

Today's Highlights

**Cross Specialty:
Echocardiography & Cardiac
Surgery**

Cross Specialty: Neurology & Intervention

What We Know? What We Don't Know
08:40-10:10 AM / Rm. Walker 2

Healthcare Policy

Education Workshop

08:40 AM - 17:10 PM / Rm. Cosmos

New Frontiers in Cardiology 3

Plenary Lecture in Heart Failure
10:20-11:50 AM / Rm. Theatre

Arrhythmia 6

*Current Practice and Future
Perspective in Cardiac Arrhythmia*
10:20-11:50 AM / Rm. Walker 1

Cross Specialty: Acute Myocardial Infarction & Basic Research

Ethics Workshop

14:00-15:30 PM Rm. Theatre

New Frontiers in Cardiology 3

The Past, Present and Future of Heart Failure Medical Therapy



Barry Greenberg, MD
University of
California San Diego
Medical Center, USA

Heart failure is pandemic with prevalence increasing throughout the world. Patients suffering from heart failure experience frequent hospitalizations, reduced survival and markedly impaired quality of life. Fortunately, medical therapy for heart

failure has improved over the past several decades. An increasing number of drugs and devices that have been shown to favorably alter the natural history of patients with heart failure with reduced ejection fraction (HFrEF) are now available. Unfortunately, for patients with heart failure with preserved ejection fraction (HFpEF), currently available medical therapy has not been shown to alter the natural history of the disease.

Whereas in the past, heart failure was considered a cardiac disorder that resulted in congestion due to the 'back-up' of blood from the heart, it is now recognized as a complex systemic disease involving the vasculature, kidneys, skeletal muscle and brain. The use of vasodilator drugs to unload the heart was an important breakthrough, as treatments had previously depended on a combination of digitalis glycosides to improve myocardial contractility and diuretics to relieve congestion. Recognition that the renin-angiotensin system (RAS) and its main effector molecule, angiotensin II (Ang II), played an important role in regulating vascular tone, resulted in the development of angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin

receptor blockers (ARBs). These agents not only improved the clinical course of heart failure patients but also provided insights into the pathophysiology of heart failure, as their beneficial effects transcended their ability to unload the heart. Understanding the central role of neurohormonal activation in promoting cardiac remodeling and progression of heart failure led to the development of additional strategies including the use of beta-blockers and mineralocorticoid receptor antagonists, both of which have been shown to greatly alter the natural history of patients with HFrEF.

The role of neurohormones in modulating the progression of heart failure, however, is more nuanced than simply blocking activities of maladaptive systems. Counter-regulatory systems that are compensatory including the natriuretic peptides, bradykinin, prostaglandins, adrenomedullin, and others are also activated. Angiotensin receptor neprilysin inhibitors (ARNIs) combine blockade of Ang II with neprilysin inhibition which augments levels of compensatory peptides by blocking their breakdown. In the recent PARADIGM-HF study, the combination of valsartan and sacubitril was superior to an ACEI in reducing mortality as well as improving quality of life in patients with HFrEF. Subsequent studies have demonstrated the safety and efficacy of initiating an ARNI during heart failure hospitalization.

Gene and stem cell therapies are novel strategies for treating heart failure. Gene therapy is based on the premise that delivery of a critically important gene that has either been down-regulated or in which a mutation has altered,

the production or function of its product will help restore normal cardiac function. Novel viral vectors that allow the gene of interest to be preferentially taken up by cardiomyocytes are being explored. A variety of stem cells that either promote regeneration of cardiomyocytes or help repair adjacent myocardium through the release of signaling molecules are also being tested as therapies for heart failure. Mesenchymal precursor cells, previously shown in early studies to favorably affect clinical outcomes in heart failure, are now being evaluated in the Phase 2b/3 DREAM-HF trial. Dr. Greenberg will provide a lecture regarding an overview of the past, present, and future medical therapies, with emphasis on drugs used to treat HFrEF.

The Past, Present and Future of CRT



Eugene S. Chung, MD
The Christ Hospital,
USA

Cardiac resynchronization therapy (CRT) has been used to treat systolic heart failure with evidence of electrical dyssynchrony for two decades. The evolution of evidence from feasibility trials to completion of pivotal

Continued on page 6



First and on1st

국내 유일 아토르바스타틴+에제티미브 복합제 **아토젯TM**으로
고지혈증 환자의 지질관리를
시작해주세요. ^{3,4}

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Program at a glance: Day 3, Oct 20, 2019

