APHRS Asia Pacific Heart Rhythm Society

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Kiyo Tokyo Building 6F, 2-5 Kanda Ogawamachi, Chiyoda-ku, Tokyo 101-0052
E-mail: office@aphrs.org
TEL: +81-3-3219-1956 FAX: +81-3-3219-1955
www.aphrs.org

CONTENTS

Statin Therapy for Primary Prevention of Atrial Fibrillation

Chen-Ying Hung, MD[1]; Tsu-Juey Wu, MD, PhD[1,2]

Cardiovascular Center, Taichung Veterans General Hospital, Taichung, Taiwan[1]
National Yang-Ming University School of Medicine, Taipei, Taiwan[2]

Atrial fibrillation (AF) is the most common arrhythmia and is associated with increased ischemic stroke, heart failure, health care costs, and all-cause mortality.[1] The overall burden due to AF is likely to increase in coming decades. Therefore, there is an urgent need to implement measurements at the population level to prevent the occurrence of new-onset AF. Upstream therapies, which reverse atrial substrate derangement, could be used for AF prevention. Therefore, the current focus of AF prevention has shifted to these therapies, such as statins, angiotensin-converting enzyme inhibitors (ACEI), and angiotensin receptor blockers (ARB).

An increasing number of clinical studies and animal experiments have investigated the role of statins for AF prevention.[2] Meta-analyses have shown that the use of statins was significantly associated with a decreased risk of this arrhythmia.[3] The 2010 European Society of Cardiology (ESC) guideline suggested that statins could be used for AF prevention in those receiving cardiac surgery or with heart failure.[4] However, whether statins prevent AF in other patient subgroups remains a subject of debate.

While clinical studies yielded conflicting results with respect to the use of statins for AF prevention, meta-analyses revealed that statin therapy is useful for primary prevention of AF.[3] However, there was significant heterogeneity across studies. Therefore, some authors assumed that the AF prevention effect of statins may be diverse in different clinical settings.[5] These findings suggest that underlying co-morbidities may play an important role in selecting suitable patients for statin therapy.[6]

The $CHADS_2$ scoring system, which was initially developed for risk stratification of stroke in patients with AF, is a convenient way to evaluate the complexity of cardiovascular co-morbidities. Recent studies also showed that high $CHADS_2$ scores are associated with electroanatomical remodeling of the left atrium, and an increased risk of new-onset AF.[7] In our recent research, we analyzed data from Taiwan National Health Insurance Research Database and found that $CHADS_2$ score may be of value in identifying the patients who would benefit most from statin use for AF prevention.[8] The nationwide cohort included 27,002 elderly hypertensive patients from Taiwan. It showed that $CHADS_2$ score was useful for predicting the effectiveness of statins. Patients with a $CHADS_2$ score of 1 gained no significant benefits, and those with a $CHADS_2$ score ≥2 had a 31% reduction in risk of AF.[8]

Another study, which included 171,885 Taiwanese patients aged ≥50 years, showed that statin therapy provided the best effect for those with a $CHADS_2$ score of 2 and had no obvious beneficial effect in those with a $CHADS_2$

score of 0.[9] Those with higher $CHADS_2$ score had a higher risk of AF, and gained a greater benefit from statin therapy than those with a lower $CHADS_2$ score. This implies that the $CHADS_2$ score can be used to guide statin therapy for AF prevention. (see Figure 1)

The CHA_2DS_2VASc scoring system was recently developed for stroke risk stratification of AF patients. Our study also showed that patients with a CHA_2DS_2VASc score ≥ 1 benefited from statin use, especially those with score ≥ 3.[9] In contrast, the therapy provided no obvious beneficial effect in those with a CHA_2DS_2VASc score of 0. (see Figure 1) Therefore, $CHADS_2$ and CHA_2DS_2-VASc scores are both useful scoring systems for predicting the effectiveness of statins in AF prevention. However, the exact role of these scores in upstream therapy for AF requires further study.

Statins can act on atrial remodeling by exerting anti-inflammatory and antioxidant effects, modulating endothelial function, and altering ion channel conductance. Clinical studies[10-14] have proved that statin treatment can reduce inflammation, which may explain the potential beneficial effect of statins for AF prevention. Thus, the anti-arrhythmic effect of statins may be more pronounced in patients with more systemic inflammation and damaged atrial tissue. Patients with no systemic inflammation or those with normal atrial substrate are less likely to benefit from statin therapy for AF prevention.

