APHRS NEWSLETTER

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Contents

LEFT BUNDLE BRANCH PACING IN A PATIENT WITH FABRY DISEASE ASSOCIATED HYPERTROPHIC CARDIOMYOPATHY, AND ADVANCED ATRIOVENTRICULAR BLOCK: A CASE REPORT

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Introduction

Fabry disease is an X-linked lysosomal storage disorder resulting from deficient α-galactosidase A activity, causing accumulation of globotriaosylceramide within lysosomes of multiple cell types [1]. Cardiac involvement, including left ventricular hypertrophy (LVH), valvular thickening, conduction abnormalities, and arrhythmias, is a major determinant of prognosis [1][2].

Conduction system disease in Fabry cardiomyopathy arises from glycosphingolipid deposition and myocardial fibrosis involving the atrioventricular(AV) node, His bundle, and Purkinje system [2]. Permanent pacing is frequently required for advanced AV block. However, right ventricular apical pacing (RVP) can produce ventricular dyssynchrony, leading to pacing-induced cardiomyopathy and heart failure, especially in patients with preexisting LVH [3].

Left bundle branch pacing (LBBP) has recently gained attention as a physiologic pacing method capable of recruiting the intrinsic conduction system, achieving near-normal QRS morphology and synchronous activation [4]. We report a patient with Fabry disease-associated hypertrophic cardiomyopathy (HCM) and high-degree AV block who underwent successful LBBP implantation, demonstrating both procedural and physiologic success.

Case Presentation

A 63-year-old man with genetically confirmed Fabry disease-associated HCM presented with fatigue and recurrent syncope.

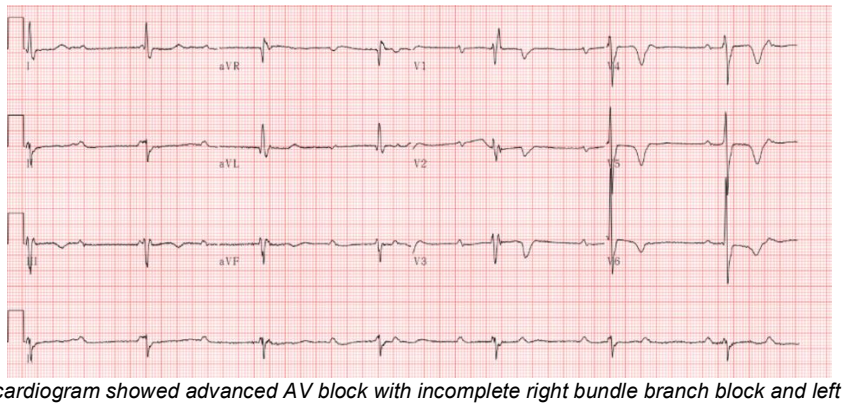

Figure 1. 12-lead electrocardiogram showed advanced AV block with incomplete right bundle branch block and left anterior fascicular block.

On admission, his 12-lead electrocardiogram (ECG) showed advanced AV block with incomplete right bundle branch block and left anterior fascicular block (Figure 1). Echocardiography revealed marked concentric LVH with preserved LV systolic function (LV ejection fraction: 53%) and impaired global longitudinal strain of −8.26%; no LV outflow tract obstruction was noted. Cardiac magnetic resonance demonstrated marked LVH and global low T1 value at all segments of LV, which were compatible with cardiac Fabry disease. In addition, focal mid-wall and epicardial late gadolinium enhancement patterns at basal anteroseptal and basal inferoseptal walls were detected.

Due to advanced AV block and recurrent syncope, we proceeded to pacemaker implantation. The procedure began with antiseptic preparation of the parasternal and left chest wall. A small incision below the left clavicle exposed the left cephalic vein via surgical cut-down, establishing vascular access for lead placement. A C315 delivery catheter (Medtronic, Mineapolis, MN) was advanced into the RV high septum to facilitate LBBP using a SelectSecure™ Model 3830 pacing lead (Medtronic, Mineapolis, MN).