	Theatre Main Arena (B1)	Grand 4 (B1)	Walker 1 (1F)	Walker 2 (1F)	Cosmos (3F)	Calla (3F)	Art (4F)	Pine (4F)	Oak (4F)	Grand Hall (B1)		Vista Hall (B2)	
										Abstract / Case Zone 1	Abstract / Case Zone 2	Abstract / Case Zone 3	Abstract / Case Zone 4
08:40 - 10:10	Cross Specialty: Echocardiography & Cardiac Surgery Management of Ischemic Mitral Regurgitation; Where We Stand?	Healthcare Policy 1 미생물지 심혈관계질환에 미치는 영향 및 개선 대책	Arrhythmia 5 Syncope and Sudden Cardiac Death	Cross Specialty: Neurology & Intervention What We Know? What We Don't Know	Education Workshop 1: Intervention The Role of Interventional Therapy Disease	Cardio- Oncology 1 All About the Cardiotoxicity	Adult Congenital Heart Disease 1 Pulmonary Hypertension in Adult Congenital Heart Disease	Hypertension 1 Debate: Start with Single Pill Combination		CAD 6 251-258	Heart Failure 4 259-266	Epidemiology & Prevention 2 267-274	Arrhythmia 6 275-283
10:20 - 11:50	New Frontiers in Cardiology 3 Plenary Lecture in Heart Failure	Healthcare Policy 2 심장재활과 2차 예방	Arrhythmia 6 Current Practice and Future Perspective in Cardiac Arrhythmia	Intervention 3 My Secret Tips as an Interventional Cardiologist	Education Workshop 2: Echocardiography Echocardiography in Adult Congenital Heart Diseases	Cardio- Oncology 2 Collaboration of Cardiology and Hemato-Oncology	Adult Congenital Heart Disease 2 How to Manage Cardiovascular Disease in Pregnant Women	Hypertension 2 How to Improve Hypertension Control		CAD 7 284-291		Epidemiology & Prevention 3 292-299	Arrhythmia 7 300-308
12:00 - 12:40	Scientific Session [Novartis]		Scientific Session [Daewoong / Daiichi Sankyo]		Scientific Session [Hanmi]					E-Poster 1-83			
12:40 - 14:00	임상심장학 연수강좌 1 [12:30 - 13:50] 최신 가이드라인 리뷰									Case Presentation			
										-	Arrhythmia 2 57-63	Echo & Imaging 2 64-69	Intervention 3 70-76
										Moderated Poster Presentation			
14:00 - 15:30	Ethics Workshop [필수교육] 의사가 알아야 할 최신 윤리	임상심장학 연수강좌 2 Hot Issues in 2019 - 부정맥	Cross Specialty: Acute Myocardial Infarction & Basic Research Myocardial Damage and Salvage Strategies	Intervention 4 Structural Heart Intervention: Who Are Appropriate Candidates?	Education Workshop 3: Arrhythmia Current Practice in Management of AF	Echocardiography 6 Role of Multimodality Imaging in Various Disease	Pediatric Cardiology 3 Save the Aorta	Healthcare Policy 3 신로필수가제 관련 빅데이터를 임상에 어떻게 활용할 것인가?	Cardio- Oncology 309-316	Intervention 5 317-324	전공의 Awards Oral 1-8	Arrhythmia 8 341-349	
		임상심장학 연수강좌 3 개원자가 알아야 할 최신 임상연구 리뷰	Arrhythmia 7 CIED Summit	Intervention 5 'How to' Issues in Antithrombotic Treatment	Education Workshop 4: Heart Failure Management of Acute Heart Failure	Echocardiography 7 Interesting Imaging Cases from Percutaneous Procedures	Pediatric Cardiology 4 Inherited Cardiac Diseases and More	Healthcare Policy 4 공익적 임상연구 (대한심장학회/ 심장학연구재단 미래 전략연구소)	CAD 8 325-332	Intervention 6 333-340	전공의 Awards Case 1-8		
15:40 - 17:10													

Scientific Sessions

Scientific Session 8 [Novartis]

Latest Update for Heart Failure Treatment with ARNI
» Oct 20, 12:00-12:40 PM Rm. Theatre

Scientific Session 9 [Daewoong/Daiichi Sankyo]

Effective Statin Treatment Strategy in Korean Population
» Oct 20, 12:00-12:40 PM Rm. Walker 1

Scientific Session 10 [Hanmi]

New Insight on Intensive Treatment for Hypertension and Dyslipidemia Management
» Oct 20, 12:00-12:40 PM Rm. Cosmos

Cross Specialty: Echocardiography & Cardiac Surgery

Evaluation and Mechanism of Ischemic Mitral Regurgitation



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Korea

Ischemic mitral regurgitation (MR) is a complication of the chronic healing phase of myocardial infarction (MI). As the left ventricle remodels after MI, the normal geometry of the mitral apparatus becomes distorted, resulting in incomplete leaflet closure and valvular regurgitation.

Mitral valve closure is a dynamic process in which two opposing forces, a tethering force and a closing force act simultaneously on the leaflets, determining their instantaneous position throughout systole. The tethering force, imparted by the PM and chords, pulls the leaflets away from the annular plane, while the closing force, generated by LV contraction, drives them in the opposite direction (Figure 1). Ischemic MR results from an imbalance

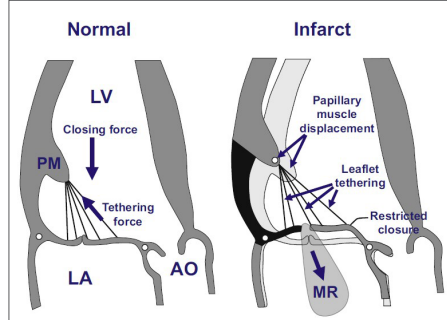


Figure 1. Closing and tethering forces in the normal ventricle (left) and after inferior MI (right)

between these forces, tipped in favor of the former. As the left ventricle remodels after MI, increased tethering impairs the systolic excursion of the leaflets toward the annulus, and valvular competence becomes increasingly dependent on closing forces.

Echocardiography plays an important role in the evaluation of ischemic MR. Localized or diffuse changes in LV size and shape due to post-MI remodeling can be readily appreciated. Echocardiography is also useful in characterizing deformational changes in the mitral leaflets caused by tethering.

As our understanding of its pathogenesis continues to evolve, additional insights will undoubtedly lead to the development of innovative therapies in the future.

Management of Severe Ischemic Mitral Regurgitation (Repair vs. Replacement)



Young-Nam Yoon, MD, PhD
Yonsei University
Severance Hospital,
Korea

Ischemic mitral regurgitation (MR), which occurs in about 20–30% of patients with prior myocardial infarction, is associated with aggravation of heart failure symptoms and an increase in cardiovascular mortality and morbidities. It should be corrected surgically if certain hemodynamic severity criteria are met and in patients who continue to experience symptoms of heart failure despite optimal medical

therapy. However, current guidelines do not suggest which of the available approaches to mitral valve surgery-mitral valve (MV) repair or replacement (MVR) is superior for this indication. While MV repair has been known to confer improved survival, MVR may provide higher rates of freedom from recurrent MR.

Current guidelines suggest that MV surgery should be considered for patients with severe ischemic MR who are symptomatic despite being on optimal pharmacological heart failure therapy.

MV surgery for secondary MR, including that due to ischemic injury, predominantly involves either MV repair utilizing a restrictive annuloplasty or chordal sparing MVR. Observational studies generally have reported lower perioperative morbidity and mortality with MV repair than with MVR. However, valve repair is less durable and is associated with a greater than 50% recurrence of ≥moderate MR as follow-up progresses. The Cardiothoracic Surgical Trials Network (CSTN) performed a randomized controlled trial comparing MV repair versus MVR specifically for ischemic MR. The primary endpoint of the CSTN trial was LV end-systolic volume index (LVESVI), a marker of LV reverse remodeling linked to clinical outcome. At 1-year and 2-year follow-ups, no significant difference was found in LVESVI and mortality between the two strategies.

