ruptured. He developed evidence of right heart failure over the course of about four weeks. This was secondary to both the left-to-right shunt



Archiving Working Group
International Society for Nomenclature of
Paediatric and Congenital Heart Disease
ipccc-awg.net

from the aorta to the RA and the right ventricular inflow obstruction due to aneurysmal tissue from the SVA.

Aneurysms from the right sinus of Valsalva are most common (65-85%) with the majority of the rest originating from the noncoronary sinus (10-30%). An aneurysm from the left sinus occurs in less than 5% of cases.^{1,7} SVA rupture is usually into a right heart chamber—more frequently into the RV for right SVA and into the RA for non-coronary SVA, but either can occur. Rarely, rupture occurs into the left ventricle, left atrium, pulmonary artery, or pericardium.¹

Congenital aneurysms occur more frequently than acquired aneurysms, but are still rare among congenital lesions. Only 0.14-1.5 percent of congenital heart repairs are for congenital SVA.² They are thought to be due to abnormal elastic media between the aortic sinus and the aortic annulus and lack of continuity with the annulus fibrosus.^{2,6} Syndromes associated with SVA include Klippel Feil, Turner, Marfan, Ehlers-Danlos, and Loeys-Dietz. Trisomies 13 and 15, osteogenesis imperfecta, and rheumatoid arthritis have also been associated. We presume that our patient's SVA was congenital and likely due to his Partial Trisomy 15.

Acquired SVA has been attributed to bacterial endocarditis, syphilis, tuberculosis, atherosclerosis, and cystic medial necrosis. Trauma and hematoma as a complication of aortic valve surgery have also been linked to aneurysm formation.^{2,7} Several classification schemes of SVA have been proposed. The earliest was by Sakakibara and Konno in 1962, and later by Ring, Guo, and most recently Xin-Jin et al.^{2,3,8} No one classification system is widely used.

The patient's laboratory derangements were non-specific and could have been attributed to viral illness, injury of his single kidney, or poor nutrition. The red flags in this case were the loud continuous murmur and prominent gallop rhythm. These findings represented a definite change from his baseline cardiac exam and were harbingers of a serious cardiovascular problem. In this case, he was already known to have a SVA extending into the RA, thus rupture had to be a chief consideration. A new cardiac murmur, especially if continuous and accompanied by signs of right heart failure, should prompt concern for a ruptured SVA in any patient, and this clinical concern should guide appropriate echocardiogram image acquisition.

Historically, cardiac catheterization with aortography was the "gold standard" for diagnosing SVA; however, advances in echocardiography have allowed this non-invasive modality to be the initial diagnostic test of choice. Echocardiography can delineate the origin of the SVA, aneurysm size, protrusion into adjacent cardiac structures, and rupture, including shunt gradient. Aortic stenosis or regurgitation, tricuspid valve obstruction or insufficiency, chamber sizes, ventricular function, and pericardial effusion can also be investigated by echocardiography.^{1,9}

Echocardiogram can detect anomalies associated with SVA as well. Ventricular septal defects are the most commonly associated anomaly. Some reports indicate a higher incidence with right sinus of Valsalva involvement. Aortic valve regurgitation is the second most common association, especially if rupture has occurred. In that case, up to 50 percent may require aortic valve surgery.^{1,10} Other less common associations include other congenital cardiac anomalies, such as pulmonary stenosis, atrial septal defect, bicuspid aortic valve, Tetralogy of Fallot, patent ductus arteriosus, coarctation of the aorta, and subaortic stenosis.¹

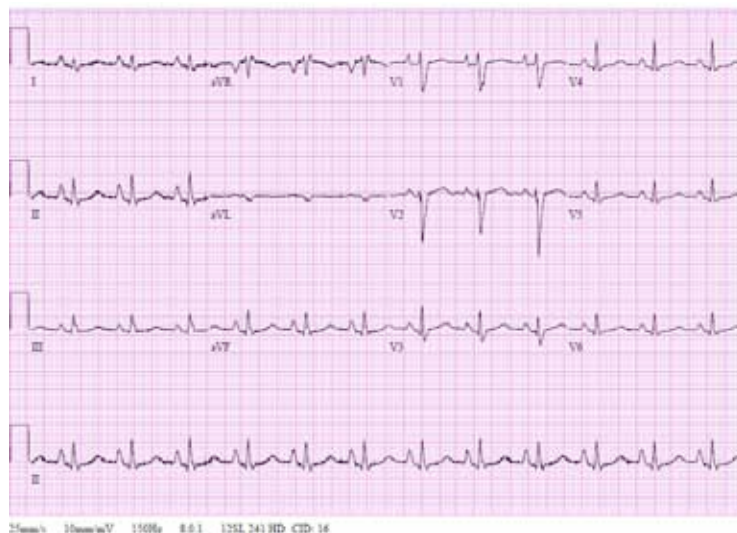


Figure 1. ECG showing sinus rhythm, right atrial enlargement, non-specific intraventricular conduction delay, and mildly prolonged QTc.

TTE is often sufficient for proceeding with surgical planning when the imaging is congruent with the clinical presentation and exam. It is also convenient and safe in more critically ill patients. If TTE is not sufficient, then TEE is likely to be valuable for defining the anatomy, sites of rupture, and valve involvement. Diagnostic cardiac catheterization is now reserved for stable patients in whom coronaries need to be better visualized.^{1,9}

Untreated ruptured SVAs have demonstrated a mean survival period of one to four years in various series; therefore, early surgical intervention is needed. Congestive heart failure is the primary cause of death, but endocarditis may play a role in about 8% of patients. Indications for surgery include a ruptured SVA with left-to-right shunting, even in asymptomatic individuals. Urgent surgical intervention should be undertaken in any patient with signs of congestive heart failure. The indications for intervening in the case of an asymptomatic patient with an unruptured aneurysm are less clear. Increasing aneurysm size, evidence of RV inflow or outflow tract obstruction, coronary compression, new symptoms, or presence of an underlying connective tissue disease should prompt consideration of surgery.^{1,10}

The goal of surgical intervention is to repair the ruptured SVA, excise the aneurysmal tissue, and repair any associated defects without interrupting atrioventricular nodal conduction or causing valvular incompetence. There are no clinical trials indicating that any one surgical technique is superior.³ Overall, surgical outcomes for ruptured SVA and symptomatic unruptured SVA are excellent with low morbidity and mortality in most series. Patients should continue to be monitored for recurrence of aneurysm formation, late aortic or tricuspid valve insufficiency, and residual septal defects.¹⁰

Transcatheter closure of ruptured SVAs with a septal occluder or similar device has been reported. While a non-operative approach may have several potential advantages, the long-term safety of such an

LIVE CASES

JUNE 26 – 28, 2014
FRANKFURT, GERMANY

**CSI 2014 – CONGENITAL,
STRUCTURAL & VALVULAR
INTERVENTIONS**
www.csi-congress.org





Medtronic

Technologies to Manage
Congenital Heart Disease

Every Step of the Way

**Melody® Transcatheter Pulmonary Valve
Ensemble® Transcatheter Valve Delivery System**

Indications: The Melody TPV is indicated for use in a dysfunctional Right Ventricular outflow Tract (RVOT) conduit (≥ 16 mm in diameter when originally implanted) that is either regurgitant (\geq moderate) or stenotic (mean RVOT gradient ≥ 35 mm Hg)

Contraindications: None known.

Warnings/Precautions/Side Effects:

- DO NOT implant in the aortic or mitral position.
- DO NOT use if patient's anatomy precludes introduction of the valve, if the venous anatomy cannot accommodate a 22-Fr size introducer, or if there is significant obstruction of the central veins.
- DO NOT use if there are clinical or biological signs of infection including active endocarditis.
- Assessment of the coronary artery anatomy for the risk of coronary artery compression should be performed in all patients prior to deployment of the TPV.
- To minimize the risk of conduit rupture, do not use a balloon with a diameter greater than 110% of the nominal diameter (original implant size) of the conduit for pre-dilation of the intended site of deployment, or for deployment of the TPV.
- The potential for stent fracture should be considered in all patients who undergo TPV placement. Radiographic assessment of the stent with chest radiography or fluoroscopy should be included in the routine postoperative evaluation of patients who receive a TPV.
- If a stent fracture is detected, continued monitoring of the stent should be performed in conjunction with clinically appropriate hemodynamic assessment. In patients with stent fracture and significant associated RVOT obstruction or regurgitation, reintervention should be considered in accordance with usual clinical practice.

Potential procedural complications that may result from implantation of the Melody device include: rupture of the RVOT conduit, compression of a coronary artery, perforation of a major blood vessel, embolization or migration of the device, perforation of a heart chamber, arrhythmias, allergic reaction to contrast media, cerebrovascular events (TIA, CVA), infection/sepsis, fever, hematoma, radiation-induced erythema, and pain at the catheterization site.

Potential device-related adverse events that may occur following device implantation include: stent fracture resulting in recurrent obstruction, endocarditis, embolization or migration of the device, valvular dysfunction (stenosis or regurgitation), paravalvular leak, valvular thrombosis, pulmonary thromboembolism, and hemolysis.

For additional information, please refer to the Instructions for Use provided with the product or call Medtronic at 1-800-328-2518 and/or consult Medtronic's website at www.medtronic.com.

Humanitarian Device. Authorized by Federal law (USA) for use in patients with a regurgitant or stenotic Right Ventricular Outflow Tract (RVOT) conduit (≥ 16 mm in diameter when originally implanted). The effectiveness of this system for this use has not been demonstrated.

Melody and Ensemble are trademarks of Medtronic, Inc.
UC201303735 EN © Medtronic, Inc. 2013;
All rights reserved.