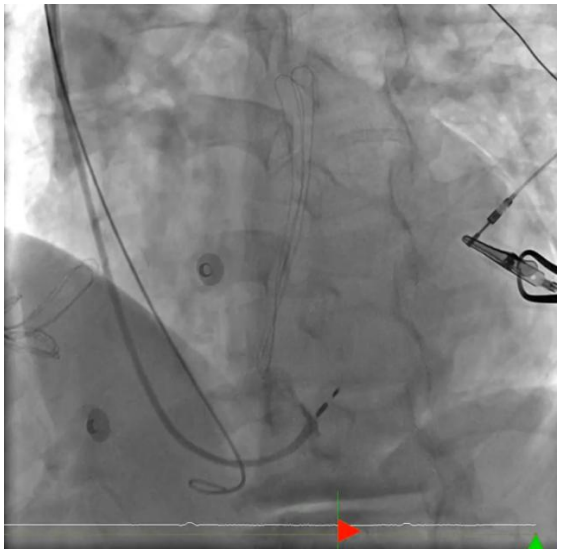

Figure 2. Cardioangiography confirmed the precise location of the pacing lead's tip and ring electrodes, demonstrating their spatial relationship to the interventricular septum and proper alignment within the target region of the left bundle branch conduction system.

The procedure was guided by both electrophysiological and imaging methods to ensure optimal lead positioning. Specifically, cardioangiography was employed to confirm the precise location of the pacing lead's tip and ring, allowing accurate visualization of their relationship to the septal wall and ensuring that the lead was properly aligned within the target region of the LBB conduction system (Figure 2). The current of injury (COI) was also monitored as an electrophysiological marker to assess the depth of lead penetration into the septal myocardium, ensuring that the lead reached the appropriate depth to achieve conduction system capture without excessive intrusion that could risk perforation (Figure 3).

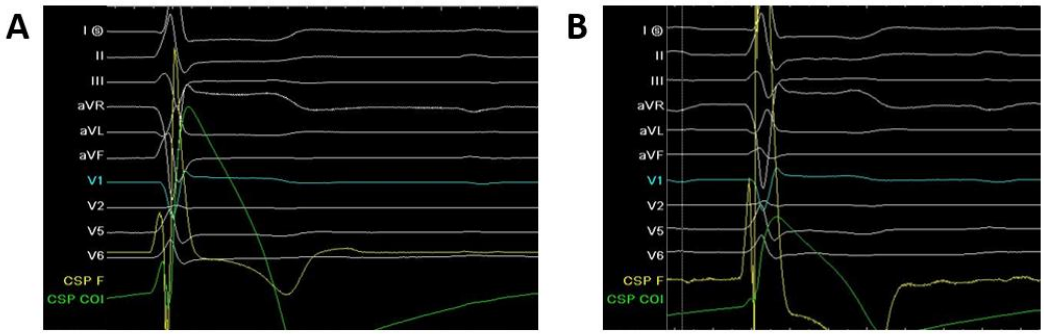

Figure 3. Monitoring of the current of injury (COI) could serve as an electrophysiological marker to assess the depth of lead penetration into the septal myocardium. From A to B, a sudden decrease in tip COI was observed, indicating loss of myocardial engagement and confirming adjustment of lead depth to optimize conduction system capture while avoiding septal perforation.

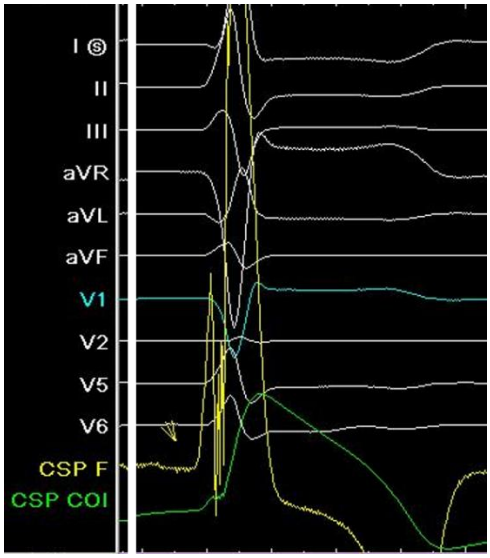

Figure 4. Distinct left bundle branch potential was recorded, confirming adequate engagement of the conduction system.

After one optimal attempt at lead deployment, successful LBBP was achieved, characterized by a distinct left bundle branch potential (Figure 4), a LV activation time (LVAT) of 64 milliseconds. The pacing QRS duration measured 118 milliseconds (Figure 5), confirming effective conduction system engagement and physiological ventricular activation.