Once the decision is made to intervene on the MV, the surgeon must choose between repair and replacement. In the most severely ill patients of whom the life expectancy is below 5 years with extensive

comorbidities, chordal sparing MVR should be considered, as such patients derive no benefit from MV repair, and a reliable and rapid operation is recommended. In patients with stable functional ischemic MR, repair with a ring annuloplasty is associated with good durability and seems to improve survival when compared with MVR. The American Association for Thoracic Surgery for management of patients with severe ischemic MR recommended the consideration for performing MVR over MV repair in patients with basal aneurysm/dyskinesis, substantial leaflet tethering, and/or moderate to severe LV remodeling (LV end-diastolic dimension >65 mm [COR IIa, LOE A]) and MV repair in the absence of these features (COR IIb, LOE B).

All studies related are limited by short-duration follow-up, inclusion of patients with different types of MV diseases other than ischemic MR, and lack of information on the important secondary outcomes, such as quality of life. Currently, MV repair with ring annuloplasty appears to be optimal to correct ischemic MR provided that there is no major leaflet or chordal pathology. However, if major leaflet or chordal procedure is required, especially on the anterior leaflet, then MVR is likely to be most effective for prevention of recurrent MR.

Cross Specialty: Echocardiography & Cardiac Surgery
Management of Ischemic Mitral Regurgitation; Where We Stand?

» Sunday, Oct 20, 08:40-10:10 AM / Theatre

KSC 2019 Case Competiton

12:40-14:00
GRAND HALL/VISTA HALL

Today's Interview

10:30-11:00 Cross Specialty: Intervention & Arrhythmia
Interviewer: Choong Hwan Kwak, Eun-Seok Shin
Interviewee: Keun-Ho Park, Young-Hoon Jeong, Jong-Il Choi, Hee Tae Yu

11:30-12:00 Cross Specialty: Neurology & Intervention
Interviewer: Jong Sung Kim, Do-Sun Lim
Interviewee: Jun Lee, Jung-Sun Kim, Young-Hoon Jeong

12:30-13:00 New Frontiers in Cardiology 3
Interviewer: Dong Ju Choi, Eui-Young Choi
Interviewee: Eugene S. Chung, Jon A. Kobashigawa

Cross Specialty: Acute Myocardial Infarction & Basic Research

Influence of Local Myocardial Infarction on Endothelial Function, Neointimal Progression, and Inflammation in Target and Non-Target Vascular Territories in a Porcine Model of Acute Myocardial Infarction



Hyun Kuk Kim, MD
Chosun University
Hospital, Korea

Pre-existing plaque vulnerability in non-culprit lesions was considered as a major determinant of major adverse cardiac events (MACE). However, there are several reports on how plaque vulnerability alone cannot fully explain future adverse cardiac events after acute myocardial infarction

(AMI). Endothelial dysfunction and inflammation could be the triggering factors for MACE in AMI non-culprit lesions. However, it is difficult to distinguish the cause from effect between these factors and AMI in a human study.

Dr. Hyun Kuk Kim and his colleagues conducted a study to assess why non-culprit lesions in AMI patients have poor outcomes than that of stable coronary artery patients. The schematic method and results are presented in Figure 1. In this study, endothelial function, neointimal progression, peri-strut inflam-

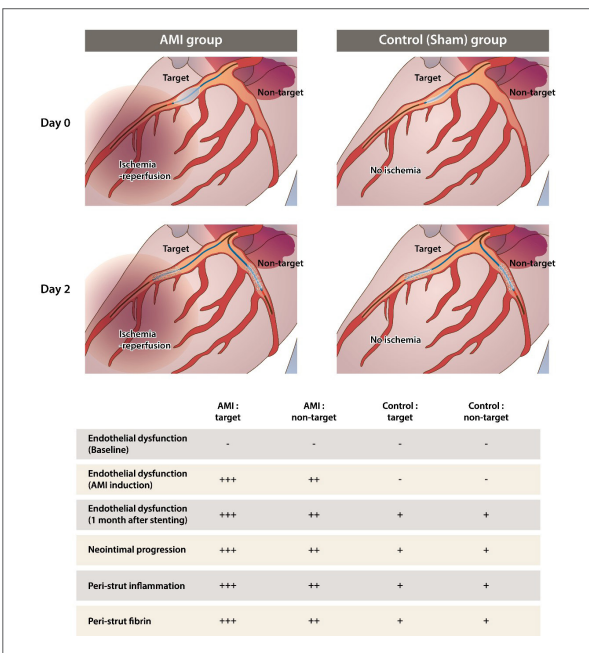


Figure 1. Schematic illustration of the methods and results

mation, peri-strut fibrin formation, and systemic inflammation were evaluated. AMI events induced endothelial dysfunction, inflammation, and neointimal progression in both the target and non-target vessels compared with the same procedure control. According to Dr. Kim, "These findings support the facts that AMI patients have worse clinical outcomes than those with stable coronary artery disease despite revascularization, and non-culprit lesions also involve more adverse cardiovascular events regardless of underlying plaque vulnerability".

Cross Specialty: Acute Myocardial Infarction & Basic Research
Myocardial Damage and Salvage Strategies

» Sunday, Oct 20, 14:00-15:30 PM / Walker 1

Arrhythmia

Keynote Lecture: State-of-art: Inherited Arrhythmia Predisposing Sudden Cardiac Death

Discoveries of genetic basis pave the path for personalized medicine



Inherited primary arrhythmia syndromes (IPAS) develop ventricular tachycardia or ventricular fibrillation by some genetic disorders, leading to sudden cardiac death. IPAS are also called "channelopathies" since many of these are caused by an abnormality in myocardial ion channels. It is becoming in-

most of which are in SCN5A. Furthermore, even in the SCN5A mutation-positive carriers, their phenotypes are not completely consistent to BrS, but may cause other IPAS including LQTS, cardiac conduction defect, sick sinus syndrome, and dilated cardiomyopathy. On the other hand, a recent Japanese BrS registry demonstrated that the pore-region mutations in SCN5A are significantly associated with a risk of lethal cardiac events (**Figure 1**).

Furthermore, a genome-wide association study revealed that a common variant in SCN10A or HEY2 in addition to SCN5A is associated with BrS, thus, BrS may not be a monogenic Mendelian disease but probably an oligogenic disease.

In this session, Dr. Aiba will present the basic genetic and pathophysiological findings of the IPAS, particularly LQTS and Brugada syndrome, and outline a rational approach to genetic testing, management, and family screening.