The Melody® TPV offers children and adults a revolutionary option for managing valve conduit failure without open heart surgery.

Just one more way Medtronic is committed to providing innovative therapies for the lifetime management of patients with congenital heart disease.

Innovating for life.

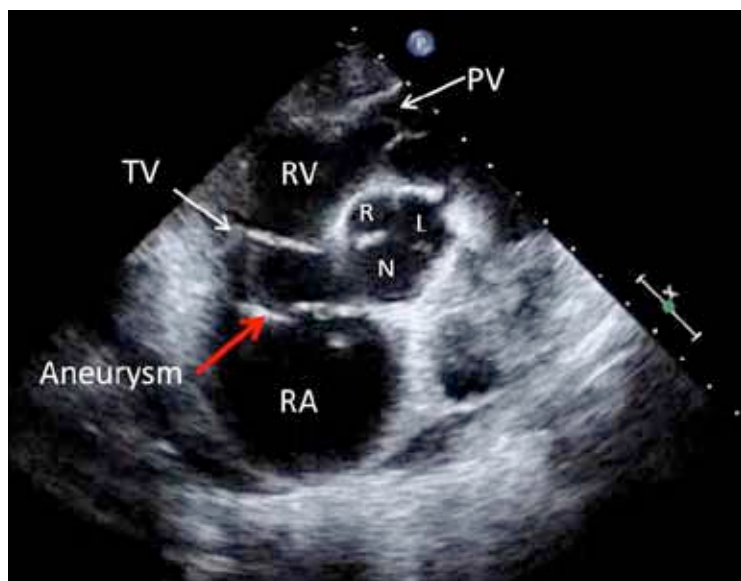


Figure 2a. Transthoracic echocardiogram: 2D parasternal short-axis view at the level of the aortic valve showing an aneurysm originating from the non-coronary sinus of Valsalva and protruding into the right atrium at the level of the tricuspid valve. RA-right atrium, RV-right ventricle, TV-tricuspid valve, PV-pulmonary valve, R, L, & N-right, left, and non-coronary aortic cusps.

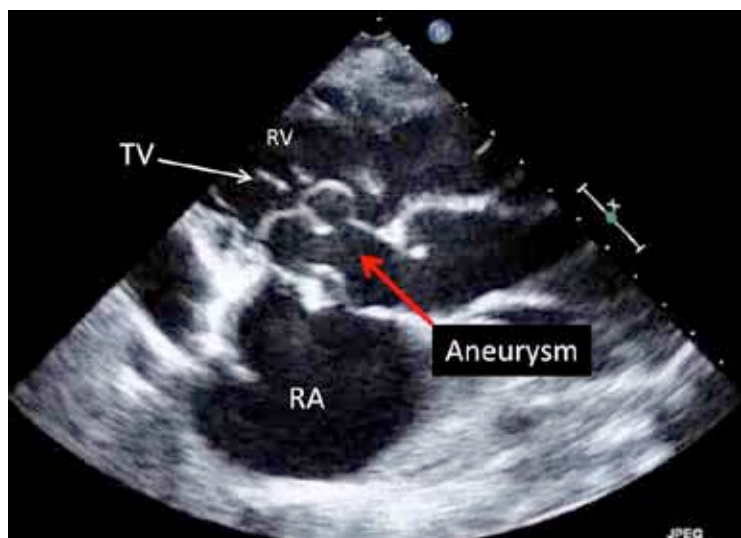


Figure 2b. Transthoracic echocardiogram: 2D modified parasternal long-axis view angled toward the tricuspid valve showing aneurysm of the non-coronary sinus of Valsalva obstructing right ventricular inflow. RA-right atrium, RV-right ventricle, TV-tricuspid valve.

approach is unknown. Surgery remains the gold standard of intervention for ruptured SVAs.^{11,12}

In summary, SVAs are rare but potentially life-threatening defects that are usually congenital in origin. Even large aneurysms may be silent until rupture or obstruction occurs, most likely in the second or third decade of life. A harsh continuous murmur, especially if new and accompanied by signs of congestive heart failure, should trigger concern for ruptured SVA, regardless of whether the patient has a known history of aneurysm. Transthoracic echocardiogram is the diagnostic imaging modality of choice. Surgical repair of ruptured SVAs can typically be undertaken with good long-term outcomes.

Additional cases like the one above will be reviewed at *Congenital Heart Disease in the Adult: An International Symposium*, June 10-13, 2014 in Cincinnati. To register, visit www.cincinnatichildrens.org/achdprogram.

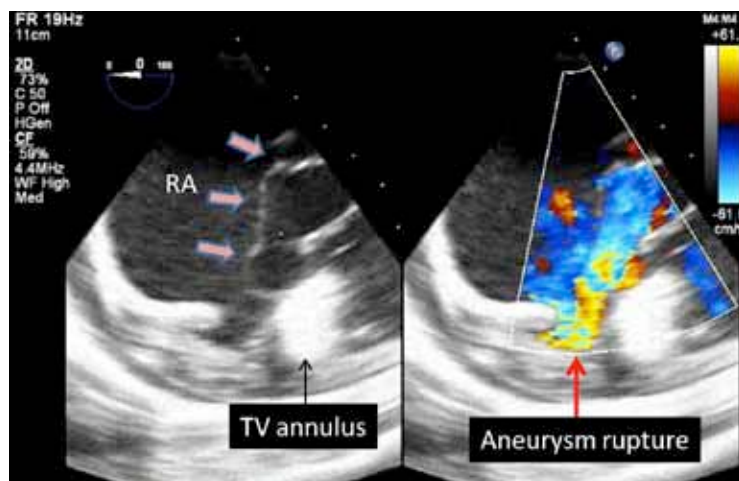


Figure 3a. Transesophageal echocardiogram: color Doppler image showing an enlarged right atrium, aneurysmal tissue superior to the tricuspid valve annulus, and color Doppler indicating high-velocity shunting from the aneurysm toward the right atrial appendage. RA-right atrium, wide arrows indicate non-coronary sinus of Valsalva aneurysm.

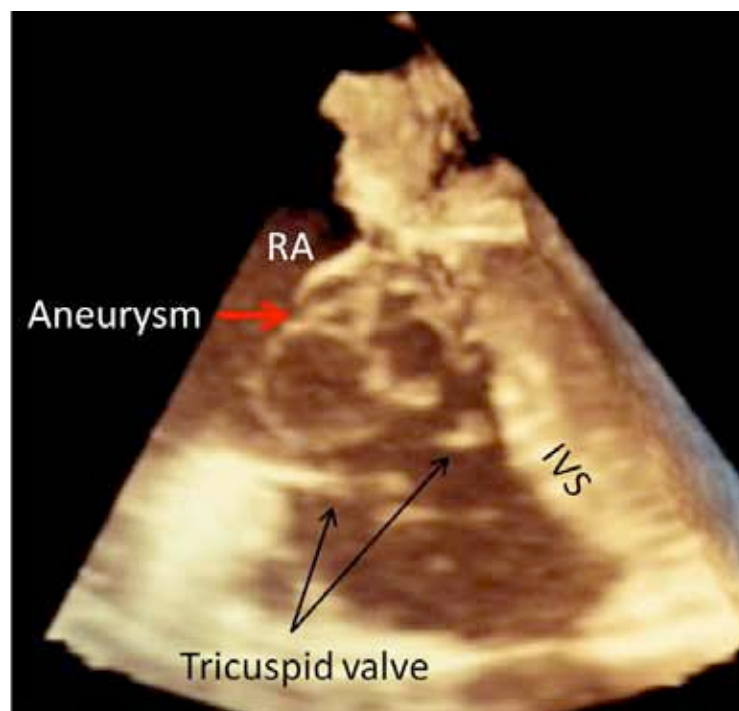


Figure 3b. Transesophageal echocardiogram: 3D image showing aneurysmal aortic sinus tissue extending across most of the right ventricular inlet. RA-right atrium, IVS-interventricular septum.

Principal Author Biographical Sketch

Nicole Brown, MD completed residency in Internal Medicine and Pediatrics at the University of Cincinnati Medical Center and Cincinnati Children's Hospital Medical Center. She then completed a fellowship in Pediatric Cardiology at Children's Hospital of Pittsburgh of UPMC, where she also began her training in the care of adults with congenital heart disease. Dr. Brown is now participating in a two-year advanced fellowship in Adolescent and Adult Congenital Heart Disease at Cincinnati Children's Hospital Medical Center. She is board-certified in Internal Medicine and Pediatrics and board-eligible in Pediatric Cardiology. She also intends to sit for the Adult Congenital Heart Disease boards.

