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ATRIOVENTRICULAR BLOCK IN TRANSCATHETER CLOSURE OF VENTRICULAR SEPTAL DEFECT: A PRELIMINARY STUDY

By Song Zhiyuan, MD; Zhang Zhihui, MD; He Guoxiang, MD; Su Maoqin, MD; Hu Houyuan, MD; Gong Shifei MD; Liu Jianping, MD; and Ran Boli, MD

Abstract

To determine the incidence of atrioventricular block (AVB) in patients having closure of ventricular septal defect (VSD) with the Amplatzer device, one hundred and thirty-six patients were included in a retrospective analysis. Electrocardiogram (ECG) and dynamic electrocardiogram were analyzed before, during and after catheterization. There were a total of 7 patients with AVB, 4 cases occurred during catheterization, 3 cases occurred after catheterization. The AVB disappeared after adjustment of catheter position in one case during catheterization. In three patients, the procedures was abandoned due to persistent AVB. Among the three cases with AVB after catheterization, one patient developed complete left bundle branch block on the third day after catheterization. The other two patients fainted suddenly on the fourth and sixth days after catheterization. Their ECGs showed intermittent complete AVB. One recovered after treatment with a seven day course of high-dose dexamethasone. The other recovered one month after catheterization. Although we consider closure of VSDs using the Amplatzer device to be effective and safe, AVB during or after the procedure are important complications. High-dose dexamethasone may be helpful for patients who develop AVB after catheterization.

Introduction

Isolated ventricular septal defect (VSD) is the most commonly recognized form of cardiac malformation and constitutes over 20% of all congenital heart disease[1]. Persistent left-to-right shunt enhances the risk of endocarditis and arrhythmias due to the effects of high velocity blood flow. Surgery is a well-established method used for the treatment of VSD, however, complications may occur. They include post-pericardiotomy syndrome, complete atrio-ventricular block, bundle branch block, early and late cardiac arrhythmias, residual shunt or tricuspid regurgitation. Deaths have also been reported.

Therefore, as an alternative approach to surgery, transcatheter closure of VSD has been attempted using a variety of occluding devices. In 1997, Masura et al[2] performed a transcatheter closure of atrial septal defect with the use of a new type of Amplatzer occluder. The introduction of this method into clinical practice was a breakthrough in non-surgical treatment of some congenital heart disorders. In 1999, Thanopulos[3] and Tofeig[4] introduced Amplatzer technology to close defects in the muscular part of the interventricular septum. Since then, the Amplatzer VSD occluder has been used widely [5-9]. However, there are still some serious complications, including atrioventricular block (AVB), that may occur during and after device closure. In this study, we report our experience with the Amplatzer VSD occluder and discuss how to treat patients with AVB developing during and after catheterization.

Methods

Patients

From October 2002 to April 2005, 136 patients with clinical and echocardiographic findings of a large VSD underwent transcatheter closure with the Amplatzer VSD occluder. Seven patients (4 male, 3 female) developed AVB during or after catheterization. Their ages ranged from three to eight-

een years, (average 7.7 years), and their median body weight was 17.5kg (range 10-45kg). Six patients had membranous VSD (2 simple membranous and 4 perimembranous VSD), and one had a muscular VSD. Transthoracic echocardiography indicated that the average diameter of the membranous VSDs was 4.9 + 1.7 mm (range 3-8 mm) and the mean distance from the aortic valve was 4.3 + 1.1 mm (range 3-6 mm). The muscular VSD had a diameter of 3.5 mm and was located in the trabecular muscular septum. All cases had indications for transcatheter closure and no contraindications. Informed parental consent for the procedures was obtained for each patient.

Procedure

Transcatheter closure was performed using general anesthesia to allow for continuous echocardiographic visualization of the defect. The diameter of the defect on the left ventricular size was measured during diastole. Left ventricular angiography was performed in the long axis view (60°left-anterior oblique, 20°cranial), and the size of the defect on the left ventricular side was measured.