Recent studies demonstrate that $CHADS_2$ score is useful for predicting CRP levels, left atrium thrombus formation, and prognosis in patients with AF.[15,16] The relationship between $CHADS_2$ score and CRP has potential implications for predicting the effect of statin on AF prevention. We propose that those with higher $CHADS_2$ scores have more severe inflammation, and the anti-inflammatory effect of statins may be more obvious in these patients. Furthermore, our recent research showed that $CHADS_2$ and CHA_2DS_2-VASc scores are related to hs-CRP, MCP-1, and VCAM-1 in an incremental manner. Therefore, patients with higher scores may have a more severe inflammation, and the anti-inflammatory effect of statin may be more obvious in these patients.

Previous meta-analyses have shown that statins have a type-dependent efficacy of statin in reducing the risk of new-onset AF.[6] The beneficial effect was noted in the atorvastatin and simvastatin treatment groups, but not in the pravastatin or rosuvastatin groups.[6] One of our recent studies, which included 135,275 patients in Taiwan, showed a difference among statin types. The results revealed that the level of efficacy in reducing the risk of new-onset AF was related to the type of statin.[17] Fluvastatin and pravastatin provided no significant AF risk reduction. Lovastatin demonstrated the strongest AF preventive effect, followed by simvastatin, rosuvastatin, and atorvastatin. (see Figure 2) Other studies also showed similar findings, i.e. atorvastatin was more effective than pravastatin.[18]

The effect of gender was another interesting finding in our recent nationwide cohort

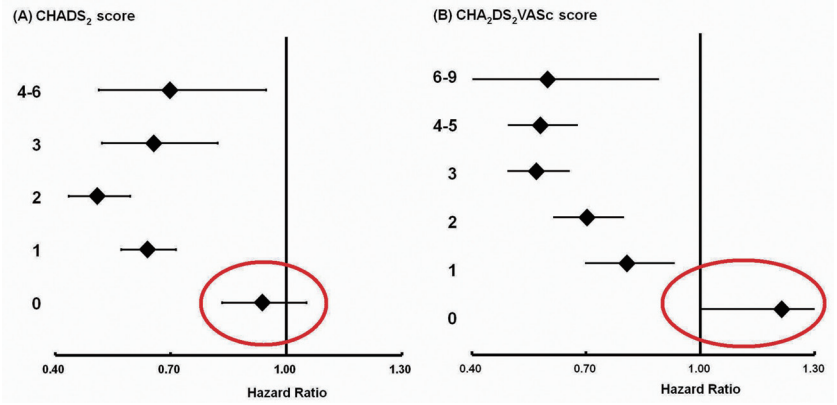

Figure 1. Efficacy of statin for AF prevention according to (A) $CHADS_2$ score, and (B) CHA2DS2VASc score. (Modified from Ref. 9)

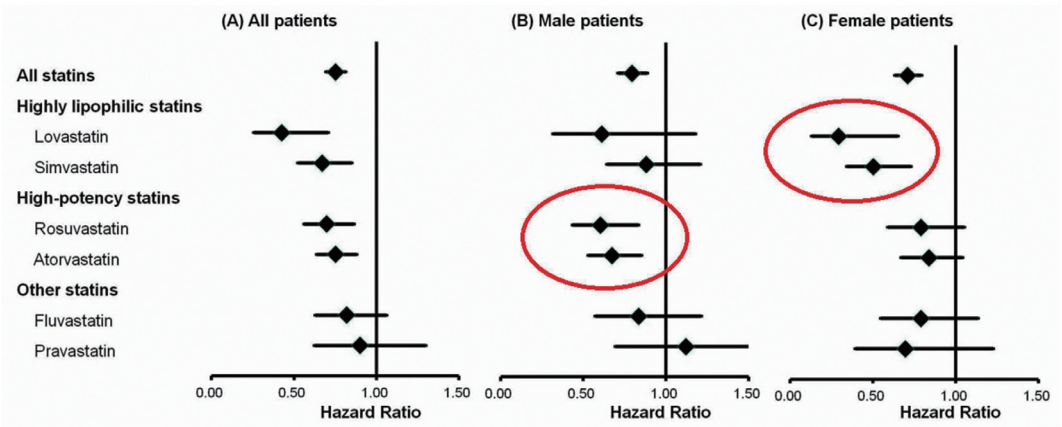

Figure 2. Efficacy of different statins for AF prevention in (A) all, (B) male, and (C) female patients. (Modified from Ref. 17)