References

1. Moustafa S, Mookadam F, Cooper L, Adam G, Zehr K, Stulak J, Holmes D. Sinus of Valsalva aneurysms--47 years of a single center experience and systematic overview of published reports. *Am J Cardiol.* 2007;99:1159-64.
2. Guo H-W, Sun X-G, Xu J-P, Xiong H, Wang X-Q, Su W-J, Lin Y, Hu S-S. A new and simple classification for the non-coronary sinus of Valsalva aneurysm. *Eur J Cardiothorac Surg.* 2011;40:1047-51.
3. Xin-Jin L, Xuan L, Bo P, Hong-Wei G, Wei W, Shou-Jun L, Sheng-Shou H. Modified Sakakibara classification system for ruptured sinus of Valsalva aneurysm. *J Thorac Cardiovasc Surg.* 2013;146:874-8.
4. Katayama S, Umetani N, Sugiura S, Hisada T. The sinus of Valsalva relieves abnormal stress on aortic valve leaflets by facilitating smooth closure. *J Thorac Cardiovasc Surg.* 2008;136:1528-35, 1535.e1.
5. Charitos EI, Sievers H-H. Anatomy of the aortic root: implications for valve-sparing surgery. *Ann Cardiothorac Surg.* 2013;2:53-6.
6. Edwards JE, Burchell HB. The pathological anatomy of deficiencies between the aortic root and the heart, including aortic sinus aneurysms. *Thorax.* 1957;12:125.
7. Takach TJ, Reul GJ, Duncan JM, Cooley D a, Livesay JJ, Ott D a, Frazier OH. Sinus of Valsalva aneurysm or fistula: management and outcome. *Ann Thorac Surg.* 1999;68:1573-7.
8. Ring WS. Congenital Heart Surgery Nomenclature and Database Project: aortic aneurysm, sinus of Valsalva aneurysm, and aortic dissection. *Ann Thorac Surg.* 2000;69:S147-63.
9. Cheng TO, Yang Y-L, Xie M-X, Wang X-F, Dong N-G, Su W, Lü Q, He L, Lu X-F, Wang J, Li L, Yuan L. Echocardiographic diagnosis of sinus of Valsalva aneurysm: A 17-year (1995-2012) experience of 212 surgically treated patients from one single medical center in China. *Int J Cardiol.* 2014;173:33-39.
10. van Son JA, Danielson GK, Schaff HV, Orszulak TA, Edwards WD, Seward JB.. Long-term outcome of surgical repair of ruptured sinus of Valsalva aneurysm. *Circ.* 1994; 90: 1120-9.
11. Kerkar PG, Lanjewar CP, Mishra N, Nyayadish P, Mammen I. Transcatheter closure of ruptured sinus of Valsalva aneurysm using the Amplatzer duct occluder: immediate results and mid-term follow-up. *Eur Heart J.* 2010;31:2881-7.
12. Miranda D, Peter AA, Osorion J, Ferreira AC. Ruptured aneurysm of the noncoronary sinus of valsalva. *Tex Heart Inst J.* 2005;32:586-8.

CCT



Nicole Brown, MD
Adolescent and Adult Congenital Heart Disease Fellow
Heart Institute, Cincinnati Children's Hospital Medical Center
3333 Burnet Ave.
Cincinnati, OH 45229-3026 USA
Phone: 513-803-2243
Fax: 513-803-0079
nbrown1606@gmail.com

Corresponding Contributor



Gruschen Veldtman, MBChB, FRCP
Director, Inpatient Adult Congenital Heart Disease Services
Heart Institute, Cincinnati Children's Hospital Medical Center
3333 Burnet Ave.
Cincinnati, OH 45229-3026 USA
Phone: 513-803-2243
Fax: 513-803-0079
gruschen.veldtman@cchmc.org



David Luís Simón Morales, MD
The Heart Institute
Chief, Pediatric Cardiothoracic Surgery
Professor of Surgery and of Pediatrics
Clark-Helmsworth Chair of Pediatric Cardiothoracic Surgery
Cincinnati Children's Hospital Medical Center
The University of Cincinnati College of Medicine



Christopher Learn, MD
Assistant Professor, University of Cincinnati Department of Pediatrics and Internal Medicine
Heart Institute, Cincinnati Children's Hospital Medical Center
Cincinnati, OH USA

Letters to the Editor

Congenital Cardiology Today welcomes and encourages Letters to the Editor. If you have comments or topics you would like to address, please send an email to: LTE@CCT.bz, and let us know if you would like your comment published or not.



CARDIOSTIM2014
 EHRA EUROPACE
 June 18-21
 Nice - France

World congress on cardiac electrophysiology

Register from now on!

Advance Program available
 on www.cardiostim.com

Placement of an Implantable Cardioverter Defibrillator in a Pediatric Patient with Heparin-Induced Thrombocytopenia with Thrombosis, Bridging with Argatroban

By Joshua A. Daily, MD; Cristina Tarango, MD; Richard J. Czosek, MD; Jeffrey B. Anderson, MD; David S. Spar, MD

Case Report

A 17-year-old male (weight: 69 kg; body surface area: 1.27 m²) presented following cardiac arrest secondary to ventricular fibrillation. He was witnessed to collapse after running up and down a ramp. He was unresponsive and pulseless, and cardiopulmonary resuscitation was initiated. An automatic external defibrillator demonstrated that his rhythm was ventricular fibrillation. He received an initial shock of 200 Joules (J) which briefly converted him to sinus rhythm, but he reverted to ventricular fibrillation. He received a second shock of 300 J with conversion to sinus rhythm.

The patient developed significant respiratory failure and Acute Respiratory Distress Syndrome, and on hospital Day 5 was put on veno-venous Extracorporeal Membrane Oxygenation (ECMO) for 9 days. While on ECMO, he developed Heparin-Induced Thrombocytopenia with Thrombosis (HITT) secondary to the unfractionated heparin infusion with thrombocytopenia and thrombosis of his left cephalic and right basilic veins. The Heparin-PF4 antibody and 14C-serotonin release assay were positive consistent with a diagnosis of heparin-induced thrombocytopenia (HIT). The heparin was discontinued and argatroban, a direct thrombin inhibitor, was initiated. The thrombophilia work up was negative including normal protein C, normal protein S, normal antithrombin, negative Factor V Leiden, and negative prothrombin G20210A mutation. Full body ultrasound demonstrated no additional sources of thrombus.

He underwent an extensive cardiac work-up to determine the etiology of his ventricular fibrillation arrest. Cardiac CT and cardiac MRI were normal. His initial echocardiogram showed mildly depressed biventricular function with eventual normalization of function on subsequent echocardiograms.

We proceeded with diagnostic electrophysiology study and cardiac catheterization. Unfortunately, the post-ECMO course was complicated by chronic fevers which delayed these procedures. In the interim, he was transitioned to warfarin to both minimize the infection risk associated with a central line and to enable him to be discharged from the Intensive Care Unit (ICU); our institutional policy requires that patients on argatroban remain in the ICU. When the fevers resolved, we performed an electrophysiology study with ventricular stimulation protocol along with a hemodynamic cardiac catheterization with an endomyocardial biopsy all of which were normal. The warfarin was discontinued 5 days prior to the cardiac catheterization secondary to bleeding risk during arterial access so the patient was restarted on argatroban as a therapeutic bridge through the study.

We placed an implantable cardioverter defibrillator for secondary prevention approximately 30 days from the diagnosis of HITT. Since the patient had just transitioned back to argatroban for cardiac catheterization and had previously taken three weeks to become therapeutic on warfarin, the decision was made to use an argatroban bridge protocol for the ICD placement developed in conjunction with the hematology service (Table 1). The argatroban was initially dosed at 4 mcg/kg/min via continuous IV infusion. Argatroban was then stopped 6 hours prior to surgery. An international normalized ratio (INR), activated partial thromboplastin time (aPTT) (drawn 2 hours prior to surgery) and

activated clotting time (ACT) (drawn 1 hour prior to surgery) were verified and normalized prior to proceeding. The patient then underwent placement of a transvenous single chamber ICD in the left suprapectoral region without complication. Argatroban was then restarted at half the original dose, 2 mcg/kg/min continuous infusion 12 hours post-ICD placement. Warfarin was restarted 24 hours post-ICD placement after ensuring that there was no post-surgical hematoma formation. While the patient was on argatroban, PT, aPTT, INR, and diluted thrombin time were drawn every 6 hours and the infusion was titrated as needed for goal aPTTs 1.5-3 times baseline. Argatroban was continued until the warfarin was in a therapeutic range (goal chromogenic X activity of 17%-25%, INR was not used since it is also prolonged by Argatroban).

Clinical Follow-Up Evaluation

The patient did not have any bleeding complications during his ICD placement, his remaining hospitalization, or 6-months post-ICD implant. He was discharged after warfarin reached therapeutic levels. Subsequent ultrasounds demonstrated complete resolution of all thrombi and the warfarin was discontinued after 4 months. The etiology of his cardiac arrest continues to remain unclear.

Discussion

Pediatric patients requiring anticoagulation that necessitate placement of a Cardiac Implantable Electronic Device (CIED) represent a high-risk group. The primary complication of ICD placement is pocket hematoma formation which has an incidence in adults between 3.5-5% for those on warfarin or no anticoagulation^{1,2} and greater than 20% for those bridged with Heparin.^{3,4} For children, the risk of bleeding complication based upon type of anticoagulation is not well-defined, but in a review of a multicenter registry the overall incidence of hematoma was found to be 1.8%.⁵ When deciding on an anticoagulation plan for ICD placement, we had to balance the risk of hematoma formation with the risk of

Table 1. Argatroban Protocol

Time Relative to Surgery	Action
	Argatroban 4 mcg/kg/min.
6 hours before	Discontinue Argatroban.
2 hours before	Verify that INR and aPTT have normalized.
1 hour before	Verify that ACT has normalized.
12 hours after	Restart Argatroban 2 mcg/kg/min and titrate for goal aPTT 1.5-3 times baseline (drawn every 6 hours).
24 hours after	Restart warfarin.
Indeterminate	Continue argatroban until chromogenic X activity is 17-25%.

INR international normalized ratio
aPTT activated partial thromboplastin time
ACT activated clotting time

JOIN US FOR THE 2014 ANNUAL
INTERNATIONAL SYMPOSIUM FOR
ADULT CONGENITAL HEART DISEASE.

Why?

BECAUSE YOUR PATIENTS
TURN TO YOU AS THE
**EXPERT IN CONGENITAL
HEART DISEASE.**

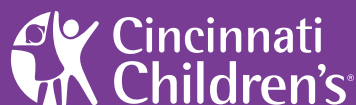
EARLY REGISTRATION ENDS SOON!