A 5-6 F Judkins right coronary curve catheter and a long floppy exchange wire (Noodle wire; AGA Medical) were used to cross the VSD from the left ventricle. A loop of the Noodle guidewire was advanced into the right atrium and was snared and pulled out through the femoral vein using a 25 mm Amplatz goose neck snare (Microvena). A loop of this guidewire was formed in the apex of the left ventricle by pushing a stiff snare guide catheter to the apex of the left ventricle, keeping the long sheath away from the mitral apparatus. A 7-8 F long sheath (AGA Medical) was then passed over the wire from the femoral vein into the apex of the left ventricle. The dilator and wire were removed.

A VSD occluder was soaked in flush solution, attached to the delivery cable and drawn into the loader. The compressed device was then introduced into the long sheath and advanced into the apex of the left ventricle. The left ventricular disc was extruded, and after checking that it was away from the mitral valve tension apparatus, it was pulled with the long sheath onto the left ventricular surface of the defect. While maintaining tension on the delivery cable, the stent and right ventricular disc were deployed.

If echocardiography showed that the device was well aligned with both aspects of the ventricular septum without tricuspid valve encroachment, the device was unscrewed from the delivery cable. Left ventricular angiography was performed and repeat haemodynamic measurements were made.

Patients were monitored by ECG for 24 hours after device implant. They were evaluated by ECG, echocardiography and 24 hour dynamic ECG four days after catheterization, and by ECG and 24-hour dynamic ECG 2 months, 6 months, and one-year after device implant.

Results

General Characteristics of VSDs

Right atrial pressure (RAP), right ventricular pressure (RVP) and systolic pulmonary artery pressure (PAP) of all 7 cases were 7.86 ± 1.25 (range 6-10) mm Hg, 30.6 ± 4.1 (range 25-38) mm Hg and 30.1 ± 3.9 (range 24-37) mm Hg respectively. VSDs were measured by ventriculography. The defect diameters of 6 patients with membranous VSDs were 4.8 ± 1.6 (range 3.5-8) mm. The other case had a twisted tubiform muscular VSD with defect size of 3.5 mm. All the occluders used (AGA Medical) had diameters between 1-3 mm larger than the VSD diameters measured on the left ventricular sides of the defects.

Complete AVB Occurrence and Treatment

There are 7 patients who developed AVB in this study. Four cases occurred during catheterization; 3 cases developed after catheterization. Among the cases with AVB during catheterization, one developed complete AVB when the right coronary artery catheter irritated the interventricular septum. AVB disappeared after adjustment of catheter position. Catheterization in this patient was completed and a device was implanted. This patient was monitored for one week after catheterization. No AVB recurred. The muscular VSD patient developed complete AVB before device placement, and was treated with dexamethasone (10 mg i.v.) over 30 minutes. The patient's AVB resolved and no device was implanted. The other two cases developed AVB at the time of occluder placement. AVB disappeared when the occluders in these patients were taken out. We replaced and adjusted the positions of these occluders repeatedly, but CAVB recurred each time. These procedures were abandoned. Diameters of these occluders were 2 and 3 mm larger than the VSD diameters measured by left ventriculography. Among the three cases with AVB after catheterization, one patient developed complete left bundle branch block on the third day without any specific symptoms. This patient recovered after treatment with high dose hydrocortisone (total dose of 2500 mg). The other two patients presented with syncope on the fourth and sixth days after catheterization. ECG indicated intermittent complete AVB