study.[17] Male and female patients gained different AF preventive effects depending on the type of statin used. Male patients gained obvious beneficial effects from high-potency statins (rosuvastatin and atorvastatin), whereas female patients gained these benefits from lipophilic statins (lovastatin and simvastatin). (see Figure 2) This finding is comparable to the result of subgroup analysis in the JUPITER trial,[19] which showed that male benefited from the AF preventive effect of rosuvastatin, while females did not. The distinct efficacy of different statins between genders might be attributable to a complex mechanism involving atherosclerotic and inflammatory status.[17] The implications of these findings warrant further investigation.

Another interesting question is whether these co-morbidity scores could be used to predict the effect of other upstream therapies, such as ACEI and ARB. Our recent study, in which we also analyzed data from Taiwan National Health Insurance Research Database, shed light on these treatment effects. We studied 22,324 hypertensive patients in Taiwan aged ≥45 years. The results showed that ACEI and ARB seemed to have a different pattern of efficacy for AF prevention: these therapies were less effective in patients with high $CHADS_2$ score. This finding implies that the main mechanisms of AF prevention by upstream therapies may be different.[20]

Conclusions

Statin therapy was significantly associated with a decreased risk of AF in the selected population. The therapy provided limited benefits in primary prevention of AF in patients with low $CHADS_2$ and CHA_2DS_2-VASc scores. Those with higher $CHADS_2$ and CHA_2DS_2VASc scores will benefit most from statin use for the prevention of AF.[21] Other upstream therapies may have a different pattern of efficacy for AF prevention. The $CHADS_2$ and CHA_2DS_2VASc scoring systems are useful for identifying patients who will benefit most from statins and other upstream therapies. Further studies on the biological mechanisms underlying these effects are needed.

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Address for correspondence: Tsu-Juey Wu, M.D., Ph.D., Director of Electrophysiology, Cardiovascular Center, Taichung Veterans General Hospital, Professor of Medicine, National Yang-Ming University School of Medicine; Tel: 886-4-23592525 Ext: 4042, 4043; Email: tjwu@vghtc.gov.tw

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Optimization of Cardiac Pacing Outcomes by Use of MultiPoint™ Pacing Cardiac Resynchronization Therapy (CRT) Compared with Conventional CRT

A case study from Royal Prince Alfred Hospital, Sydney, Australia

Prof. Ian Wilcox, PhD, Prof. Michael Vallely, PhD
Dr. Michele McGrady, PhD

Introduction

While the 2009 introduction of quadripolar lead technology led to improved acute hemodynamic response to CRT,[1,2] non or low-responder rates still remain a challenge. By providing an additional left ventricular (LV) stimulation vector, MultiPoint™ pacing (MPP) can improve resynchronization and hemodynamic outcomes.[3-5] While the patient in this case had a good clinical response to conventional LV single site CRT in terms of QRS interval reduction and increased ejection fraction, a switch to MultiPoint. pacing improved these outcomes further.

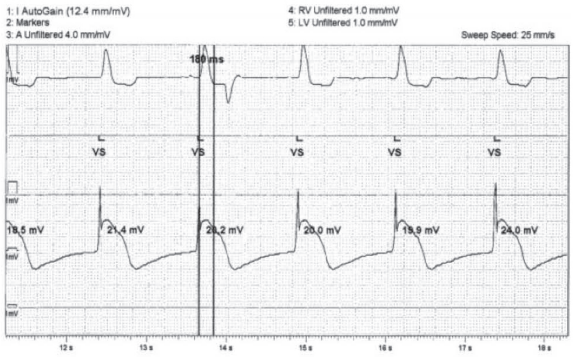
Patient History

- 85 year-old female
- History of coronary artery disease (CAD)
- QRS duration = 180ms
- Left bundle branch block (LBBB)
- Baseline ejection fraction (EF) = 36%
- Heart rate (HR) range 38-89bpm on Holter monitoring

The patient had moderate LV systolic dysfunction with regional variation in contraction probably not entirely attributable to LBBB, but consistent with CAD.