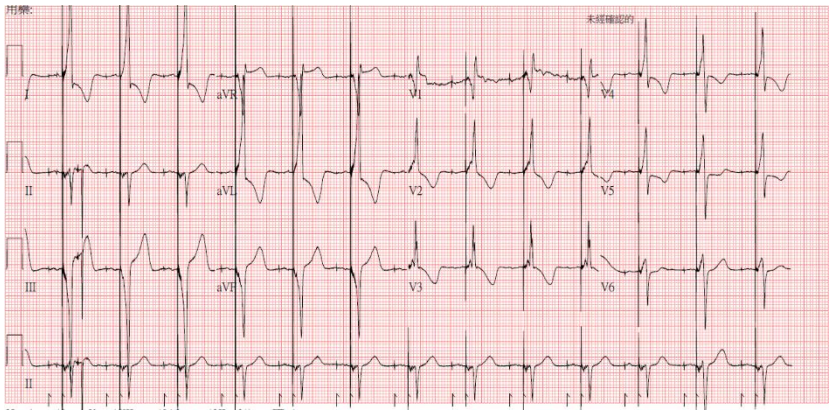

Figure 5. Post-implantation electrocardiogram demonstrated left bundle branch pacing.

Final intraoperative testing confirmed optimal lead parameters: the atrial lead showed a P wave amplitude of 1.0 mV, impedance of 532 ohms, and threshold of 0.75 V at 0.4 ms, while the LBBP ventricular lead demonstrated superior electrical performance with a R wave amplitude of 20 mV, impedance of 665 ohms, and a low threshold of 0.25 V at 0.4 ms.

Discussion

In this case, successful LBBP implantation was achieved in a patient with Fabry disease-associated HCM and and advanced AV block. Cardioangiography allowed precise visualization of the pacing lead's relationship to the interventricular septum, confirming optimal depth and orientation of the tip and ring electrodes. Concurrent monitoring of the COI provided a real-time electrophysiologic marker of lead penetration, ensuring conduction system capture while minimizing the risk of septal perforation.

These techniques improved procedural accuracy and safety in the setting of marked septal hypertrophy and fibrosis where conventional guidance may be inadequate [5]. The resulting physiologic pacing pattern demonstrated effective recruitment of the conduction system, narrow QRS morphology, and stable pacing thresholds, suggesting preserved ventricular synchrony and reduced risk of pacing-induced dysfunction.

In summary, this case highlights the feasibility and clinical advantage of LBBP in Fabry cardiomyopathy with conduction disease. Integrating cardioangiography and COI monitoring enables precise septal lead deployment, ensuring durable and efficient pacing. LBBP may thus represent a preferred pacing strategy for Fabry patients requiring permanent pacing, offering a physiologic alternative that maintains electrical synchrony and potentially mitigates the progression of Fabry-related cardiomyopathy.

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IMPACT OF IVABRADINE ON ATRIAL ARRHYTHMIA AND CHRONIC KIDNEY DISEASE IN HEART FAILURE: A NARRATIVE REVIEW

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Introduction

Heart failure (HF) is frequently complicated by atrial arrhythmias and progressive renal dysfunction, both of which contribute to adverse cardiovascular outcomes and increased mortality. Elevated heart rate is an independent risk factor for both HF progression and renal impairment. Ivabradine, by selectively inhibiting the If current in the sinoatrial node, lowers heart rate without affecting contractility or blood pressure. Its clinical benefits in reducing HF hospitalization and improving cardiac function have been demonstrated in pivotal trials such as SHIFT.

However, ivabradine has been reported to increase the risk of atrial fibrillation (AF) in some meta-analyses and observational studies. On the other hand, improved hemodynamics and renal perfusion associated with heart rate reduction may have protective renal effects. The interplay between ivabradine, atrial arrhythmia (AA), and Chronic Kidney Disease (CKD) remains incompletely understood.

Mechanistic Considerations

Ivabradine and Atrial Electrophysiology

Ivabradine acts at the sinoatrial node, but indirect effects on atrial substrate are possible. In patients with HF, atrial remodeling involves calcium handling dysregulation, fibrosis, and increased automaticity, especially from pulmonary vein myocardial sleeves. While earlier analyses suggested a higher AF incidence with ivabradine, this may reflect unmasking of pre-existing substrate rather than a direct proarrhythmic effect. Conversely, in patients without prior arrhythmia, ivabradine-induced heart rate control may reduce atrial stretch and neurohumoral activation, potentially delaying atrial remodeling and lowering the incidence of new-onset AA.