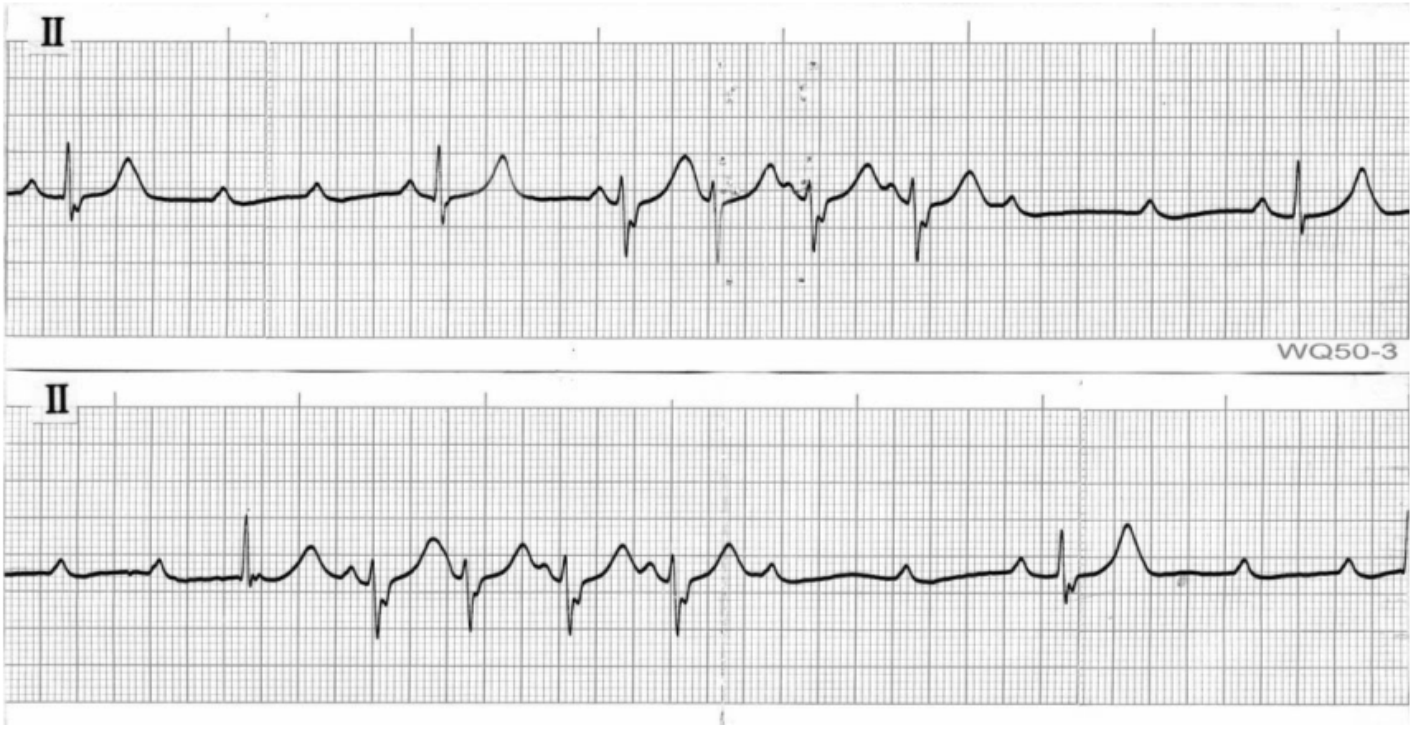

Figure 1. Continuous record of ECG (lead II) indicates an intermittent CAVB.

(Figure 1) in both patients. One of these patients recovered completely after treatment with high dose hydrocortisone for seven days. This patient had no further symptoms and had complete recovery of AV conduction (Figure 2). The other patient was also treated with high dose hydrocortisone, but continued to have intermittent compete AVB, but no symptoms for the two weeks of monitoring in hospital.

Follow-ups

One patient had intermittent complete AVB after discharge from hospital. This patient was treated with prednisone for one month and monitored by dynamic ECG. The average follow up for the seven patients was 9.1 months (range 2-24 months). At the last follow-up all patients were asymptomatic and ECGs and dynamic ECGs were normal.

Discussion

Complete AVB is a significant complication of surgery for congenital heart disease. In 1970s, the incidence of AVB in patients with cardiac surgery ranged from 0.5% to 3% in China. This incidence is about 0.9% of all cardiac surgery patients and 0.7% of patients having VSD surgery.[10] Although the incidence is lower in recent years, AVB is still a major problem. It can occur at the operation or several months after surgery. With the advent of the Amplatzer VSD occluders, most membranous and muscular VSDs can be safely and effectively closed. However complete AVB is also reported (11,12). Furthermore, it is not known how to prevent or treat AVB.