Arrhythmia 5 Syncope and Sudden Cardiac Death

» Sunday, Oct 20, 08:40-10:10 AM / Walker 1

Keynote Lecture: State-of-art: Past, Current & Future of Device Therapy in Cardiac Arrhythmias

Big wave: Paradigm shifts in cardiac implantable electronic device



Over the past 6 decades, conduction system disorders have been treated with electronic pacemakers, which is developed to electrically stimulate the heart muscle. Cardiac pacing began in the 1930s with Hyman's 'artificial pacemaker'. After World War II, public perception changed and daring pioneers made great advances. Initially designed as external devices, technological advances resulted in miniaturization of the electronic device and these advances continue to date. In this session, Dr. Lee will present the history of the pacemaker, the current status of device therapy in Korea, limitation of device therapy in past and present, and also current or future device therapy (**Figure 2**).

The number of device implantations in Korea, including pacemaker and implantable cardiac defibrillator (ICD), is gradually increasing. Traditionally, a pacing electrode was implanted in the right ventricular apex for ventricular excitation, which is known to accompany several complications including vascular complications, tricuspid valve problem, and pacing-induced cardiomyopathy. Also, many pacemaker-related complications (infection, thrombosis, lead failure, and pneumothorax) are related to particularly the leads. To improve these limitations, leadless pacemaker, His-bundle pacing device, and subcutaneous ICD (S-ICD) have been developed. Furthermore, MRI compatible devices are available these days. Device volumes are getting smaller, and meanwhile, longevity is becoming much longer.

Remote monitoring via cardiac rhythm devices, not yet available in Korea due to legal limitations, will be presented. In the future, batteryless devices, or biological pacemaker would be expected to be available, which could overcome the clinical limitations of present devices.

Arrhythmia 7 CIED Summit

» Sunday, Oct 20, 15:40-17:10 PM / Walker 1

Intervention

How to Choose the Optimal Blood Thinner after Structural Cardiovascular Intervention?



After transcatheter interventions of structural heart diseases, careful patient management should include optimal antiplatelet or antithrombotic medication. Unfortunately, there are still unmet needs with regard to optimal antithrombotic therapy after these procedures. Stemming from the experience of coronary stents, the first trials evaluating transcatheter aortic valve replacement (TAVR) used dual antiplatelet therapy (DAPT) with aspirin and clopidogrel for up to 6 months. The risk-benefit trade-off of DAPT has been the subject of debate in recent years. In patients with high bleeding risk, single antiplatelet therapy (SAPT) with aspirin or clopidogrel alone may now be considered (Class IIb), according to the 2017 recommendations of the European Society of Cardiology.

The 2017 focused update of the American Heart Association and American College of Cardiology has granted a Level IIb recommendation to the use of vitamin K antagonists (VKA) within the first 3 months post-TAVR in patients without high risk of bleeding. Several ongoing randomized trials are evaluating different antithrombotic regimens after TAVR.

After atrial septal defect (ASD) closure, pharmacological therapy is commonly accepted currently, based on clinical practice and randomized clinical trials. Aspirin and clopidogrel are started preprocedurally, with a loading dose of 300 and 600 mg, respectively. Patients usually should receive DAPT for 3 months and continue administering aspirin up to 6 months after procedure.

After percutaneous left atrial appendage (LAA) closure, warfarin and aspirin are administered for 45 days, followed by DAPT-clopidogrel until 6 months, and aspirin for life. New studies suggest that the use of novel oral anticoagulant (NOAC) instead of warfarin within the first 45 days after interventional LAA closure is safe and effective.

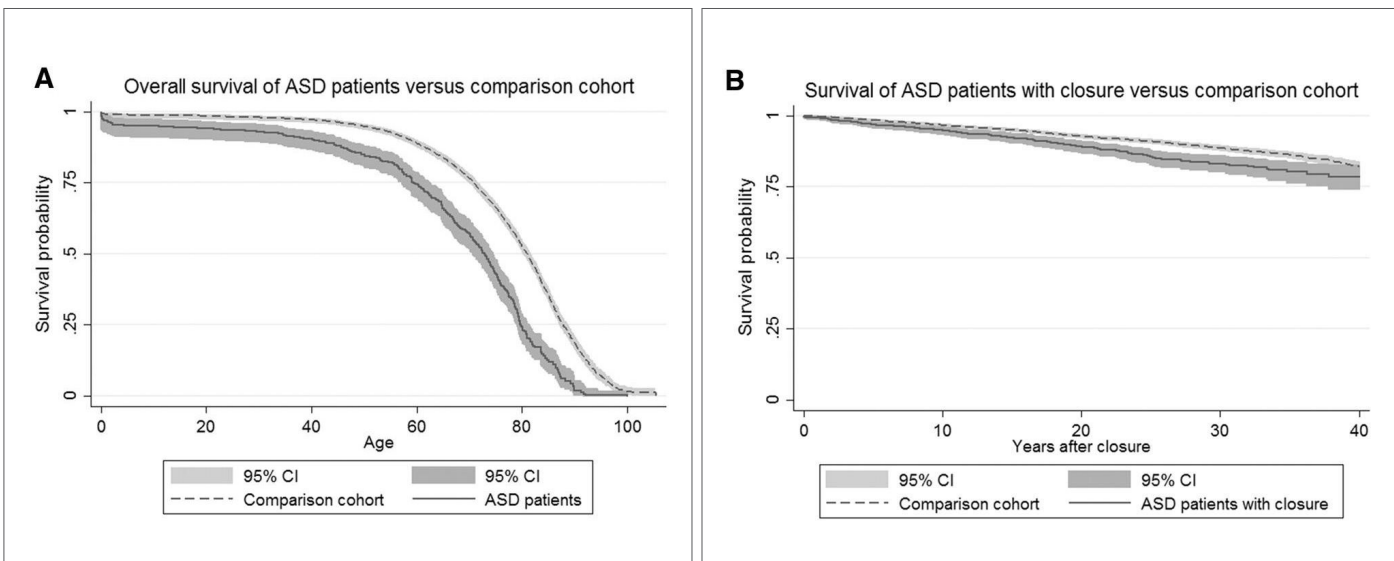


Figure 1. Overall increased mortality risk of ASD patients (Adapted from Eur Heart J 2018;39:993-998.)

Does All ASD Needs to be Closed?