Ivabradine and Kidney Function

Advanced CKD alters atrial electrophysiology, increasing AF susceptibility via disrupted calcium homeostasis and structural remodeling. Moreover, tachycardia itself is a predictor of renal function decline. Ivabradine may mitigate renal deterioration through improved hemodynamics, decreased sympathetic drive, and better renal perfusion. However, its role in early versus advanced CKD remains to be clarified.

Clinical Evidence

Concerns about atrial arrhythmia (AA) and its link to CKD remain unresolved. Our study has drawn on data from Taiwan's National Health Insurance Research Database (NHIRD). We evaluated whether ivabradine use in heart failure (HF) is associated with AA and renal outcomes using Taiwan's NHIRD. Adults with HF with reduced ejection fraction were 1:1 propensity-matched (ivabradine vs no ivabradine) and patients with arrhythmia in the preceding 2 years were excluded. We analyzed 6,284 patients with HF (ivabradine users n=3,142; non-users n=3,142) with follow-up through 2021. During follow-up, ivabradine users had a lower incidence of AA than controls (7.5% vs 10.0%, p=0.001), with no difference in SSS (1.8% vs 1.7%, p=0.847). Incident CKD was comparable between groups (19.8% vs 19.1%, p=0.503), whereas progression to advanced CKD was less less frequent with ivabradine (≈1% vs 2.0%, p<0.001). Moreover, the hazard ratio for the incidence of AA, SSS, and CKD are presented in Table 1.

Event outcome	Number	Crude hazard ratio (95% confidence interval)	*p*
Nonadvanced CKD	1222 (19.4)	1.06 (0.947–1.186)	0.3093
Advanced CKD	90 (1.4)	0.427 (0.272–0.67)	0.0002
Atrial arrhythmia*	551 (8.8)	0.753 (0.636–0.892)	0.0010
Sick sinus syndrome	109 (1.7)	1.057 (0.725–1.542)	0.7733

*Table 1. Effect of ivabradine on cardiorenal event risk, Advanced CKD: CKD requiring erythropoietin therapy or dialysis. Nonadvanced CKD: CKD not requiring erythropoietin therapy or dialysis. *Including atrial fibrillation and atrial flutter.*

In our real-world cohort, patients treated with ivabradine were found a significantly lower incidence of AA (p<0.001), no between-group difference in SSS, no benefit in non-advanced CKD, but a markedly lower risk of progression among those reaching advanced CKD thresholds (p<0.001). Further, better clinical outcomes over time such as AA or advanced CKD in HF patients with ivabradine usage are summarized in Figure 1.

In contrast to prior meta-analyses and Taiwanese observational study linking ivabradine to higher AF risk, our exclusion of patients with recent arrhythmia targeted relatively intact atria, likely explaining the divergence and suggesting a preventive effect on AA when initiated before marked atrial remodeling. Ivabradine reduced the risk of advanced CKD requiring erythropoietin therapy or dialysis but not with a reduced risk of early CKD. These findings suggest a preventive effect on AA and reduced risk of advanced CKD progression in HF patients initiating ivabradine therapy early, before significant atrial or renal damage occurs.