In catheter closure of VSD, many factors may predispose to AVB. The most common are: close proximity of the VSD to the bundle of His, the left or right bundle branch, and compression and/or damage of the conduction pathways from the occluders or wires. We encountered four patients who developed AVB during catheterization. In order to prevent irreversible AVB, we abandoned the procedures in three patients without implanting devices. In some patients, the His bundle is very superficial and near the left ventricular crest of the septum. Passage of the catheter across the defect in one such patient in our series caused transient AVB. We were able to implant a device in this patient without recurrence of AVB.

The reason for delayed AVB is not clear. It may be related to cellular edema of cardiac muscle after mechanical compression by occluders. This hypothesis is supported by the following evidence. First, most AVB cases occur in the first week after VSD closure without any clinical signs of acute upper respiratory

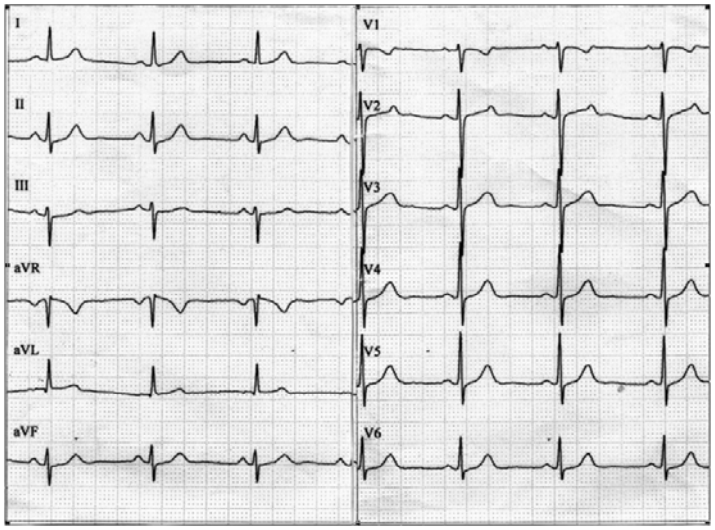

Figure 2. For same patient in Figure 1, ECG record (12 leads) became normal after treatment with hormone for 7days.

tract infection and acute viral myocarditis. Second, a large dose of steroids improves AVB in many patients. However, there are also some phenomena which can not be explained by muscle edema. For example, some delayed AVB is intermittent but associated with ventricular pauses of more than 10 seconds. In the same patient, normally conducted heart rates in excess of 100 beats per minute may be observed. The reasons for this apparent paradox are unclear.

Delayed AVB after transcatheter closure of VSD is unusual. However, it is a significant complication especially if it occurs after hospital discharge. Because of our experience with 3 cases of delayed AVB, we believe the following points are important to prevent and treat delayed AVB. First efforts should be made to select the proper size occluder. Over-sized occluders are a common cause of AVB. We suggest dividing the diameter of VSDs into three groups: ≤5 mm, 6-9 mm and ≥10 mm according to left ventriculography. Occluders should be selected which are 0-1mm, 1-2 mm and 2-3 mm larger than VSDs respectively in each group. Second, after catheterization, the patient should be monitored in the hospital for one week in order to identify delayed or intermittent complete AVB. Finally, when delayed complete AVB or bundle branch block occurs, we suggest using steroids aggressively.

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PREVIEW—CARDIOLOGY 2007

By Gil Wernovsky, MD

The Cardiac Center at The Children's Hospital of Philadelphia is pleased to present *Cardiology 2007: 10th Annual Update on Pediatric Cardiovascular Disease-New and Evolving Practices*. The course will be held at Disney's Yacht and Beach Club Resorts, in Lake Buena Vista, Florida, from February 21st through 25th, 2007. See www.chop.edu/cardiology2007 for more detail.

While there are many excellent meetings for sub-specialists in pediatric cardiology, intensive care, surgery, nursing and perfusion, this annual meeting brings together all who care for children with cardiovascular disease, highlighting the teamwork necessary to provide optimal care. The course faculty will present over 150 plenary and subspecialty lectures in all aspects necessary to the care of neonates, children and young adults with cardiovascular disease. The multidisciplinary, multi-institutional faculty chosen from 23 institutions in the United States, Canada, Great Britain and Australia will provide a balanced, and sometimes controversial, approach to diagnosis and management. In addition, special lectures on the increasingly important matters of hospital and practice administration will be held daily.