Baseline ECG

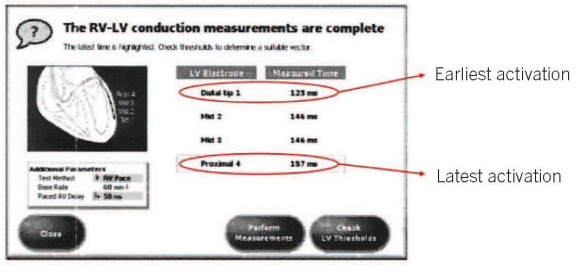
Sinus rhythm, no stimulation QRS = 180ms, 25mm/s



Response to Conventional LV Single Site Pacing

Pacing site	QRS duration (ms)
Right ventricle (RV) paced	168
D1 LV pacing only	180
P4 LV pacing only	191
Simultaneous biventricular pacing at P4	144

RV-VLV conduction measurements (RV pacing):



MultiPoint™ Pacing Therapy

The patient was implanted with a Quadra Assura MP™ CRT-D CD3371-40QC and Quartet™ LV lead 1458Q/86.

Programming

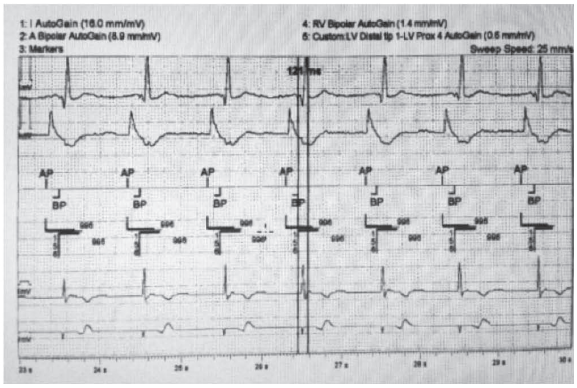
The anatomical method, i.e. selection of the two furthest poles with no phrenic nerve stimulation (PNS) and satisfactory thresholds, was used in this patient.

Two methods were used to determine LV1 and LV2:

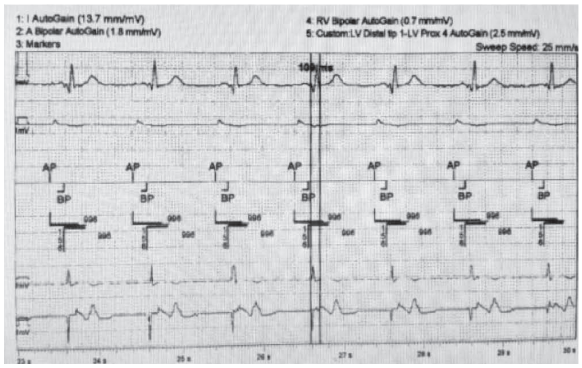
1) latest activation = LV1, and earliest activation = LV2
2) earliest activation = LV1, and latest activation = LV2

	MPP programming (anatomical method)				
	LV1	LV2	LV1-LV2	LV1-RV	QRS
Prog. 1	P4 to RVC (latest)	D1 to RVC (earliest)	5ms	25ms	121ms
Prog. 2	D1 to RVC (earliest)	P4 to RVC (latest)	5ms	25ms	109ms

Program 1: QRS = 121ms



Program 2: QRS = 109ms



Improved Hemodynamic Outcomes with MultiPoint™ Pacing Therapy

Each MultiPoint™ pacing configuration (vectors and timing) provided improved electrical synchronization (assessed by QRS width) versus RV only, LV only and simultaneous RV-VLV stimulation.

In this case, programming using the shortest delay between LV1 and LV2 (5ms) produced incremental benefit for the patient compared with traditional CRT.

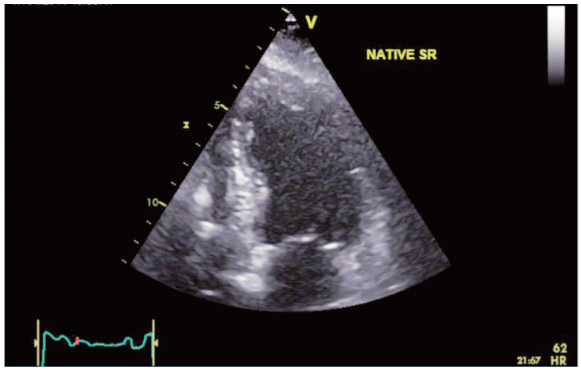
Ventricular Remodeling following Implantation

The patient returned for echo optimization of CRT at 3 months following implantation and turning on of MultiPoint™ pacing. At this visit her intrinsic (unpaced) EF was found to have increased from pre-implantation baseline value (36%) to 39%, suggesting that some remodeling may have already taken place.