Atrial septal defect (ASD) is one of the most common congenital heart diseases in adults, accounting up to 15-20% of all adult congenital heart diseases. Current clinical treatment guideline recommends that ASD should be closed in patients with significant shunt (signs of right ventricular volume overload) and pulmonary vascular resistance (PVR) <5 wood unit (WU) regardless of symptoms. Also, ASD closure should be considered for patients with suspicion of paradoxical embolism (exclusion of other causes) regardless of its size. Guideline mentions that patients with PVR >5 WU but <2/3 systemic vascular resistance or pulmonary artery pressure <2/3 and evidence of net left to right shunt (Qp:Qs) >1.5 may be considered for ASD closure.

Early studies argued that there is no major benefit of ASD closure in adults. However, latest studies revealed there was a higher incidence of major cardiovascular events (MACE) in unrepaired ASD patients, even in the hemodynamically insignificant (no right ventricular enlargement, thromboembolic complications, Qp:Qs <1.5) ASD. Compared with the normal population, death by heart failure was increased and a higher burden of chronic diseases was found and impaired submaximal exercise capacity was higher in the hemodynamically insignificant ASD patients (**Figure 1**). Also hidden burden of atrial and ventricular tachyarrhythmia were detected in this group of patients.

Recent nationwide population-based cohort study based on the national medical registries in Denmark and Taiwan demonstrated that correction of the small size ASD defect may help to decrease long-term mortality and MACE.

Based on these recent studies, several interventional cardiologists are insisting that all ASD, irrespective of their size, should be closed. It would be worthwhile

reconsidering therapeutic approaches for ASD patients.

Intervention 4 Structural Heart Intervention: Who Are Appropriate Candidates?

» Sunday, Oct 20, 14:00-15:30 PM / Walker 2

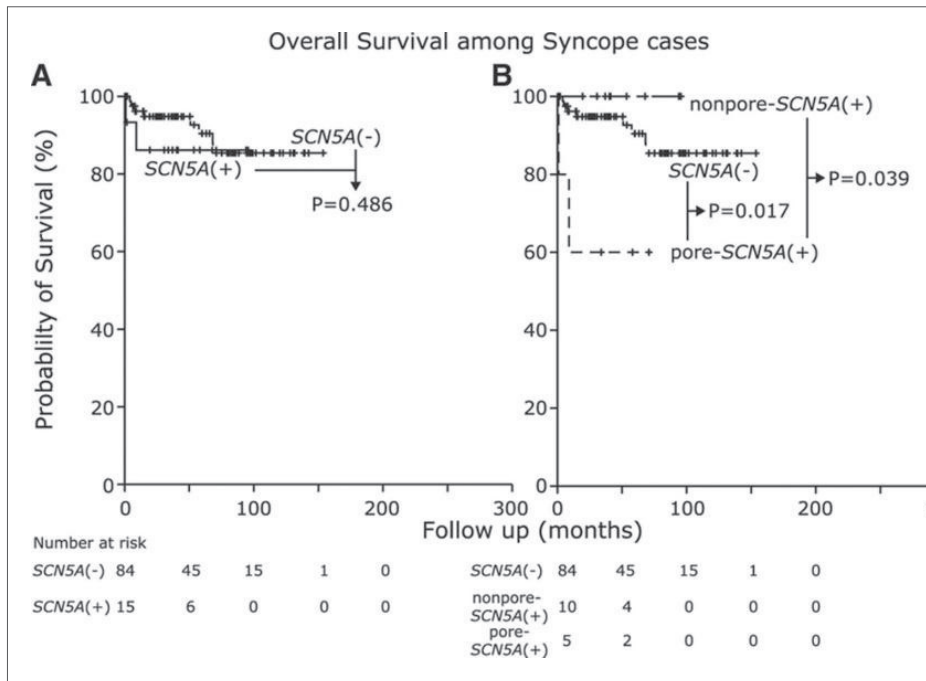


Figure 1. Kaplan-Meier analysis of the cardiac event-free survival in the prior syncope subgroup (Adapted from Yamagata K, et al. Circulation 2017;135:2255-2270.)

creasingly clear that collaborative efforts are needed to understand and manage these relatively rare but potentially lethal diseases. Congenital long-QT syndrome (LQTS) is the most well-documented IPAS, which may be seen in 0.1% of the general population. More than 15 disease-causing genes have been identified in almost 70% of LQTS patients and genetic testing is well-applied to not only clinical diagnosis but also risk stratification and gene-based therapeutic strategy for each person with LQTS. Thus, in LQTS, gene-based personalized medicine can be realized.

Unlike the LQTS, genetic testing for the Brugada syndrome (BrS) is still controversial since only 20% of patients can be identified the causing gene mutations,

CENTRAL ILLUSTRATION: An Overview of the History of Cardiac Pacing

Paradigm Shifts in Cardiac Pacemakers



Figure 2. An overview of the history of cardiac pacing (Adapted from Mulpuru SK, et al. J Am Coll Cardiol 2017;69(2):189-210.)

Healthcare Policy

Effect of Particulate Matter on Cardiovascular Disease and Arrhythmia



Weon Kim, MD, PhD
Kyung Hee University Medical Center, Korea

Air pollution is increasingly recognized as an important and modifiable factor of cardiovascular disease in developed countries, especially in urban communities. Epidemiological evidence indicates that exposures to fine particulate matter (PM 2.5) air pollution contribute to the global burden of increased risk of cardiovascular morbidity and mortality. Acute exposure has been linked to adverse cardiovascular events, including hospital admissions with angina, myocardial infarction, and heart failure. Long-term exposure increases a lifetime risk of death from cardiovascular disease.

The main mechanism of these adverse effects seems to be combustion-derived nanoparticles. Inhalation of this particulate matter leads to pulmonary inflammation with secondary systemic effects or, after translocation from the lung into the circulation, to direct toxic cardiovascular effects (Figure 1). Through the induction of cellular oxidative stress and proinflammatory pathways, including systems such as interleukin-1b, MCP, CD 40, vF-factor, particulate matter augments the development and progression of atherosclerosis via detrimental effects on platelets, fibrinolytic factors, vascular endothelium, electrophysiologic system such as action potential duration, heart rate variability and the myocardium. However, the mechanisms by which PM 2.5 exposure induces cardiovascular injury remain unclear. PM 2.5-induced endothelial dysfunction and systemic inflammation have been implicated, but direct evidence is lacking. A better understanding of the mediators and mech-

anisms of these processes is necessary if strategies to protect individuals at risk and reduce the effect of air pollution on cardiovascular disease need to be developed.