JUNE 10-13, 2014

CINCINNATI, OHIO

**SPACE IS LIMITED,
PLEASE REGISTER TODAY
BY VISITING**

cincinnatichildrens.org/achdprogram



Heart Institute

Discover the latest in adult congenital heart disease (ACHD) care and treatment during "Congenital Heart Disease in the Adult: An International Symposium." Continuing medical education credits will be awarded.

Pediatric and adult cardiovascular specialists and nurse practitioners worldwide are invited to learn about:

- Perioperative & critical care management
- Cardiac catheterization, including congenital & other structural interventions
- Exercise physiology, pathology & rehabilitation
- Electrophysiology: consensus guidelines for clinical management
- Pregnancy & heart disease
- Transitional medicine in congenital heart disease: concepts and practices

Presented by the Adolescent and Adult Congenital Heart Disease Program, part of the Heart Institute at Cincinnati Children's Hospital Medical Center.

Cincinnati Children's Hospital Medical Center is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians. This activity has been approved for AMA PRA Category 1 Credit™.

Contact hours will be awarded to nurses who attend the entire program and complete an evaluation tool. Cincinnati Children's Hospital (OH-046, 9/1/2015) is an approved provider of continuing nursing education by the Ohio Nurses Association (OBN-001-91), an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation.

thromboembolic complications due to HIT. In adults with HIT, the incidence of thromboembolic complications is 38-76%⁶, often within 30 days of diagnosis, but it is significantly lower in those treated with Argatroban.⁷ Pediatric data is much more limited, but overall the risk in children with and without treatment seems similar to adults.⁸ In contrast to adults, though, HIT associated thrombosis in children is more often venous than arterial.⁹

Our review revealed no published experiences of anticoagulation in patients with HIT requiring ICD placement. In both children and adults with HIT and HITT, Argatroban has been used successfully to maintain patency in the cardiopulmonary bypass apparatus during cardiac surgery¹⁰ and to prevent thrombosis during cardiac catheterization.¹¹ In addition, the safety, efficacy, and pharmacokinetics of Argatroban in children have been previously demonstrated,⁸ and our institution's practice has been to use Argatroban in patients with HIT.

Our patient was receiving continuous anticoagulation with Argatroban at the time of his ICD placement. Rather than wait for him to become therapeutic on warfarin and significantly delay ICD implantation (previously therapeutic levels were reached in 3 weeks), the team decided to proceed with an Argatroban bridge. Given the high risk of hematoma formation in combination with the data suggesting that the risk for thrombus formation of short-term interruption of anticoagulation is very low, even in high thromboembolic risk patients¹², the decision was made to hold his Argatroban for the case. The rate of pocket hematoma formation in patients bridged with heparin has been demonstrated to be the same whether heparin was restarted 6 hours or 24 hours after surgery, therefore, we chose to restart the Argatroban 12 hours after surgery.⁴

This report illustrates the unique challenges in treating patients at high thromboembolic risk who require a CIED. Though the patient tolerated the argatroban bridging protocol, this being a single reported case is a limitation of the protocol. This relatively uncommon occurrence of HIT in a patient requiring urgent device implantation will likely preclude extensive study on this population, so we chose to present this case and our argatroban protocol for other institutions that may face a similar clinical dilemma.

Disclosures/ Conflicts of Interest: none

References

1. Wiegand UK, LeJeune D, Boguschewski F, et al. Pocket hematoma after pacemaker or implantable cardioverter defibrillator surgery: influence of patient morbidity, operation strategy, and perioperative antiplatelet/anticoagulation therapy. *Chest* 2004;126:1177-86.
2. Poole JE, Gleva MJ, Mela T, et al. Complication rates associated with pacemaker or implantable cardioverter-defibrillator generator replacements and upgrade procedures: results from the REPLACE registry. *Circulation* 2010;122:1553-61.
3. Kutinsky IB, Jarandilla R, Jewett M, Haines DE. Risk of hematoma complications after device implant in the clopidogrel era. *Circulation Arrhythmia and electrophysiology* 2010;3:312-8.
4. Michaud GF, Pelosi F, Jr., Noble MD, Knight BP, Morady F, Strickberger SA. A randomized trial comparing heparin initiation 6 h or 24 h after pacemaker or defibrillator implantation. *Journal of the American College of Cardiology* 2000;35:1915-8.
5. Berul CI, Van Hare GF, Kertesz NJ, et al. Results of a multicenter retrospective implantable cardioverter-defibrillator registry of



Congenital Cardiology Today Can Help You Recruit:

- Pediatric Cardiologists
- pediatric Interventional Cardiologist
- Adult Cardiologist focused on CHD
- Congenital/Structural Heart Surgeons
- Echocardiographers, EPs
- Pediatric Transplant Cardiologist

Reach over 6,000 BC/BE Cardiologists focused on CHD worldwide:

- Recruitment ads include color!
- Issues's email blast will include your recruitment ad!
- We can create the advertisement for you at no extra charge!

Contact:

Tony Carlson

+1.301.279.2005 or tcarlsonmd@gmail.com



BASIC AND ADVANCED FETAL CARDIOLOGY SYMPOSIUM & WORKSHOP 2014

1ST ANNUAL MEETING

SAVE THE DATE JUNE 5-6, 2014; CHICAGO IL USA

<http://fetalcardiacsymposium.com>

- pediatric and congenital heart disease patients. *Journal of the American College of Cardiology* 2008;51:1685-91.
6. Hirsh J, Heddle N, Kelton JG. Treatment of heparin-induced thrombocytopenia: a critical review. *Archives of internal medicine* 2004;164:361-9.
 7. Lewis BE, Wallis DE, Berkowitz SD, et al. Argatroban anticoagulant therapy in patients with heparin-induced thrombocytopenia. *Circulation* 2001;103:1838-43.
 8. Young G, Boshkov LK, Sullivan JE, et al. Argatroban therapy in pediatric patients requiring nonheparin anticoagulation: an open-label, safety, efficacy, and pharmacokinetic study. *Pediatric blood & cancer* 2011;56:1103-9.
 9. Klenner AF, Lubenow N, Raschke R, Greinacher A. Heparin-induced thrombocytopenia in children: 12 new cases and review of the literature. *THROMBOSIS AND HAEMOSTASIS-STUTTGART* 2004;91:719-24.
 10. Martin ME, Kloecker GH, Laber DA. Argatroban for anticoagulation during cardiac surgery. *Eur J Haematol* 2007;78:161-6.
 11. Lewis BE, Matthai WH, Jr., Cohen M, Moses JW, Hursting MJ, Leya F. Argatroban anticoagulation during percutaneous coronary intervention in patients with heparin-induced thrombocytopenia. *Catheterization and cardiovascular interventions : official journal of the Society for Cardiac Angiography & Interventions* 2002;57:177-84.
 12. Phan TG, Koh M, Wijdicks EF. Safety of discontinuation of anticoagulation in patients with intracranial hemorrhage at high thromboembolic risk. *Archives of neurology* 2000;57:1710-3.

CCT

Corresponding Contributor



Joshua A. Daily, MD
Cincinnati Children's Hospital Medical Center
Division of Pediatric Cardiology
The Heart Institute
3333 Burnet Avenue MLC 2003
Cincinnati, OH 45229, USA
Ph: (513)636-4888
Fax: 513-636-7996
joshua.daily@cchmc.org



Cristina Tarango, MD
Cincinnati Children's Hospital Medical Center
Division of Hematology
Cancer and Blood Diseases Institute
Cincinnati, OH, USA



Richard J. Czosek, MD
Cincinnati Children's Hospital Medical Center
Division of Pediatric Cardiology
The Heart Institute
3333 Burnet Ave. MLC
Cincinnati, OH 45229, USA



Jeffrey B. Anderson, MD, MPH
Cincinnati Children's Hospital Medical Center
Division of Pediatric Cardiology
The Heart Institute
3333 Burnet Ave. MLC
Cincinnati, OH 45229, USA



David S. Spar, MD
Cincinnati Children's Hospital Medical Center
Division of Pediatric Cardiology
The Heart Institute
3333 Burnet Ave. MLC
Cincinnati, OH 45229, USA

CONGENITAL CARDIOLOGY TODAY

CALL FOR CASES AND OTHER ORIGINAL ARTICLES

Do you have interesting research results, observations, human interest stories, reports of meetings, etc. to share?

Submit your manuscript to:
RichardK@CCT.bz

- Title page should contain a brief title and full names of all authors, their professional degrees, and their institutional affiliations. The principal author should be identified as the first author. Contact information for the principal author including phone number, fax number, email address, and mailing address should be included.
- Optionally, a picture of the author(s) may be submitted.
- No abstract should be submitted.
- The main text of the article should be written in informal style using correct English. The final manuscript may be between 400-4,000 words, and contain pictures, graphs, charts and tables. Accepted manuscripts will be published within 1-3 months of receipt. Abbreviations which are commonplace in pediatric cardiology or in the lay literature may be used.
- Comprehensive references are not required. We recommend that you provide only the most important and relevant references using the standard format.
- Figures should be submitted separately as individual separate electronic files. Numbered figure captions should be included in the main Word file after the references. Captions should be brief.
- Only articles that have not been published previously will be considered for publication.
- Published articles become the property of the Congenital Cardiology Today and may not be published, copied or reproduced elsewhere without permission from Congenital Cardiology Today

CONGENITAL CARDIOLOGY TODAY CALL FOR CASES AND OTHER ORIGINAL ARTICLES

Do you have interesting research results, observations, human interest stories, reports of meetings, etc. to share?