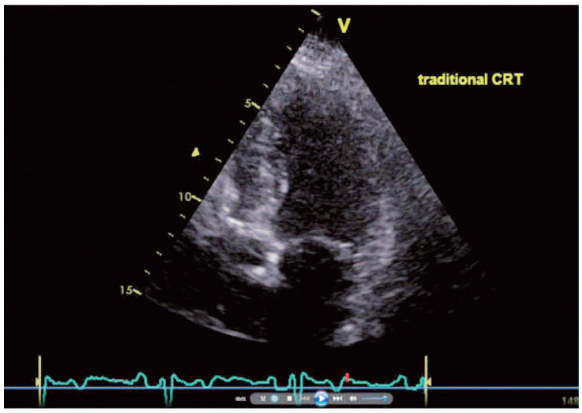
	Ejection fraction (%)	Percentage increase (%) compared with baseline
Baseline	36	
Intrinsic (unpaced) at 3 months	39	8
Traditional CRT	49	36
MultiPoint™ pacing	62	72

Echo imaging

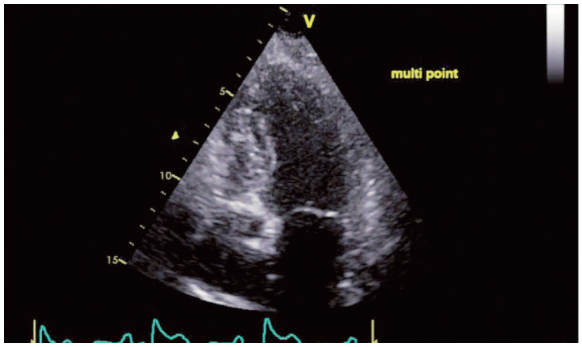
Baseline echo: QRS = 180ms; EF = 36%



CRT echo: QRS = 140ms; EF = 49%



MPP CRT echo: QRS = 109ms; EF = 62%



Conclusions

Developments in MultiPoint™ pacing programming have provided multiple options, not currently available with traditional CRT, which can help improve patient outcomes. This case study demonstrates that MultiPoint™ pacing can offer a significantly improved acute hemodynamic response to CRT, compared with traditional single site left ventricular pacing.

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ECG Quiz

The model commentary will be provided in the next issue No.21

Masahiko Fukatani, MD

Director, Division of Cardiology, Department of Internal Medicine
Chikamori Hospital, Chikamori Medical Group, Kochi, JAPAN

A 59 year-old male was pointed out an electrocardiographic tachyarrhythmia at the admission because of acute renal failure, hyperkalemia, dehydration and diabetes mellitus. The same tachyarrhythmia was persistent during these 9 months. The 12 leads ECG before ablation is shown below (Fig.1).