In addition to discipline-specific breakouts in echocardiography, electrophysiology, catheterization, nursing, perfusion, intraoperative and postoperative care, plenary sessions will be held daily on common congenital heart disease and important topics in our current practice. Each day will include a debate on controversial topics in the field.

Patient Safety. An increased emphasis on patient safety has been recognized as a crucial element in today's practice. Topics in this session will include improving information transfer during shift change and patient handover, preventing nosocomial infections, how to change institutional culture and maintain a quality improvement program, and a discussion on work hour restrictions, culminating in a debate on the topic between Dr. Alan Freidman (Yale, USA) and Mr. Martin Elliot (Great Ormond Street, London).

"In addition to discipline - specific breakouts in echocardiography, electrophysiology, catheterization, nursing, perfusion, intraoperative and postoperative care, plenary sessions will be held daily on common congenital heart disease and important topics in our current practice."

Cardiopulmonary Resuscitation and Mechanical Support. The new guidelines for pediatric CPR have been published—but are they relevant for children with congenital heart disease? In the opening session on the State of the Art of Cardiac Resuscitation, faculty will discuss the current guidelines, the science upon which they are based, and their applicability to patients with CHD. Teamwork during CPR will be emphasized, as well as new techniques to improve outcome. A debate on open chest cardiac massage will be held between Dr. Jim Tweddell (Milwaukee, USA)

Featured Lecturers

	Surgery	Nursing	Cardiology
2007	Pedro del Nido, MD	Martha A. Q. Curley, RN, PhD	Philipp Bonhoeffer, MD
2006	Martin Elliott, MD	Kathleen Mussatto, RN	Andrew Redington, MD
2005	Edward Bove, MD	Elisabeth C. Smith, RGN, RSCN	Norman Silverman, MD
2004	Bill Williams, MD	Mary Fran Hazinski, RN, MSN	Jane Newburger, MD
2003	Thomas Spray, MD	Patricia Hickey, RN, MSN, MBA	Welton Gersony, MD
2002	Aldo Castaneda, MD	Catherine K. Madigan, RN, MSN	Thomas Graham, MD
2001	Marc de Leval, MD	Nancy Eckle, RN, MSN	
2000	Tom Karl, MD	Jane Barnsteiner, RN, PhD	

"New this year!!!: A special ½ day seminar will be held for trainees and junior faculty early in their career to provide guidance on career choices, work-life balance and practical guidelines for long-term success in cardiovascular medicine. An up-to-date "job-board" and other career opportunities will be hosted by Congenital Cardiology Today."

and Dr. Tom Spray (Philadelphia, USA) to close the session. In a follow-up session, the use of extracorporeal membrane oxygenation in critically ill cardiac patients will be critically reviewed.

Coronary Artery Disease in Children. Although a major focus for our colleagues in adult cardiology, this topic has received little attention to date. However, there is increasing recognition that native or post-surgical coronary abnormalities may become a significant problem in pediatric cardiology in the next decade. Topics will include imaging and techniques to diagnosis coronary insufficiency in children, and will close with a debate on whether surgery should be performed in asymptomatic children with structural coronary abnormalities between Dr. Tim Feltes (Columbus, USA) and (in his second debate) Dr. Jim Tweddell.

Audience Interaction. Controversies in management, ethical issues and variability in practice patterns will be revealed with an interactive audience response system. Registered course attendees are encouraged to pre-submit challenging cases for discussion by the faculty at the open forum on February 24th, 2007. Deadline for submission is February 1st. Information will be provided upon course registration.

In addition, live demonstrations of anatomical specimens and review of perioperative care, imaging, intraoperative video and follow-up care for:

- Double Outlet Right Ventricle
- Complete Atrioventricular Canal/ Atrioventricular Septal Defect
- Heterotaxy/Isomerism

New this year!!!: A special ½ day seminar will be held for trainees and junior faculty early in their career to provide guidance on career choices, work-life balance and practical guidelines for long-term success in cardiovascular medicine. An up-to-date "job-board" and other career opportunities will be hosted by *Congenital Cardiology Today*.