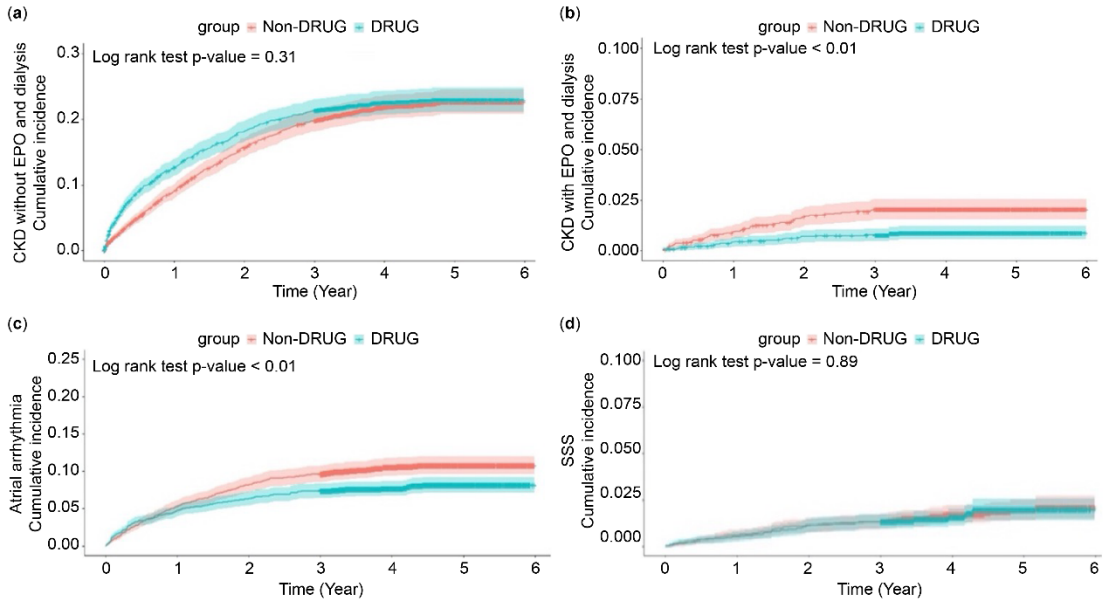

Figure 1. Kaplan–Meier survival curves of cumulative incidence of various conditions in patients with heart failure. (A) Nonadvanced CKD: CKD not requiring erythropoietin therapy or dialysis. (B) Advanced CKD: CKD requiring erythropoietin therapy or dialysis. (C) Atrial arrhythmias. (D) Sick sinus syndrome. Abbreviations: CKD, chronic kidney disease. *Including atrial fibrillation and atrial flutter.*

Comparison with Prior Studies

Previous meta-analyses (e.g., Swedberg et al., 2010; Fox et al., 2014) and Taiwanese observational data linked ivabradine to increased AF incidence, especially in older patients or those with pre-existing arrhythmic substrates In contrast, the NHIRD cohort excluded patients with prior arrhythmia, potentially explaining the divergent findings and suggesting a timing effect: ivabradine may prevent AA when started early but unmask arrhythmia

when used in patients with pre-existing substrate.

Regarding renal outcomes, clinical data have been limited. The NHIRD study is among the few to show a significant reduction in progression to advanced CKD with ivabradine, likely mediated by heart rate reduction and improved hemodynamics rather than direct nephroprotective mechanisms.

Clinical Implications

1. Patient selection matters – ivabradine may lower AA risk when initiated in patients without prior arrhythmia but could increase AF risk if atrial remodeling is advanced.

2. Renal protection may occur at later CKD stages – suggesting benefits in delaying progression to dialysis or erythropoietin therapy.

3. No increase in SSS – indicating ivabradine does not cause clinically significant sinus node dysfunction in this Population.

Conclusion

Ivabradine is associated with a lower incidence of atrial arrhythmias and progression to advanced CKD in heart failure patients without prior arrhythmia or CKD, without increasing the risk of sick sinus syndrome. These findings highlight the potential preventive role of early ivabradine initiation on atrial and renal outcomes in HF. Further randomized studies are warranted to validate these observational findings and explore underlying mechanisms.

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THE ELECTROPHYSIOLOGY TEAM AT GLENEAGLES JPMC, BRUNEI DARUSSALAM

Written by: Sofian Johar, MD

Brunei Darussalam is a country which lies on the north coast of Borneo Island in South-East Asia. It became independent from the United Kingdom in 1984 and has universal healthcare for its citizens and residents. Brunei has a population of just over **450,000** people. The country is divided into 4 districts, with most of the population living in the Brunei Muara district. It has a GRP per capita of over USD 30,000 with most of its revenue coming from the oil and gas industry. It has undergone a shift in the leading causes of death from communicable diseases to non-communicable diseases over the last few decades, with the top 2 leading causes of death being **cardiovascular disease** and **cancer**.

Despite being relatively affluent, the overall mortality rate for cardiovascular disease remains high, at **224.3 per 100,000** of the population according to WHO figures in 2020. Therefore, there have been great efforts to try and improve the situation by the Ministry of Health with a comprehensive primary healthcare system to promote early detection of hypertension, diabetes and hypercholesterolemia, with medication essentially free for most of the population as well as providing free investigations and treatment in secondary and tertiary care cardiology.