Submit your manuscript to:
RichardK@CCT.bz

Does Renal Denervation Have a Future? TRENDS Conference Gives a Resounding “Yes”

By Sameer Gafoor, MD

The recent *TRENDS* conference in Frankfurt discussed at length the current status and future of renal denervation. Device-Basedand Systemic Interventions was the focus of the recent *TRENDS* conference held in Frankfurt, Germany, to discuss at length the current status and future of renal denervation. Recent trial data has called the future of renal denervation into question, and this was discussed extensively.

Friday, February 7, 2014

This started with an Overview of Hypertension (Roland Schmieder) and an Understanding of Measurement Modalities for Blood Pressure (Guido Grassi), an overview of the the Guidelines (Orfeas Liangos), and an overview of What You Need to Do to Test for Secondary Hypertension (Stefan Bertog), Optimize Medical Management (Orfeas Liangos) and Patient Selection to Implement a Renal Denervation Practice (Oliver Vonend, Thomas Schmitz).

After these presentations, it was time to take a step back to discuss the anatomy and physiology by such renowned scholars as Dagmara Hering, Ariel Sverdlik, and Paul Sobotka. We took a look at: Renal Denervation and Pulse Wave Velocity (Kai Mortenson), and Clinical Endpoints (Robert Melder).

A special “first-in-man” experience of a new device with impressive results was presented by Dr. van der Heijden, proving how there was still much hope and promise for the future.

After lunch, it was time to take a “deep dive” into the different companies and their current updates: Simplicity (Marc Sapoval and Justin Davies), Vessix (Gerard Goh), Enlighthn (Vasiolos Papademetriou), Covidien (Stefan Bertog), and Paradise Recor Medical (Joost Daemen).

Dr. Darrel Francis came and stole the show with simple explanations as to why Simplicity 3 turned out the way it did. He explained in-depth why the press releases were ill-worded and badly interpreted. This was “irrational exuberance” followed by “unjustified despondency,” and stated that now is the time for an exciting future-based on science. He said all this was due to: “Big Day” bias, “Check Once More” bias, and “I’ll Take Them Now” bias that had harmed this trial. There were solutions to all of these issues that future trials would have to pay attention to if they were to have any hope of success.

The other effects of renal denervation were the focus of the next presentations. These

“Recent trial data has called the future of renal denervation into question, and this was discussed extensively.”

presentations included: This included: “Effects on Arrhythmias” (Jennifer Franke), “Insulin Resistance” (Dagmara Hering), “Chronic Kidney Disease” (Roland Schmieder), “Sleep Apnea” (Aleksander Prejbisz), “Exercise Tolerance” (Mathias-Christoph Brandt), “Renovascular Hypertension” (Yvonne Bausback), and “Psychometric Aspects/Quality of Life” (Dagmara Hering).

Once we reviewed all of the above, it was time for a step-by-step approach to the different devices. This started with a lecture on Angiography (Mark Wholey), followed by Instructions for Symplicity Flex and Covidien One Shot (Martin Bergmann), Vessix (Gerard Goh), Enlighthn (Vasiolos Papademetriou), and Recor Medical (Joost Daemen). This was followed by a talk on Invasive Imaging Results after the Four Different Devices (Joost Daemen) and a talk on Optimal Follow-up (Peter de Leeuw).

Saturday, February 8, 2014

Saturday started with an exciting mix of talks and cases together.

The talks went beyond the earlier presentations with a discussion of the new fields of baroreceptor stimulation, pulmonary artery denervation, new techniques for renal denervation, and more.

This started with an overview of the exciting new field of baroreceptor activation and other new systemic interventions. “What is Baroreceptor Activation” was covered by Joern Schmitt, followed by Barostim in “Renal Denervation Nonresponders” (Hannes Reuter), “Chronic Kidney Disease Patients” (Michael Koziolok), and “Heart Failure” (Rolf Wachter).

The kidney nerves are not the only nerves that play a major role in hypertension. Paul Sobotka discussed “Carotid Body Ablation,” followed by the “Promise and Current Status of Pulmonary Artery Denervation for Pulmonary Hypertension” (Alexander Rothman and Yao-Jun Zhang).

The subject of new techniques for renal denervation was broken into two parts because

of the wide plethora of devices available. Overview presentations included: “Advantages and Disadvantages of Radiofrequency” (Justin Davies), “Ultrasound” (Martin Bergmann), “Histopathology after Ultrasound” (Joost Daemen). Other techniques included: “Cardiosonic” (Michael Jonas), “Instant Renal Denervation with TIVUS” (Michael Jonas), “Terumo Iberis Renal Denervation” (Benjamin Honton), “Cordis Thermocool” (Martin Bergmann), “Bullfrog Catheter and Radiation Therapy” (Jan Kulow), “Medtronic Spyral” (Erwin Blessing), and “Local Anesthesia” (Simon Lam).

The second part included: “Externally Focused Ultrasound” (Omar Dawood), “Denervx Cooled Microwave Technology” (Stefan Bertog), “Perivascular Chemical Renal Denervation” (Tim Fischell), “Northwind Medical” (Mark Wholey), “Chemical Renal Denervation by Vicristine” (Christodoulos Stefanadis) and “VERVE Medical with an Invasive Non-vascular Approach” (Richard Heuser).

Other ‘systemic interventions’ included: “Vagus Nerve Stimulation on Arterial Hypertension” (Sekib Sokolovic), “ROX Medical AV Anastomosis Therapy” (Paul Sobotka) and “The Future of Neurohumoral Modulation.”

Cases, performed by Drs. Horst Sievert, Laura Vaskelyte, and Sameer Gafoor included: live detailed step-by-step discussion of the Recor Medical Paradise, Medtronic Symplicity, Boston Vessix, St. Jude Enlighthn, and Terumo Iberis devices. Live invasive imaging was performed to help with decision management and discussion among the participants was extensive.

This amazing *TRENDS* conference happens yearly in Frankfurt and will happen again in February 2015. We look forward to more exciting developments and discussion and invite you to be part of this conference as we discuss and grow in this exciting field.

For more information visit: www.csi-congress.org/trend-workshop.php?go=0. Selected lectures will be found online as well.

CCT

Sameer Gafoor, MD
Cardiovascular Center Frankfurt
Frankfurt, Germany

s.gafoor@cvcfrankfurt.de

Medical News, Products & Information

Winthrop University Hospital Selects Digisonics Cardiovascular Information System as Enterprise Solution

Winthrop University Hospital in Mineola, NY has selected the Digisonics Cardiovascular Information System (CVIS) as their enterprise solution for echo (adult, pediatric and fetal), cath (adult and pediatric), nuclear, and cardiac MR. The Digisonics CVIS will be implemented at three facilities across their enterprise: Winthrop University Hospital, Winthrop Cardiology Associates and Island Cardiac Specialists. The Digisonics cardiovascular information system provides Winthrop's clinicians with a comprehensive clinical database, high-powered PACS and structured cardiology reporting.

Winthrop University Hospital's cardiologists will also utilize Digisonics' newest application, WebView 4.0, which will provide them with the convenience of anywhere, anytime access to their patient images and reports. This application provides users with zero footprint review from any HTML5 capable browser or device. There is no need for ActiveX or other plug-ins and no patient health information is retained on the viewing device.

Seamless integration of the Digisonics Cardiovascular Information System with Philips QLAB, TomTec, Medis and INVIA will provide Winthrop University Hospital with advanced image quantitation and analysis for their cardiovascular studies. A single click launches the 3rd-party analysis software directly from the Digisonics cardiovascular reporting workstation and the post-processed images are saved back into the Digisonics CVIS for review. Winthrop's cardiovascular information system will be enhanced with the Digisonics Search Package, a comprehensive, user-configurable search engine. This powerful tool enables users to quickly set up search criteria to extract clinical information from their cardiovascular information system database for research purposes, compile statistics required for accreditation and generate management reports to target areas for improvement.

HL7 interfaces between Winthrop's Siemens Envision and GE Centricity systems and the Digisonics cardiovascular information system will create a fully electronic and efficient workflow. Winthrop University Hospital will implement Digisonics DigiConnect, a new application enabling the facility to launch any 3rd party systems (such as EMR/HIS, PACS or ECG management software) directly from the Digisonics cardiovascular information system workstation. Users benefit from the convenience of a single sign-on as user credentials and patient information are passed directly to the 3rd party systems. The result is a seamless cardiovascular reporting workflow with improved efficiency, accuracy and turnaround times for all facilities within Winthrop University Hospital's enterprise.

For further information, visit: www.digisonics.com.

Adult Congenital Heart Association Names New Executive Director - Seasoned Leader Brings More Than Three Decades of Experience to New Role

The Adult Congenital Heart Association (ACHA), a national nonprofit organization dedicated to improving and extending the lives of adults

with congenital heart defects (CHD), announced the appointment of Glenn R. Tringali as its new National Executive Director.

Tringali comes to ACHA with more than three decades of extensive experience working with small, mid-sized and large national and international healthcare organizations. He succeeds Gail Ober, who led ACHA as Interim Executive Director since October 2013.

"I am honored to join the Adult Congenital Heart Association as National Executive Director," said Tringali. "With more than a million adults in the United States living with congenital heart defects, there is a clear and compelling need for greater awareness and support of ACHA's critically important mission and programs. I am truly excited to have this opportunity to work in partnered leadership with our Board of Directors, Medical Advisory Board, staff and volunteers in making a meaningful difference in the lives of those affected by CHD."

ACHA Board Chair John Fernie commented, "The majority of those born with congenital heart defects are unaware of the need for continued care. An important aspect of our mission is to reach out, support and educate all of those with CHD. Glenn's demonstrated success in expanding outreach, member support and funding brings strong leadership to these critical efforts."