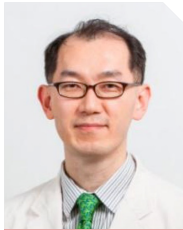
Many clinical data showed that air pollution is significantly associated with cardiovascular disease, such as ischemic stroke mortality, MI mortality, sudden cardiac death and long-term mortality. In summary, air pollution related literatures provide consistent evidence on the health effects of particulate matter. PM 2.5 appears to be the primary mediator of these adverse effects. Further, health effects of air pollution are probably still underestimated by decision makers around the world. Lastly, regulation of air quality needs sustained reassessment of emerging and existing air quality measures in addition to a standard for PM 2.5.

Healthcare Policy 1

Effects of Particulate Matter on Cardiovascular Diseases and Countermeasures

» Sunday, Oct 20, 08:40-10:10 AM / Grand 4

Role of Cardiac Rehabilitation Program in the Era of Statins and PCI



Jidong Sung, MD, PhD
Sungkyunkwan University Samsung Medical Center, Korea

Cardiac rehabilitation (CR) program has been established as an effective and essential treatment modality for management and secondary prevention of various cardiovascular diseases, and is strongly recommended by many guidelines. Among them, evidence for the effect of exercise-based CR is most abundant in coronary artery diseases. However, there is still a significant gap between the guideline recommendations and real-world practice due to various hurdles in the implementation. Moreover, recently there is a concern whether the effect of CR is still significant with optimal medical treatments and wide application of percutaneous coronary intervention (PCI). It has been argued that major advances in medical management of coronary artery disease may have led to a reduction in the incremental effect on mortality of exercise-based

CR compared with usual care alone. Other concerns include the inclusion of small, poor-quality RCTs, which may have resulted in overestimation of the benefits of CR, and the almost exclusive recruitment of low-risk, middle-aged, post-myocardial infarction male subjects in early trials, thereby reducing the generalizability of their findings to the broader population of coronary patients.

Recently updated meta-analyses mostly showed that exercise-based CR reduces cardiovascular mortality and in hospital admissions, while improving quality of life. However, not all meta-analyses showed the same results, and there are studies with a different conclusion that CR is not clearly associated with improved outcomes. Such results are probably associated with patient groups with lower risk and optimal treatment, and also there is a possibility that irregular and/or poor adherence to CR has some role in the dilution of its effect in trials. Overall, it would be fair to say that while it is difficult to deny the effect of CR entirely, there are certainly a trend of decreasing outcome benefit. On the other hand, modern CR program is aiming at a comprehensive program, which includes patient education and behavioral modification and not just exercise training. Long-term behavioral modification is an important aspect of patient management which cannot be replaced by pharmacological and/or procedural treatment, while proving its effect in trials is much more difficult compared to other pharmacological or procedural treatments.

In conclusion, CR is still an important and essential component in the management of cardiovascular diseases, and various efforts should be exercised to improve the performance of CR and patient's adherence to maintain its effect in the era of statins and PCI.

Healthcare Policy 2

Cardiac Rehabilitation and Secondary Prevention

» Sunday, Oct 20, 10:20-11:50 AM / Grand 4

What is DRG?



Sukil Kim, MD, PhD
The Catholic University of Korea, Korea

The private sector in Korea plays a substantial role in patient care, medical education, and research. The market share is now above 90%. The fee-for-service (FFS) system is the basic payment system for both public and private hospitals.

Health insurance reimbursements from the National Health Insurance Service and out-of-pocket money from patients after the healthcare service provision are funding the hospitals. The local and central government funding are limited.

Center for Medicare and Medicaid Services (CMS) defines Diagnosis Related Group (DRG) as "a patient classification scheme which provides a means of relating the type of patients a hospital treats (i.e., its case mix) to the costs incurred by the hospital". DRG has been used in the US and other countries for the prospective payment of hospital reimbursement system.

The first version of the Korean Diagnosis-Related Groups (KDRG) was developed by the Seoul National University Hospital Research Institute in 1986. KDRG v4.0, announced in 2016, is the latest official version. The Korean version of the International Classification of Diseases and Related Health Problems, tenth revision (ICD-10), and the Korean health insurance fee-schedule are the base classifications of the KDRG.

The KDRG-based prospective payment system started as a pilot project in 1997. In July 2012, KDRG-based payment for seven disease categories covering both hospital payment and doctors' fee became mandatory for clinics and hospitals. It was further extended to tertiary hospitals in July 2013. Also, there is another DRG-based prospective payment called the new DRG-based payment, a variant of Japanese diagnosis procedure combination (DPC) payment. Some of the doctors' fee was separately reimbursed, based on the FFS from hospital payment. The pilot project began in 2009. However, the new Korean government which emerged 2 years ago used the Japanese style payment system to reduce patients' out-of-pocket money. The government has been trying to recruit more hospitals to participate in the new DRG payment scheme by distributing excessive incentives.

Public hospitals were pushed to join the new scheme. Some private hospitals voluntarily joined the payment scheme because of a better reimbursement rate in total than the usual FFS scheme. There are still reasonable long-term resistances from the suppliers because of the inappropriate DRG classification and inadequate coverage of the care cost without incentives.

Healthcare Policy 3

The New DRG

» Sunday, Oct 20, 14:00-15:30 PM / Pine

Cross Specialty: Neurology & Intervention

Intraarterial Treatment (Thrombolysis and Thrombectomy) for Acute Ischemic Stroke: What Are Evolving?



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Since around 1995, intravenous recombinant tissue plasminogen activator (IV rtPA) has been a gold standard for patients with acute ischemic stroke. IV rtPA, however, is not always effective especially on intracranial large vessel occlu-

sion (LVO). On this ground, endovascular treatment (EVT) had been thought as an alternative treatment option for the LVO, but clinical trials had failed to prove its efficacy until early 2013. Since a successful clinical trial was firstly presented in October 2014, other prospective randomized control studies have also proved the efficacy and safety of EVT for intracranial LVO.