Finally, three preconference symposia will be held in the morning and afternoon of February 21st, 2007, including PALS recertification, CPR/Mock Codes and an in-depth review of cardiac anatomy. We are very excited about the program and faculty this year, and Orlando in February is a terrific vacation location for a break from the winter doldrums. Hope to see you there. If you have comments on the program, or suggestions for next year, please feel free to contact me.

~CCT~

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MEDICAL NEWS, NEW PRODUCTS AND INFORMATION

Genetic 'Missing Link' Sheds Light on Sudden Cardiac Death

An electrical imbalance caused by a malfunctioning gene triggers a potentially fatal heart rhythm disorder, according to researchers at Baylor College of Medicine (BCM) and Texas Children's Hospital in Houston, TX.

The findings were reported in the Nov. 21, 2006 print edition of the Journal Circulation, a publication of the American Heart Association. The journal has posted the findings online.

Electrical impulses originate in the top of the heart's right atrium and travel through the muscle fibers, causing the heart to contract. The genetic and molecular basis of sudden cardiac arrest, which occurs when these impulses are disrupted, is not well understood.

Study investigators began to close this gap of understanding by becoming the first to isolate a gene called Caveolin-3, which influences the electrical-muscular impulses that drive the heart's rhythm. A mutation of the gene can trigger arrhythmia associated with long QT syndrome, a hereditary disorder that can occur in otherwise-healthy people of all ages, and increases the risk of sudden cardiac death.

"This is part of a totally new concept in which the structural part of the heart is intertwined and connected with the electrical part," said first author Dr. Matteo Vatta, assistant professor of pediatrics at BCM and pediatric cardiac researcher at Texas Children's Hospital. "This is the missing link between the heart's electrical and muscular activities."

Vatta says that conventional treatments for long QT syndrome have targeted ion channels, or proteins that govern membrane structure, rather than proteins that regulate the heart's electrical impulses through these channels.

"Many people that have arrhythmia abnormalities take medications that do not work," said Vatta. "Perhaps they should target the other proteins that are modulating the ion channels rather than the ion channel itself."

The effect of the mutation may also be enhanced by medications for unrelated conditions, such as asthma, increasing the risk of cardiac arrhythmia.

Caveolin-3 regulates the cardiac sodium channel, an important protein that can cause rapid structural changes in the heart in response to fluctuations in the electrical field. Heart muscle disease can also disrupt Caveolin-3.

Muscle diseases called cardiomyopathies are the leading cause of sudden cardiac death, of which there are more than 300,000 cases every year in the United States. Electrical abnormality in the heart is the leading cause of sudden cardiac death in the absence of muscle disease.

The study, funded by the National Institutes of Health, reviewed 905 cases of patients of all ages with long QT syndrome. Dr. Jeffery Towbin, professor of pediatrics at BCM and chief of pediatric cardiology at Texas Children's Hospital, served as senior author.

The study was also conducted at the Mayo Clinic and at the University of Wisconsin.

Medication Costs Infrequently Addressed by Physicians

A new UCLA study, which appeared in the November issue of the American Journal of Managed Care, found that physicians discuss cost and aspects of obtaining newly prescribed medications only about one-third of the time during patient/doctor interactions.

But questions about pricing and prescription drug insurance coverage are critical — the high costs of drugs, including out-of-pocket payouts such as co-payments, are linked to patient non-adherence in maintaining their dosage schedules, said Dr. Derjung Tarn, assistant professor of family medicine at the David Geffen School of Medicine at UCLA and the study's lead author.

"Though cost discussions are not always necessary, especially if physicians know a patient's financial situation and the best formulary choice for a medication, physicians must have a high level of awareness about medication cost and issues impeding acquisition to medication, because these can be important barriers to patient medication adherence," Tarn said.