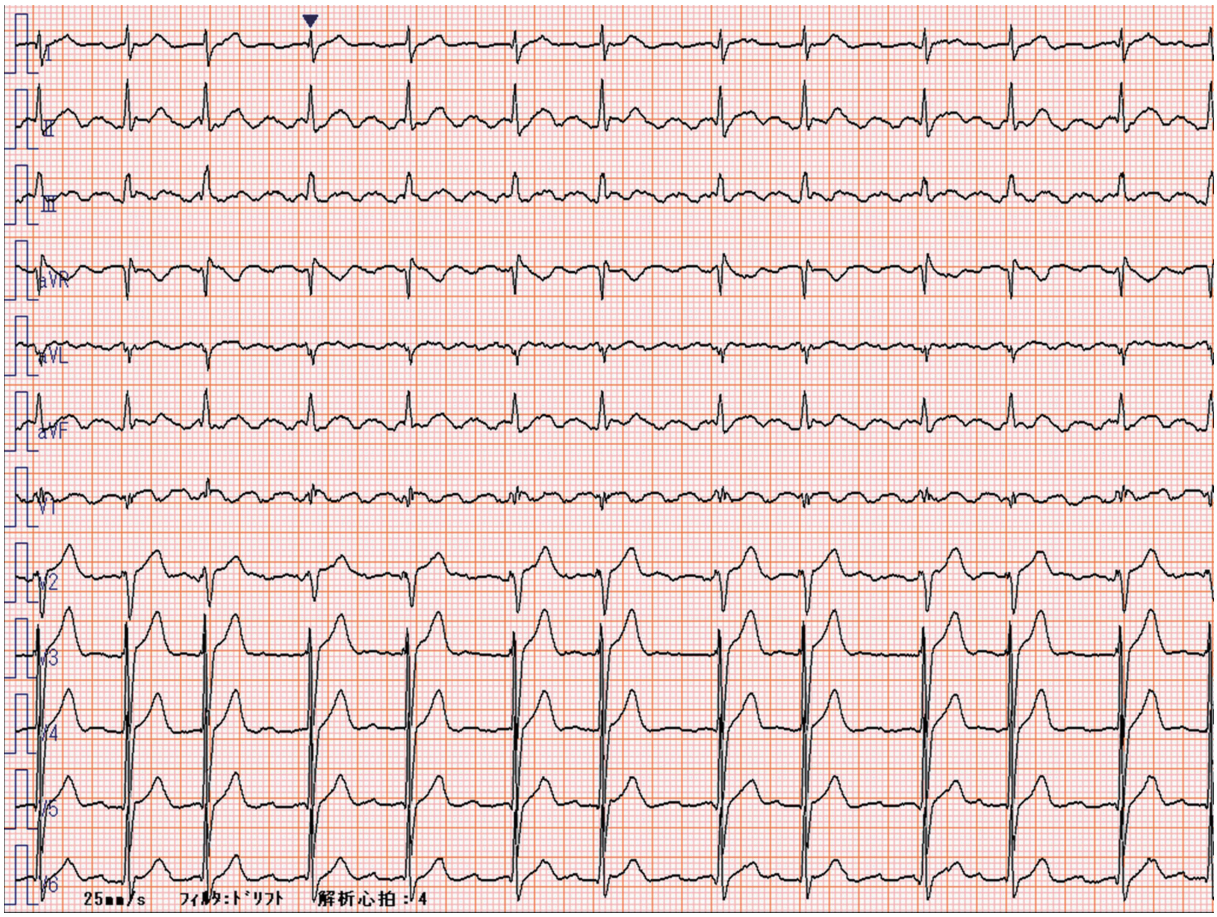

Fig. 1. Twelve leads ECG

Question: What is the diagnosis of this ECG ?

1. Focal Left Atrial Tachycardia
2. Peri-Mitral Atrial Tachycardia
3. Typical Atrial Flutter (Right atrium, CTI dependent, CCW)
4. Reversed Typical Atrial Flutter (Right atrium, CTI dependent, CW)

CTI = cavotricupid isthmus
CCW = counterclockwise
CW = clockwise

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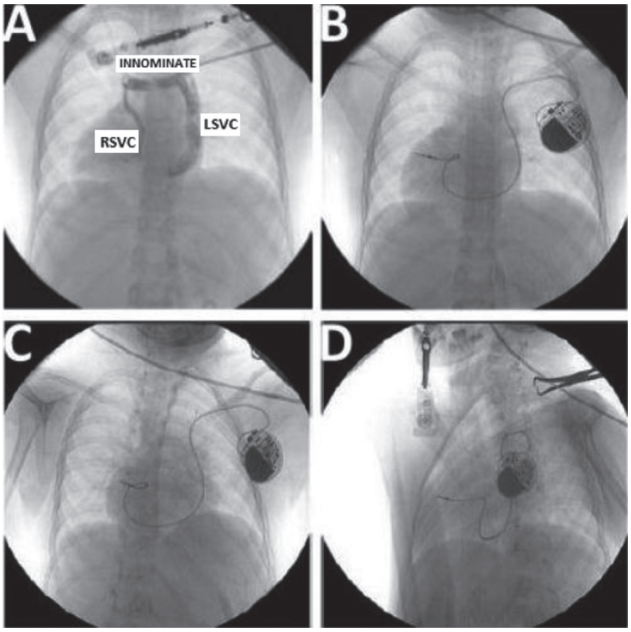
EP Image: RVOT Pacing in Dextrocardia with Persistent Left Superior Vena Cava

Yoga Yuniadi, Erika Maharani

Department of Cardiology and Vascular Medicine, Faculty of Medicine, University of Indonesia and National Cardiovascular Center Harapan Kita, Jakarta

A 9-year-old female child, weighing 20 kilogram, was admitted to the hospital for ventricle septal defect (VSD) clossure. During operation procedure the surgeon found bilateral superior vena cava with the present of innominate vein. Right SVC was small and left SVC was big and drained into coronary sinus. Small left IVC went to coronary sinus and right IVC goes to the middle of right atrium. Right and left ventricle were side by side. Clossure of the VSD using patch and repair of the tricuspid valve was done. Permanent pacing was indicated because of persistent atrioventricular block despite administration of methylprednisolon for 2 weeks.