Tringali began his nonprofit career with the March of Dimes, where he served in Executive and Regional Director positions over the course of 10 years. He then honed his skills in development, volunteer coordination and fundraising events in various positions at organizations such as The National Conference for Community and Justice, the Cancer Research Institute and Juvenile Diabetes International. He also served as a Senior Manager Nonprofit Consultant for KPMG Peat Marwick.

He later became the Chief Executive Officer of the National Alliance for Autism Research (NAAR), during which time he managed the expansion of research, development, public relations and government advocacy programs. Under his leadership, NAAR grew from a \$4.2 million organization with a few thousand supporters to a \$13.1 million organization with tens of thousands of supporters throughout the United States, Canada and the United Kingdom. Tringali also grew NAAR's research program commitments by \$6 million. This exemplary success led to the successful merger of NAAR with Autism Speaks, where he assumed the role of Executive Vice President.

Since 2010, Tringali served as the CEO of the Cerebral Palsy International Research Foundation, working to advance the organization's research programs, community awareness and operational funding needs.

"Glenn has a fantastic track record of proven success and his breadth of experience is remarkable. This is an exciting new chapter for ACHA," added Dr. Curt Daniels, Chair of ACHA's Medical Advisory Board and Professor of Internal Medicine and Pediatrics in the Division of Cardiology at The Ohio State University and Division of Pediatric Cardiology at Nationwide Children's Hospital.

Tringali received his undergraduate degree from Rutgers University. He currently resides in central New Jersey.



10th Global Forum on Humanitarian Medicine
in Cardiology and Cardiac Surgery

Memphis, TN, USA

October 17th - 19th, 2014

HOSTED BY



International Children's
Heart Foundation
Where Hope Comes to Life



Register Today!
Early Bird Rates End May 15th
Abstracts Accepted through July 7th
Exhibit Space Available!
www.GFHM.ch
www.Babyheart.org

Study Evaluates Role of Infliximab in Treating Kawasaki Disease Antibody Treatment Helps Children with Dangerous Heart Disorder

Newswise — Kawasaki Disease (KD) is a severe childhood disease that many parents, even some doctors, mistake for an inconsequential viral infection. If not diagnosed or treated in time, it can lead to irreversible heart damage.

Signs of KD include prolonged fever associated with rash, red eyes, mouth, lips and tongue, and swollen hands and feet with peeling skin. The disease causes damage to the coronary arteries in a quarter of untreated children and may lead to serious heart problems in early adulthood. There is no diagnostic test for Kawasaki disease, and current treatment fails to prevent coronary artery damage in at least one in 10 to 20 children and death in one in 1,000 children.

Between 10 and 20 percent of patients with KD experience fever relapse following the standard therapy with a single infusion of intravenous immunoglobulin (IVIG) and aspirin. It is known that IVIG resistance increases the risk of heart damage, most commonly a ballooning of the coronary arteries called aneurysms. These children require additional therapy to interrupt the inflammatory process that can lead to damage of the coronary arteries.

A study led by physicians at the University of California, San Diego School of Medicine and Rady Children's Hospital-San Diego looked at intensification of initial therapy for all children with KD in order to prevent IVIG-resistance and associated coronary artery abnormalities by assessing the addition of the medication infliximab to current standard therapy. The results of their study will be published in the February 24, 2014 online issue of the medical journal *Lancet*.

Tumor necrosis factor α (TNF α) is a molecule made by the body that plays a role in the development of inflammation in KD; therefore, treatment with a TNF α antagonist is a logical therapeutic intervention, according to the researchers. Early experience with infliximab – a monoclonal antibody that binds TNF α – showed promising results. A Phase 1 trial in children with KD and persistent fever following standard therapy found no infusion reactions or serious adverse events, and subsequent studies suggested that infliximab led to faster resolution of fever and fewer days of hospitalization than a second IVIG infusion.

The UC San Diego researchers conducted a trial of 196 subjects at two centers – Rady Children's Hospital-San Diego, a research affiliate of UC San Diego School of Medicine, and Nationwide Children's Hospital in Columbus, Ohio – to assess whether infliximab could reduce IVIG treatment resistance.

"While the addition of infliximab to primary treatment in acute KD did not reduce treatment resistance, it was safe and well-tolerated, achieved a greater reduction in the size of the left coronary artery, and reduced the number of days of fever and laboratory markers of inflammation," said the study's first author, Adriana H. Tremoulet, MD, of the UC San Diego Department of Pediatrics and the UC San Diego/Rady Children's Hospital-San Diego Kawasaki Disease Research Center. "We conclude that use of infliximab is safe in infants and children and that early treatment could help children with Kawasaki Disease with high levels of inflammation or early signs of coronary artery damage."

MEDICAL MEETING FOCUS

PICS ~ AICS 2014

Jun. 7-10, 2014; Marriott Chicago Downtown, Chicago, IL USA
www.picsymposium.com

PICS~AICS includes a one day program on June 7th - *Cardiac Imaging for Structural Heart Disease* with Course Directors: Roberto M. Lang, MD, FASE, FACC, FESC, FAHA, FRCP; Ziyad M Hijazi, MD, MPH, FACC, FSCAI along with an International faculty

PICS Overview: Taped case sessions, along with the hands-on demonstrations. The meeting continues to support open dialogue as well as visual learning, along with updates from all the relevant trials and societies, with opportunities for networking and sharing global clinical and research experience. The atmosphere is professional, but relaxed. "Tips and Tricks" session with hands-on demonstrations from international experts on how to work with wires, stents, devices and sheaths as well as valves. This will be followed by an interactive taped case session with four cases for discussion. The oral abstract sessions are not competing with one another so that the true scientific endeavors of our colleagues are given the platform they deserve.

PICS Course Directors: Ziyad M. Hijazi, MD, MPH; John P. Cheatham, MD; Carlos Pedra, MD; Thomas K. Jones, MD

Course Co-Directors: Damien Kenny, MD; Giacomo Pongiglione, MD; Clifford J. Kavinsky, MD, PhD; Ralf Holzer, MD; John D. Carroll, MD

Director Emeritus: William E. Hellenbrand, MD

PICS International Faculty: Teiji Akagi, MD; BG Alekian, MD; Mansour AlJufan, MD; Wail Alkashkari, MD; Zahid Amin, MD; John Bass, MD; Lee Benson, MD; Felix Berger, MD; Jacek Bialkowski, MD; Qi-Ling Cao, MD; Roberto Cubeddu, MD; Bharat Dalvi, MD; JV de Giovanni, MD; Michael de Moor, MD; Karim Diab, MD; Makram Ebeid, MD; Maiy El Sayed, MD; Peter Ewert, MD; Horacio Faella, MD; Thomas Fagan, MD; Ted Feldman, MD; Craig Fleishman, MD; Simone Fontes-Pedra, MD; Thomas Forbes, MD; Mark Galantowicz, MD; Marc Gewillig, MD; Matt Gillespie, MD; Bryan Goldstein, MD; Brent Gordon, MD; Eric Horlick, MD; Reda Ibrahim, MD; Michel Ilbawi, MD; Frank Ing, MD; Alex Javois, MD; Saibal Kar, MD; Terry King, MD; Jackie Kreutzer, MD; Yat Yin Lam, MD; Larry Latson, MD; Martin Leon, MD; Dan Levi, MD; Tarek Momenah, MD; John Moore MD; Charles Mullins, MD; Shakeel Qureshi, MD; John Rhodes, MD; Richard Ringel, MD; Masood Sadiq, MD; Dietmar Schranz, MD; Girish Shirali, MD; Horst Sievert, MD; Hideshi Tomita, MD; Michael Tynan, MD; Julie Vincent, MD; Kevin Walsh, MD; Neil Wilson, MD; Carlos Zabal, MD; Evan Zahn, MD

Cardiac Imaging Faculty: Steven Goldstein, MD; Rebecca Hahn, MD; Bijoy Khandheri, MD; Itzhak Kronzon, MD; Amit Patel, MD; Ernesto Salcedo, MD; Vinod Thourani, MD; John Carroll, MD; John Hibblen, MD

Some of the Topics Covered Include: Interventional Platform – Tips, Tricks and Application to CHD; Interventional Radiology; Interventional Neurosurgery; Use of CTO Wires in CHD; Hands-on-Demonstrations: Sheaths, Transseptal, Devices, Stents, Valves; My Nightmare Case in the Cath Lab; Complications of Congenital Cardiac Catheterization, and more....

Breakout Sessions Include: Complex and New Structural Interventions; Nursing and Associated Professionals; Spanish Session; The Left Atrial Appendage State-of-the-Art; PICES; (Young Interventionalist Group); Mitral Valve Interventions; TAVR

Lives Cases from: London, Los Angeles, Denver, Chicago, Detroit, Columbus, Córdoba and Riyadh

A REVIEW COURSE PRESENTED BY
American Academy of Pediatrics Section on Cardiology & Cardiac Surgery
— in collaboration with —
Society of Pediatric Cardiology Training Program Directors

Discounts for early registration and AAP members available.