The researchers used a combination of patient and physician surveys and transcriptions from audio-taped patient visits at

two Sacramento, Calif., healthcare systems from January to November 1999. They included a total of 185 patients on outpatient visits with 15 family physicians, 18 internists and 11 cardiologists. Some 243 new medications were prescribed during these sessions.

Of the 185 patients, who had a mean age of 55 years, half were male, 83% were white, most had health insurance and more than three-fourths paid less than half of their prescription drug costs out-of-pocket. Family physicians saw 31% of the patients, internists examined 47%, and cardiologists advised 23%.

The researchers found that in only 33% of the cases did physicians prescribing new medications communicate about issues related to medication acquisition such as cost, insurance, generic or brand name, logistics, supply and refills. Costs and insurance were covered 12% of the time, the logistics of obtaining medications 18% of the time, and 9% of the time. Patients initiated discussions about costs or insurance in only 2% of the cases.

Discussions about costs were likelier to take place when the patient earned less than $20,000 per year compared with patients whose annual income topped $60,000, the researchers found. Also, family physicians and internists were less likely than cardiologists to discuss costs, and physicians in general brought up the issue less when prescribing medications to older patients.

Other researchers on this study in addition to Tarn are John Heritage, Ron. D. Hays and Neil Wenger, all of UCLA; and Debora A. Paterniti and Richard L. Kravitz of UC Davis.

MEDICAL CONFERENCES

38th Annual Cardiovascular Conference at Snowmass
January 15-19, 2007; Snowmass, CO USA
www.scai.org

International Congress for Genetics in Pediatrics
January 24-27, 2007; Luxor, Egypt
www.genevent.org

The Conference on Neonatology
February 7-10, 2007; Lake Buena Vista, FL USA
www.pediatrix.com

322nd Annual Cardiovascular Conference at Hawaii
February 12-16 15-19, 2007; Kohala Coast, HI USA
www.scai.org

Cardiology 2007
February 21-25, 2007; Lake Buena Vista, Florida USA
www.chop.edu/cardiology2007/

Hands-on Cardiac Morphology
February 26-28, 2007; London, UK
mailto:Morphology@rbht.nhs.uk

Cardiology in Neonates and Infants 2007
March 1-2, 2007; London, UK
mailto:Morphology@rbht.nhs.uk

11th Vail Symposium on Pediatric Cardiac Diseases
March 4-6, 2007; Vail, CO USA
www.TheChildrensHosptial.org

The 6th Interventional Workshop on Interventional Pediatric Cardiology
March 28-31 2007; San Donato, Italy
www.workshopIPC.com

OPBG Cardiovascular International Valvar Heart Disease in Children
April 18-20 2007; Rome, Italy
Mailto:congressi@opbg.net

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In the last 20 years, there has been a revolution in caring for children with congenital heart disease. Thanks to advances in diagnostics, surgery and treatment, many of these children are living into adulthood and enjoying active lives. With dedicated pediatric cardiology capabilities, the iE33 system answers your need for high performance, non-invasive diagnostic imaging for these patients. Now, the new X7-2 transducer combines xMATRIX technology with PureWave crystal technology for remarkable Live 3D Echo imaging in small patients.

The X7-2 transducer's footprint is small enough to allow intercostal access in pediatric applications and it uses an extremely lightweight, ultra-flexible cable. In addition to excellent 3D image quality, the X7-2 offers PW Doppler and superb color sensitivity in an impressively compact form factor.

The X7-2 transducer joins a full complement of cardiac transducers on the iE33 system, including pediatric and adult TEEs, to enable you to image your complete range of patient types – regardless of age, disease state or body composition. In addition, a unique analysis package truly follows the patient the way you do – so you don't have to fit your requirements into an analysis package designed for adult echocardiography.

Dedicated to your pediatric cardiology clinical and workflow requirements, our commitment goes beyond the system and its technologies. Global educational programs, outstanding service and worldwide centers of excellence help you get the most out of your system every day.

FOR MORE INFORMATION

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Expanding the clinical utility of echo with Live 3D specifically developed for pediatric imaging

The new X7-2 pediatric Live 3D transducer shown actual size.

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