Three major key factors have influenced the success of the recent clinical trials when comparing previously failed and recent successful studies. First, the development of thrombectomy devices was an important factor. As stent retrievers were introduced and became widely used, the rate of

successful reperfusion has increased, resulting in the improvement of clinical outcomes. Second, in-hospital time frame such as door to puncture became much faster. Last but not least, patient selection methods have been meticulously developed for the success of clinical trials. For early time window (onset to puncture time <6h), a confirmation of LVO, National Institutes of Health Stroke Scale score ≥ 6 , and an Alberta stroke program early CT score on noncontrast CT ≥ 6 are the key inclusion criteria for endovascular treatment. For late time window (onset to puncture time 6h to 24h), clinical or radiological core-penumbra mismatch should be additionally confirmed and became widely used, the rate of

Whereas coronary artery occlusive disease are mostly caused by atherosclerosis, treatable intracranial LVO are mainly caused by embolism. Intracranial atherosclerotic occlusions, which are relatively frequent in the Asian population, fibrin clot, and tandem occlusions are challenging issues in the EVT. Further studies should be done to solve the challenging Asian-specific issues.

Cross Specialty: Neurology & Intervention

What We Know? What We Don't Know

» Sunday, Oct 20, 08:40-10:10 AM / Walker 2

Cardiology-Oncology 2

Malignant Cardiac Tumor



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Cardiac tumors are an uncommon disease, and it is sometimes difficult to distinguish them from other forms of cardiac mass, as cardiac tumors include both benign tumors and malignant tumors. Secondary metastatic cardiac tumors are more frequent with a

20-40 time higher incidence than primary cardiac tumors. Approximately 10% of primary cardiac tumors are malignant, and 90% are benign. It is very important to distinguish the tumor type because treatment strategies vary. Malignant cardiac tumors have rapid expansion and invade various cardiac structures, such as myocardium, pericardium and sometimes cardiac valves. Although surgical excision is recommended to confirm the pathology of cardiac mass, sometimes medical therapy, such as chemotherapy, is the first choice. In deciding the treatment strategy, clinicians should make a comprehensive consideration of the need for biopsy, patient's symptoms and accompanying systemic diseases, including cancer.

The role of multimodality noninvasive imaging techniques is important to diagnose and characterize the cardiac tumors their intra/extracardiac extension and predict malignancy. Echocardiography, cardiac magnetic resonance, computed tomography and positron emission tomography serve these roles. We will review various cases of malignant cardiac

tumors, which include both metastatic, secondary cardiac tumors and primary, malignant cardiac tumors.

Cancer VTE and Anticoagulation: Evidence from Clinical Trials



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Cancer-associated venous thromboembolism (cancer VTE) is a common complication associated with high morbidity and mortality. In accordance with major clinical trials comparing low-molecular-weight heparin (LMWH) with a vitamin K antagonist (VKA), LMWH is currently the standard treatment for cancer VTE, owing to its efficacy for thrombosis recurrence and improved safety profile compared to VKA. Over the past few years, direct oral anticoagulants (DOACs) have emerged as a potential alternative therapy to LMWH due to their convenient route of administration and predictable pharmacokinetics, but evidence for their use in cancer VTE is inconclusive, as only a small fraction of the study populations in these trials had cancer. Recently, three head-to-head

trials comparing DOACs to LMWH in CAT patients reported comparable efficacies of DOACs with increased bleeding risk in certain conditions. Occasionally, cancer VTE treatment can be challenging due to the heterogeneity of underlying malignancies and comorbidities. Renal insufficiency and gastrointestinal defects are the main obstacles in anticoagulant selection. Careful choice of treatment candidates and

proper anticoagulant strategies are critical for the treatment of cancer VTE; hence, more studies are required to address these challenges.

Cardio-Oncology 2

Collaboration of Cardiology and Hemato-Oncology

» Sunday, Oct 20, 10:20-11:50 AM / Calla

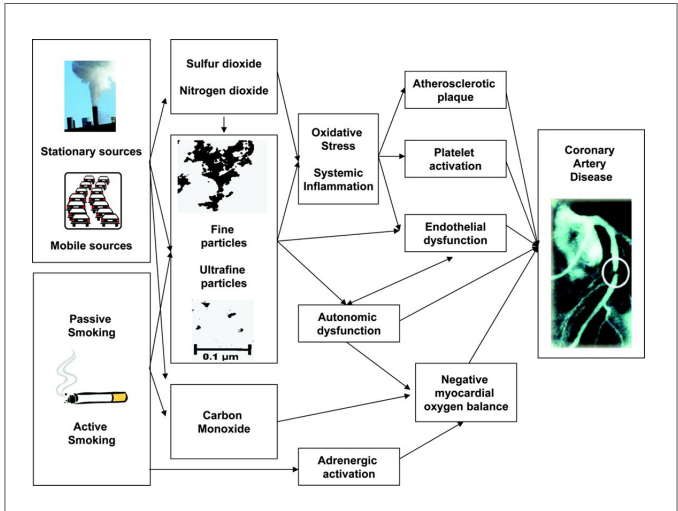


Figure 1. Overview on pathomechanism linking ambient air pollution (Adapted from Circulation 2009;120:924-927.)

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텔로스톱플러스정
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40/5/5mg, 40/5/10mg, 80/5/5mg, 80/5/10mg

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- 고혈압, 고지혈증을 동시에 치료해도 **One**
- 텔미사르탄, 알로디핀, 로수바스타틴을 동시에 처방해도 **One**

텔로스톱플러스정(Telmisartan/Amlodipine/Rosuvastatin)
40/5/5mg, 40/5/10mg, 80/5/5mg, 80/5/10mg