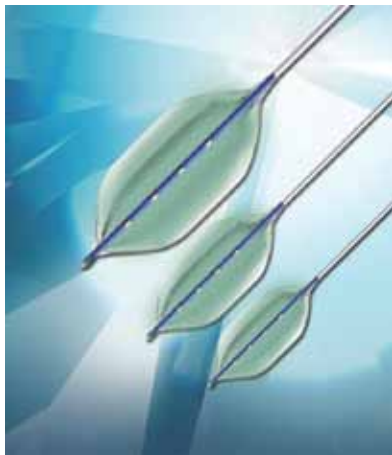
www.aap.org/Pediatric-Cardiology-2014

**SPECIALTY
REVIEW IN** **Pediatric
Cardiology**
SEPTEMBER 15-19, 2014 • CHICAGO

Additional contributors to the study include: principal investigator Jane C. Burns, MD, Susan Jimenez-Fernandez, MD, John T. Kanegaye, MD, and Beth Printz, MD, of UC San Diego Department of Pediatrics and Rady Children's Hospital-San Diego; Sonia Jain, PhD, and Xiaoying Sun, MS, UCSD Department of Family and Preventive Medicine; Joan M. Pancheri, RN, of y Children's Hospital-San Diego; and Preeti Jaggi, MD, John P. Kovalchin, MD, and Octavio Ramilo, MD of Nationwide Children's Hospital and The Ohio State University, Department of Pediatrics.

Funding for the study was provided by the U.S. Food and Drug Administration, Robert Wood Johnson Foundation, and Janssen Biotech, Inc.

The PTS-X™ Sizing Balloon Catheters Are Recommended for Use in Those Patients with Cardiovascular Defect



B Braun International Systems' PTS-X™ Sizing Balloon Catheters are recommended for use in those patients with cardiovascular defects wherein accurate measurements of the defect are important to select the appropriately sized occluder device.

- Braided inner tubing optimizes catheter trackability to prevent kinking and buckling in tortuous vasculature.
- The PTS-X™ Sizing Balloon catheter has short tapers at the distal and proximal ends for enhanced transition across the

stenosis and post-dilatation ease of withdrawal into the introducer.

- The PTS-X™ Sizing Balloon catheter is super thin for a low deflated profile that maintains tip flexibility.
- The lumen labeled with the balloon size is for balloon inflation, while the through lumen allows the catheter to track over a guidewire.
- This lumen has two radiopaque platinum marker bands under the balloon shoulders, and there are two additional radiopaque platinum marker bands 5mm on either side of the center of the balloon.

For more information on B Braun International Systems' PTS-X™ Sizing Balloon Catheters, visit: www.bisusa.org.

Clinical Opinion Published on Use of Maternal Oxygen During Labor

When a fetal heartbeat pattern becomes irregular during labor, many practitioners give oxygen to the mother. But questions remain whether this oxygen supplementation benefits the fetus or may actually be potentially harmful.

A clinical opinion written by third year resident Maureen Hamel, MD, along with maternal-fetal medicine specialists Brenna Anderson, MD and Dwight Rouse, MD, of the Department of Obstetrics and Gynecology at Women & Infants Hospital of Rhode Island and The Warren Alpert Medical School of

Brown University, has been published in the January 10, 2014 online edition of the *American Journal of Obstetrics & Gynecology*.

The manuscript, entitled "Oxygen for intrauterine resuscitation: Of unproved benefit and potentially harmful," aimed to make recommendations about the safety of the use of maternal oxygen supplementation in laboring women.

According to lead author Dr. Hamel, "Maternal oxygen is often given to laboring women to improve fetal metabolic status or in an attempt to alleviate non-reassuring fetal heart rate patterns. However, there are only two randomized trials investigating the use of maternal oxygen supplementation in laboring women. These studies did not find that supplementation is likely to benefit the fetus and may even be harmful."

Based on their research, the team concludes that until it is studied properly in a randomized clinical trial, maternal oxygen supplementation in labor should be reserved for maternal hypoxia (lack of oxygen) and should not be considered an indicated intervention for non-reassuring fetal status.

Kawasaki Disease and Pregnant Women - UC San Diego Researchers Say Risks Are Manageable, Provided Doctors Recognize Them

Newswise — In the first study of its type, researchers at the University of California, San Diego School of Medicine have looked at the health threat to pregnant women with a history of Kawasaki disease (KD), concluding that the risks are low with informed management and care. The findings are published in the March 6, 2014 online edition of the *British Journal of Obstetrics and Gynaecology*.

KD is a childhood condition affecting the coronary arteries. It is the most common cause of acquired heart disease in children. First recognized in Japan following World War II, KD diagnoses are rising among children in Asia, the United States and Western Europe. Predictive models estimate that by 2020 one in every 1,600 American adults will be affected by KD.

"A growing number of women with a history of KD are reaching child-bearing age, but there is little information available to guide their obstetrical care," said study author Jane C. Burns, MD, Professor and Director of the Kawasaki Disease Research Center at UC San Diego and Rady Children's Hospital-San Diego. "By and large, KD is virtually unknown among working obstetricians."

KD is currently diagnosed by a constellation of clinical signs, with supporting lab tests that indicate high levels of inflammation. These signs include abrupt onset of high fever, accompanied by four of five criteria, among them: widespread rash, cracked and fissured lips, "strawberry tongue," bloodshot eyes, lymph node enlargement and red, swollen hands and feet.

Without treatment, 25% of children with KD develop coronary artery aneurysms – balloon-like bulges of heart vessels – that may eventually result in heart attacks, congestive heart failure or sudden death. The condition can be treated with a high-dose of intravenous immunoglobulin and aspirin, reducing the risk of aneurysms to 5%. The long-term risk for adults with a history of KD in childhood is not known.



Join Us for Two Weeks to Save Children with Congenital Heart Defects!

We are looking for medical volunteers who can join us in our missions to underdeveloped countries to perform pediatric cardiac surgery and care while also training the local medical staff.

For more information and our 2014 mission schedule, please visit Babyheart.org



Senior study author John Gordon, MD, and colleagues conducted the first KD study of non-Japanese patients, and the first to explore the health risks to women with a history of KD and their offspring. They found that the health risks for mothers with no KD-related coronary artery damage were similar to the general population. For women with aneurysms, the risks were low with appropriate management and care.

"The main message is positive," said Burns. "Women who have had KD can successfully deliver to term without complications. C-sections are not necessarily indicated if they have aneurysms, they can labor normally, if their overall cardiovascular status is OK."

There is a genetic component to KD. The study found that two of the 21 children born to the 10 women with a history of KD also developed the disease. "There is clearly an increased risk in offspring," said Burns, "but the (study) numbers are small so we cannot really calculate a risk until there is a larger population of KD adults who have had children."

Co-authors include: C.T. Gordon, S. Jimenez-Fernandez and C. Shimizu, Department of Pediatrics, UCSD; L.B. Daniels and A.M. Kahn, Department of Medicine, UCSD; M. Tarsa, Department of Maternal and Fetal Medicine, UCSD; T. Matsubara, Juntendo University Urayasu Hospital; and John B. Gordon, San Diego Cardiac Center.

Funding for this research came, in part, from grants from the National Heart, Lung, Blood Institute of the National Institutes of Health (grant HL U54 HL108460), the American Heart Association and the Gordon and Marilyn Macklin Foundation.

First Human Totally Endoscopic Aortic Valve Replacements (TEAVR) Reported

Newsweek — Surgeons in France have successfully replaced the aortic valve in two patients without opening the chest during surgery. The procedure, using totally endoscopic aortic valve replacement (TEAVR), shows potential for improving quality of life of heart patients by offering significantly reduced chest trauma. It is described in *The Journal of Thoracic and Cardiovascular Surgery*, an official publication of the American Association for Thoracic Surgery.

Endoscopic surgery is already used by cardiovascular surgeons for procedures such

as atrial septal defect repair and coronary artery bypass grafting. This leads to faster recovery time and less pain, which improves patients' quality of life.

TEAVR had not been feasible previously because of the currently available designs of stented tissue valves. The recent advent of sutureless bioprostheses mounted on a compressible self-expanding nitinol (nickel titanium) stent, was one of the key factors enabling the surgical team to perform this procedure. Implantation required less than 45 minutes in either patient. Sutureless substitutes are not yet available for the other cardiac valves, like the mitral valve.

"In our institution, we began by adopting the mini-sternotomy technique, involving a small incision through the sternum, as routine. We then transitioned to the right mini-thoracotomy approach, involving a small incision through the thorax, first under direct view, then with an endoscopic camera. Finally we adopted a totally endoscopic technique," explains lead author Marco Vola, MD, PhD, of the Department of Cardiovascular Surgery, Centre Hospitalier Universitaire de Saint-Etienne, France.

"It is important to note that when performing TEAVR, a quick and safe conversion to mini-thoracotomy under direct view can be made if circumstances demand. This would still offer significantly reduced chest trauma," he adds.

In other fields, totally endoscopic surgery involved longer clamping and cardiopulmonary bypass (CPB) times during the learning curve. The investigators believe that clamping and CPB times were acceptable and that the learning curve could be shorter than reported for totally endoscopic coronary artery bypass grafting.

Enhancements such as endoscopic sizers, dedicated instruments for decalcification, and second-generation sutureless bioprostheses to simplify implantation, could improve the procedure further, Dr. Vola and his colleagues comment. Last but not least, surgical robots may offer additional benefits.

"These first procedures show that totally endoscopic sutureless aortic valve replacement is technically feasible. Further clinical experience and technical development are necessary to shorten operation times and to assess further the potential postoperative benefits of TEAVR," concludes Vola.