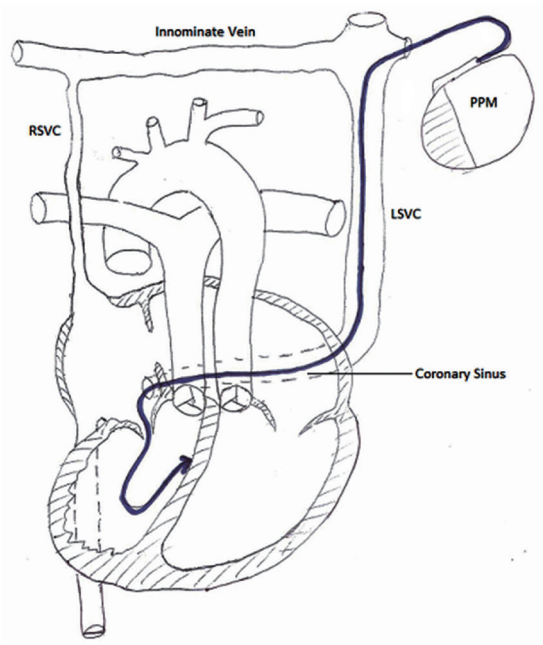


Figure 1. Venography of left subclavian vein persistent left superior vena cava was performed (panel A). It drains to coronary sinus, right superior vena cava was smaller and connect to LSVC trough innominate vein. Active lead position in and anteroposterior view (panel B), right ventricular outflow track (RVOT) from right anterior oblique 30° view (panel C), and left anterior oblique 45° view (panel D).

Address for correspondence: Yoga Yuniadi, Erika Maharani, Department of Cardiology and Vascular Medicine, Faculty of Medicine, University of Indonesia and National Cardiovascular Center Harapan Kita, Jakarta; Email: yogayun@yahoo.com

Seoul St. Mary's Hospital, The Catholic University of Korea

Yong-Seog Oh, M.D., Ph.D.

Division of Cardiology, Department of Internal Medicine, College of Medicine, Seoul St. Mary's Hospital, The Catholic University of Korea

Hospital Overview

The Catholic Medical Center began as St. Mary's Hospital in 1936, with 15 doctors and 24 sickbeds.

Today, it has the best and largest medical network, comprising the School of Medicine and the College of Nursing at the Catholic University of Korea, and eight hospitals including Seoul St. Mary's Hospital.

In 2009, Seoul St. Mary's Hospital reopened its cutting-edge facilities to the public in accordance with its vision of providing "world-class medical services based on a profound respect for human life." This has served to boost collaboration with our affiliated hospitals for future activities. With 22 upper floors, 6 basements and 1320 sickbeds, it is the largest among Korean hospitals consisting of a single building.

Hospital Information

Address : 222 Banpo-Daero, Seocho-Gu,
Seoul 137-71 Korea
Tel: 82-2-2258-5745
No. of beds: 1,335 (including 110 beds in the ICU)
No. of staff: about 4,000
Specialized clinical centers: 21 centers
Parking capacity: 1,562 cars
No. of inpatients: 427,048
No. of outpatients: 1,607,357

Division of Cardiology

Director : Yong-Seog Oh (Professor of Medicine)
No. of Cardiologist: 19
Clinical electrophysiologist: Pf. Yong-Seog Oh
Pf. Sung-Hwan Kim

Access Information

Seoul St. Mary's Hospital is located in central district of Seoul.

Electrophysiology laboratory

The Cardio-cerebro-vascular center at Seoul St. Mary's Hospital is providing medical services for diseases such as coronary heart disease, heart failure (including transplantation), hypertension, valvular disease, structural heart disease including congenital heart disease and arrhythmia, trying to systematically convey professional care of good quality, patient-

oriented medical care, and fast and efficient medical services. There are three labs in our institute of which one room designed to perform diagnostic and therapeutic electrophysiology procedures. This lab is dedicated to catheter ablations with 3D system and remote magnetic navigation (only in the Korea). In 2014, 520 electrophysiological interventions were performed, including 342 catheter ablations (AF 161, PVC/VT 42, including epicardial approach) and 156 device implantations/lead extraction (CRT-D 13, ICD 33). In 2013, we started thoracoscopic AF Ablation and will open hybrid operation room for complex procedures (hybrid AF ablation, lead extraction, subpectoral implantation of device and other interventions for cardiovascular disease).