【효능·효과】 이 약은 텔미사르탄/알로디핀 복합제의 로수바스타틴을 동시에 투여하여 하는 한지에만 사용한다. 이 텔미사르탄/알로디핀 복합제 - 알로디핀 또는 텔미사르탄 단독요법으로 혈압이 조절되지 않는 만성성 고혈압, 이 로수바스타틴 단일제 - 1. 협심증, 2. 관상동맥질환, 3. 관상동맥질환 예방, 4. 관상동맥질환 치료, 5. 관상동맥질환 예방, 6. 관상동맥질환 치료, 7. 관상동맥질환 예방, 8. 관상동맥질환 치료, 9. 관상동맥질환 예방, 10. 관상동맥질환 치료, 11. 관상동맥질환 예방, 12. 관상동맥질환 치료, 13. 관상동맥질환 예방, 14. 관상동맥질환 치료, 15. 관상동맥질환 예방, 16. 관상동맥질환 치료, 17. 관상동맥질환 예방, 18. 관상동맥질환 치료, 19. 관상동맥질환 예방, 20. 관상동맥질환 치료, 21. 관상동맥질환 예방, 22. 관상동맥질환 치료, 23. 관상동맥질환 예방, 24. 관상동맥질환 치료, 25. 관상동맥질환 예방, 26. 관상동맥질환 치료, 27. 관상동맥질환 예방, 28. 관상동맥질환 치료, 29. 관상동맥질환 예방, 30. 관상동맥질환 치료, 31. 관상동맥질환 예방, 32. 관상동맥질환 치료, 33. 관상동맥질환 예방, 34. 관상동맥질환 치료, 35. 관상동맥질환 예방, 36. 관상동맥질환 치료, 37. 관상동맥질환 예방, 38. 관상동맥질환 치료, 39. 관상동맥질환 예방, 40. 관상동맥질환 치료, 41. 관상동맥질환 예방, 42. 관상동맥질환 치료, 43. 관상동맥질환 예방, 44. 관상동맥질환 치료, 45. 관상동맥질환 예방, 46. 관상동맥질환 치료, 47. 관상동맥질환 예방, 48. 관상동맥질환 치료, 49. 관상동맥질환 예방, 50. 관상동맥질환 치료, 51. 관상동맥질환 예방, 52. 관상동맥질환 치료, 53. 관상동맥질환 예방, 54. 관상동맥질환 치료, 55. 관상동맥질환 예방, 56. 관상동맥질환 치료, 57. 관상동맥질환 예방, 58. 관상동맥질환 치료, 59. 관상동맥질환 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Adult Congenital Heart Disease

Differential Diagnosis of Pulmonary Hypertension in ACHD: Multiple Comorbidity Conditions



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Pulmonary hypertension in congenital heart disease is frequently considered as “pulmonary arterial hypertension”, although there can be various causes. When there is a shunt flow, mixed nature of pulmonary hypertension is not easily differentiated from the others in clinical practice. Classification of pulmonary hypertension is as follows: Group 1: Pulmonary arterial hypertension; Group 2: Pulmonary hypertension due to left heart disease; Group 3: Pulmonary hypertension due to lung disease and/or hypoxia; Group 4: Pulmonary hypertension due to pulmonary artery obstruction; and Group 5: Pulmonary hypertension with unclear mechanisms (**Figure 1**). In congenital heart disease, multiple organ involvement of the congenital disorder is possible. Furthermore, the survival of adult congenital heart disease (ACHD) patients is improving compared to the past few decades. Therefore, senile or degenerative

diseases common in the normal older population are also presenting in congenital heart disease patients. For example, heart failure with preserved ejection fraction (HFpEF), deep vein thrombosis with pulmonary thromboembolism, degenerative valve disease, coronary atherosclerosis, and chronic obstructive lung disease are all conditions with higher prevalence in the old age population, but the prevalence of these diseases are also high in ACHD patients as the average age of this population is also growing older. HFpEF is the most common mixed cause of pulmonary hypertension in adults, especially in age groups past middle ages. Major risk factors for developing HFpEF include old age, sex (women), and history of hypertension. Atrial fibrillation is a common aggravating factor of HFpEF. Use of loop diuretics is the treatment of choice for HFpEF for symptomatic treatment. When there is a mixed pattern of pulmonary hypertension with suspicious HFpEF in clinical bases (risk factors with pulmonary congestive symptoms), repeated follow-up of pulmonary hypertension after diuresis is helpful to determine the HFpEF associated pulmonary hypertension. When cardiac catheterization is performed to evaluate and diagnose pulmonary hypertension, pre-treatment with diuretics is helpful for

1 PAH 1.1 Idiopathic PAH 1.2 Heritable PAH 1.3 Drug- and toxin-induced PAH 1.4 PAH associated with: 1.4.1 Connective tissue disease 1.4.2 HIV infection 1.4.3 Portal hypertension 1.4.4 Congenital heart disease 1.4.5 Schistosomiasis 1.5 PAH long-term responders to calcium channel blockers 1.6 PAH with overt features of venous/capillaries (PVOD/PCH) involvement 1.7 Persistent PH of the newborn syndrome	3 PH due to lung diseases and/or hypoxia 3.1 Obstructive lung disease 3.2 Restrictive lung disease 3.3 Other lung disease with mixed restrictive/obstructive pattern 3.4 Hypoxia without lung disease 3.5 Developmental lung disorders
2 PH due to left heart disease 2.1 PH due to heart failure with preserved LVEF 2.2 PH due to heart failure with reduced LVEF 2.3 Valvular heart disease 2.4 Congenital/acquired cardiovascular conditions leading to post-capillary PH	4 PH due to pulmonary artery obstructions 4.1 Chronic thromboembolic PH 4.2 Other pulmonary artery obstructions
5 PH with unclear and/or multifactorial mechanisms 5.1 Haematological disorders 5.2 Systemic and metabolic disorders 5.3 Others 5.4 Complex congenital heart disease	

PAH: pulmonary arterial hypertension; PVOD: pulmonary veno-occlusive disease; PCH: pulmonary capillary haemangiomatosis; LVEF: left ventricular ejection fraction.

Figure 1. Updated clinical classification of pulmonary hypertension (PH)(Adapted from Eur Respir J 2019;53(1):1801913.)

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the interpretation of the hemodynamic data. When there are aggravating factors such as renal failure, drugs such as NSAIDs, uncontrolled hypertension or arrhythmia, treatment before cardiac catheterization is recommended before hemodynamic measurements. Lung disease also can be accompanied by ACHD patients. Aggravated lung disease can affect pulmonary vascular resistance and mimic aggravated pulmonary arterial hypertension. Therefore, consultation with pulmonologist about lung disease should be always considered in patients with respiratory symptoms. In situ thromboembolism is relatively common in the dilated pulmonary artery and causes pulmonary thromboembolism with distal embolization. High cardiac output due to anemia or hyperthyroidism can be associated with pulmonary hypertension and should be considered.

Adult Congenital Heart Disease 1 Pulmonary Hypertension in Adult Congenital Heart Disease

» Sunday, Oct 20, 08:40-10:10 AM / Art

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