PICS-AICS
Pediatric and Adult Interventional Cardiac Symposium

SAVE THE DATE
JUNE 7-10, 2014
Marriott Chicago
DOWNTOWN

CHICAGO

WWW.PICSYMPOSIUM.COM



LIVE CASE DEMONSTRATIONS • ABSTRACT SESSIONS • "MY NIGHTMARE CASE IN THE CATH LAB" • HOT DEBATES • WORKSHOPS • SMALLER BREAKOUT SESSIONS



RUSH UNIVERSITY
MEDICAL CENTER

Sponsored for CME credit by Rush University Medical Center



30th Annual

Echo in Pediatric and Adult Congenital Heart Disease

October 15 - 19, 2014 - Arizona Grand, Phoenix, AZ

Course Directors: Patrick W. O'Leary, M.D. and Cetta, Frank Jr., M.D.

www.mayo.edu/cme/cardiovascular-diseases-2014R195



Researchers Stress the Need to Strengthen Pediatric Clinical Trials Infrastructure

Children's National Physician Edward Connor Authors Article in *Science Translational Medicine*

Washington, DC— Strengthening the infrastructure required to conduct pediatric research is essential for obtaining information needed for the medical care and treatment of children, wrote Children's National Health System's Director of the Office of Innovation Development, Edward M. Connor, MD, MBE, the lead author of the article "Meeting the Demand for Pediatric Clinical Trials," published in the journal *Science Translational Medicine*.

In recent years, legislation and regulations around the need for high-quality data on medications used in pediatric patients have gotten stronger, increasing the demand for pediatric clinical trials. To meet this demand, the authors, who include researchers from the National Institutes of Health (NIH) and the U.S. Food and Drug Administration (FDA), highlight the need for an improved and standardized research infrastructure to better guide treatment and care for children.

"Many drugs used in children have not been adequately studied in this population," says Dr. Connor. "While this situation is improving there is still a lot of work to do to fill the knowledge gap, particularly in newborn infants and young children."

The authors suggest that collaboration among the public and private sectors is needed to ensure that the proper expertise and infrastructure are in place.

"In the past, the required trials networks were created as the need arose then dismantled when it waned," says Dr. Connor. "The increasing demand for data that meet regulatory standards for labeling means that we need to find ways to sustain infrastructure for pediatric trials. The pediatric research community, federal agencies, advocates, and industry are all making progress, but a comprehensive and urgent solution is needed."

According to Dr. Connor, pediatric trials that are used for drug labeling demand a level of quality standard that is higher than the average study. Having a network of experts in pediatric clinical trials and an enhanced infrastructure in place would ensure consistent, high-quality data are

produced to guide therapeutic decisions in pediatric clinical practice.

"Establishing such an enhanced infrastructure has been a major goal of the child health platform within the Clinical and Translational Science Award (CTSA) National Consortium," says Lisa Guay-Woodford, MD, Director of the CTSA program, a partnership between Children's National and George Washington University. "This report provides a clear roadmap for our continued efforts."

"There is a need for high-quality, compliant pediatric clinical trials infrastructure," said study co-author Steven Hirschfeld, MD, PhD, Associate Director for Clinical Research at the Eunice Kennedy Shriver National Institute of

Child Health and Human Development (NICHD). "The NICHD's Pediatric Trials Network provides needed infrastructure for evaluating drugs commonly prescribed off label for the express purpose of obtaining information needed for pediatric labeling."

Co-authors of the article were: William E. Smoyer, MD, Nationwide Children's Hospital, Jonathan M. Davis, MD, Tufts Medical Center, Anne Zajicek, MD, PharmD, and Steven Hirschfeld, MD, PhD, the Eunice Kennedy Shriver National Institute of Child Health and Human Development, Linda Ulrich, MD, Office of Orphan Products Development at the FDA, and Mary Purucker, MD, PhD, the National Center for Advancing Translational Sciences at the NIH.

Going to PICS ~AICS this June?

Do you use a tablet or smartphone?

Would you like to get the program, abstracts, daily briefing and other meeting information on your tablet?

Get the official PICS~AICS app from Congenital Cardiology Today for your Android smartphone, tablet, iPhone or iPad.

Send an email to: app@cct.bz and we'll send you an email when the app is ready.



CONGENITAL CARDIOLOGY TODAY

© 2014 by Congenital Cardiology Today (ISSN 1554-7787-print; ISSN 1554-0499-online).
Published monthly. All rights reserved.
www.CongenitalCardiologyToday.com

Mailing Address:

PO Box 444, Manzanita, OR 97130 USA
Tel: +1.301.279.2005; Fax: +1.240.465.0692
Editorial and Subscription Offices:
16 Cove Rd, Ste. 200, Westerly, RI 02891 USA

Publishing Management:

- Tony Carlson, Founder, President & Sr. Editor - TCarlsonmd@gmail.com
- Richard Koulbanis, Group Publisher & Editor-in-Chief - RichardK@CCT.bz
- John W. Moore, MD, MPH, Group Medical Editor - JMoore@RCHSD.org

Editorial Board: Teiji Akagi, MD; Zohair Al Halees, MD; Mazeni Alwi, MD; Felix Berger, MD; Fadi Bitar, MD; Jacek Bialkowski, MD; Mario Carminati, MD; Anthony C. Chang, MD, MBA; John P. Cheatham, MD; Bharat Dalvi, MD, MBBS, DM; Horacio Faella, MD; Yun-Ching Fu, MD; Felipe Heusser, MD; Ziyad M. Hijazi, MD, MPH; Ralf Holzer, MD; Marshall Jacobs, MD; R. Krishna Kumar, MD, DM, MBBS; John Lamberti, MD; Gerald Ross Marx, MD; Tarek S. Momenah, MBBS, DCH; Toshio Nakanishi, MD, PhD; Carlos A. C. Pedra, MD; Daniel Penny, MD, PhD; James C. Perry, MD; P. Syamasundar Rao, MD; Shakeel A. Qureshi, MD; Andrew Redington, MD; Carlos E. Ruiz, MD, PhD; Girish S. Shirali, MD; Horst Sievert, MD; Hideshi Tomita, MD; Gil Wernovsky, MD; Zhuoming Xu, MD, PhD; William C. L. Yip, MD; Carlos Zabal, MD

Statements or opinions expressed in Congenital Cardiology Today reflect the views of the authors and sponsors, and are not necessarily the views of Congenital Cardiology Today.

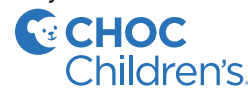


**Western
Society
Of
Pediatric
Cardiology**

25th Annual Conference of the Western Society of Pediatric Cardiologists

Especially designed for care providers of patients with pediatric, adolescent, or adult congenital heart disease. Hosted by Children's Hospital of Orange County

May 11-13, 2014; St. Regis Monarch Beach, Dana Point, CA
www.choc.org/wsopc



NIT-OCCLUD[®]

COIL SYSTEM FOR PDA CLOSURE



A SAFER, EASIER WAY TO CLOSE

The Nit-Occlud[®] coil system for PDA closure is designed to match individual morphologies and sizes

The delivery system facilitates optimal device positioning

The Nit-Occlud[®] PDA coil is repositionable and retrievable prior to release

Tight and compact windings ensure efficient occlusion

The Nit-Occlud[®] PDA coil was determined to be MRI conditional

Radiopaque

For more information or to place an order, contact your B. Braun Interventional Systems Inc. representative or call 1-877-VENA-CAV (836-2228)

pfmmedical

Manufacturer:
pfm medical, ag
Wankelstraße 60
50996 Köln, Germany
T +49 (0)2236 9641-10
F +49 (0)2236 9641-20



B. BRAUN

Interventional
Systems

Distributed by:
B. Braun Interventional Systems Inc.
824 Twelfth Avenue
Bethlehem, PA 18018 USA
Tel: 1-877-VENA CAV (836-2228) (USA)
Fax: 1-610-849-1334
www.bisusa.org

Indication: The Nit-Occlud[®] PDA coil is a permanently implanted prosthesis indicated for percutaneous, transcatheter closure of small to moderate size patent ductus arteriosus with a minimum angiographic diameter less than 4mm. Nit-Occlud Brief Statement: Do not implant the Nit-Occlud PDA into patients who have endocarditis, endarteritis, active infection, pulmonary hypertension (calculated PVR greater than 5 Wood Units), thrombus in a blood vessel through which access to the PDA must be obtained, thrombus in the vicinity of the implantation site at the time of the implantation or patients with a body weight < 11 lbs (5 kg). An angiogram must be performed prior to implantation for measuring length and diameter of the PDA. Only the pfm medical implantation delivery catheter should be used to implant the device. Administration of 50 units of heparin per kg body weight should be injected after femoral sheaths are placed. Antibiotics should be given before (1 dose) and after implantation (2 doses) in order to prevent infection during the implant procedure. Do not implant the Nit-Occlud PDA in an MR environment. Do not pull the Nit-Occlud coil through heart valves or ventricular chambers. Contrast media should not be injected through the implantation catheter. The catheter must not be connected to high pressure injectors. Patients may have an allergic response to this device due to small amounts of nickel that has been shown to be released from the device in very small amounts. If the patient experiences allergic symptoms, such as difficulty in breathing or swelling of the face or throat, he/she should be instructed to seek medical assistance immediately. Antibiotic prophylaxis should be performed to prevent infective endocarditis during first 6 months after coil implantation. Potential Adverse Events: Air embolism, Allergic reaction to drug/contrast, Apnea, Arrhythmia requiring medical treatment or pacing, Arteriovenous fistula, Bacterial endocarditis, Blood loss requiring transfusion, Chest pain, Damage to the tricuspid or pulmonary valves, Death, Embolization of the occluder, requiring percutaneous or surgical intervention, Endarteritis, False aneurysm of the femoral artery, Fever/Headache/migraine, Heart failure, Hemolysis after implantation of the occluder, Hypertension, Hypotension or shock, Infection, Myocardial infarction, Occluder fracture or damage, Perforation of the heart or blood vessels, Stenosis of the left pulmonary artery or descending thoracic aorta, Stroke/TIA, Thromboembolism (cerebral or pulmonary), Valvular Regurgitation, Vessel damage at the site of groin puncture (loss of pulse, hematoma etc.)