We have been investigating clinical studies and published every year. In addition, we trigger and lead many multicenter studies.

Address for correspondence: Yong-Seog Oh, M.D., Ph.D., Division of Cardiology, Department of Internal Medicine, College of Medicine, Seoul St. Mary's Hospital, The Catholic University of Korea, 222 Banpo-daero, Seocho-gu, Seoul 137-701, Republic of Korea; Tel: 82-2-2258-5745; Email: oys@catholic.ac.kr

ECG Commentary Related to the Quiz in the No. 19 Issue

Chang-Sheng Ma, MD

Professor of Medicine,Department of Cardiology, Beijing Anzhen Hospital, Capital Medical University, Beijing, China

Answer 1:

3) Focal atrial tachycardia

Answer 2:

2) Right inferior pulmonary vein origin

Answer 3:

1) or 2)

This focal atrial tachycardia originates either from the left atrium close to the previous circumferential lesions around the pulmonary veins (PVs), or from inside or surrounding a pulmonary vein after incomplete linear ablation via a reconnection gap between the left atrium and the PV.

ECG commentary related to the quiz:

The surface 12-lead electrocardiogram (ECG) during the episode of palpitation shows characteristic features of focal atrial tachycardia[1]. The ECG documents an atrial tachycardia with a p-p interval of 240 ms and an atrioventricular conduction ratio of 4:1.The presence of an isoelectric interval separating the P waves basically excluded macro-reentrant ATs[2].

Analyses of P wave morphology is highly useful in determining the likely site of origin of focal atrial tachycardia and has been described in detail elsewhere[3]. For this patient, positive P wave in lead V1 and I has a reasonably high predictive accuracy for localizing atrial tachycardia that originates from a right-sided pulmonary vein. Negative P wave in lead III and aVF indicates that this tachycardia originate from inferior atrium. This patient underwent a repeated electrophysiological study and catheter ablation. Activation map of the left atrium using electro-anatomical mapping (CARTO 3) during the tachycardia confirmed that this atrial tachycardia originates from the right inferior pulmonary vein using a reconnection gap. Single radiofrequency application (the red dot) at the earliest activation site terminated this tachycardia. Atrial tachycardia following atrial fibrillation ablation is often significantly symptomatic and requires repeated catheter ablation.

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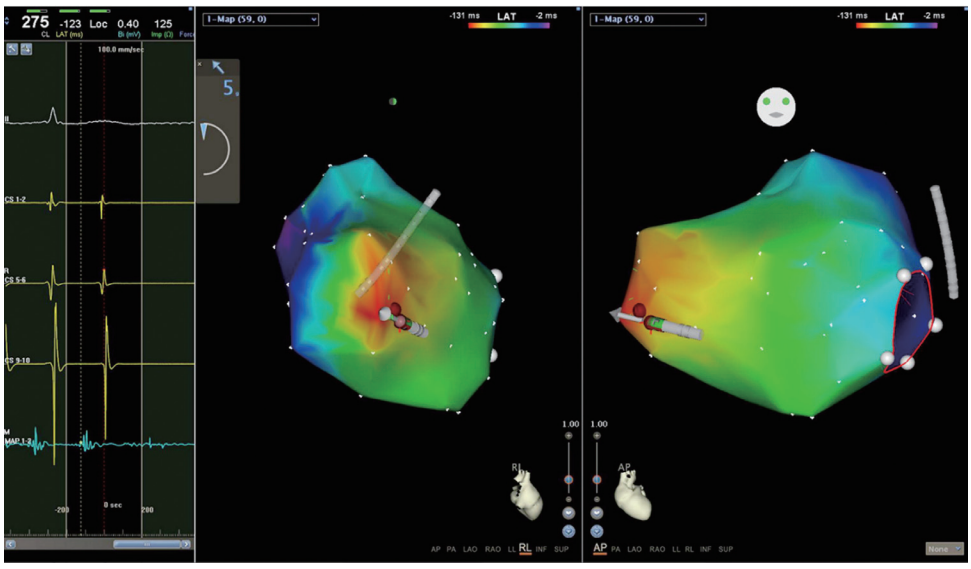

Fig. 1. Activation map of the left atrium during the tachycardia

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