Figure 1. The team performing the first leadless pacemaker implantation in Brunei Darussalam

Gleneagles JPMC is a tertiary care cardiology hospital setup as a joint venture between the Government of Brunei, represented by Jerudong Park Medical Centre and Parkway Holdings Limited and was incorporated on **15 July 2002**. The **first coronary angiogram and stenting procedures**, and **coronary artery bypass grafting** cases were done in 2003. The first **cardiac electrophysiology** cases were performed by visiting specialists in 2005 which were mostly biventricular ICDs, SVT procedures and diagnostic studies. **Surgical AF ablation** also commenced in 2006. The centre now offers the full range of cardiac services such as a nationwide primary PCI service with direct ambulance transfer by paramedics, complex PCI, access to imaging such as OCT and IVUS; TAVI, Mitraclip and other structural heart interventions; minimally invasive cardiac surgery, ECMO, Impella, and LVAD implantation. The centre currently has **2 catheter labs**, with 1 lab being used for cardiac electrophysiology procedures.

In 2012, the first **left atrial appendage occlusion** procedure was performed and in 2013, the first **3D mapping system** was installed. This was a CARTO 3 installation equipped at the time with contact force sensing technology which was one of the first on the island of Borneo. The first **AF ablation procedure** was carried out on 19 May 2013 by a local cardiac electrophysiologist and since then, the electrophysiology section has gone from strength to strength. For example, the first **leadless pacemaker** was implanted on 22 June 2015, and after Institut Jantung Negara in Malaysia, Brunei **became one of the first countries in South-East Asia** to have access to this device.

Brunei's first heart operation using 3D mapping

MONDAY, MAY 20, 2013

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Dr Sofian DP Dr Johar, electrophysiologist from RIPAS Hospital speaking to the media about Brunei's first atrial fibrillation ablation treatment using 3D-mapping equipment, CARTO3, while holding a miniature model of a heart, at Gleneagles JPMC in Jerudong. Picture: BT/Ubaidillah Masli

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Figure 2. News article about the first atrial fibrillation ablation procedure in Brunei Darussalam

Gleneagles JPMC currently offers the full spectrum of cardiac electrophysiology services and procedures with over **100 electrophysiology procedures annually**, with atrial fibrillation ablation, ventricular ectopic ablation and supraventricular tachycardia ablations being most common. The centre recently acquired **pulsed field ablation (PFA) technology** and now atrial fibrillation ablation is almost exclusively carried out with PFA. **Conduction system pacing** (initially His bundle pacing and now left bundle branch area pacing) is carried out regularly. **Subcutaneous ICDs** are also performed frequently and the centre is considering the introduction of the **extravascular ICD**. **Lead extractions** are performed using the mechanical dilator sheath. Watchman FLx devices are used for left atrial appendage occlusion. Intracardiac echo and CARTOSound are used for difficult electrophysiology cases.

It is hoped that the cardiac electrophysiology service in Brunei Darussalam will continue to develop and access to cardiac electrophysiology by the population seems to be reasonable, based on data from the APHRS White Book, certainly when compared to other countries in the region. Nonetheless, **challenges remain**, with the service currently being provided by a **limited number of doctors** and we hope to **train more electrophysiologists and device physicians** in the future.

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■ Plenary Session

13 November 10:20am ~ 11:50am

Moderator: ① Wataru Shimizu, ② Hui-Nam Pak, ③ Mina Chung
Speaker: ① Josep Brugada, ② Young-Hoon Kim, ③ Roderick Tung

■ Late Breaking Trial Session

Session 1 Room 2 12 November 12:40pm ~ 1:50pm
Session 2 Room 1 13 November 2:50pm ~ 4:00pm
Session 3 Room 1 14 November 9:50am ~ 11:00am
Session 4 Room 1 15 November 3:40pm ~ 4:50pm

■ Joint Symposium

WSA-APHRS Joint Symposium Room4 13 November 8:30am ~ 9:40am
Cardiac Electrophysiology - Next 10 years

EHRA-APHRS Joint Symposium Room4 13 November 2:50pm ~ 4:00pm
CIEDs – A beginning of a new era?

LAHRS-APHRS Joint Symposium Room4 13 November 5:30pm ~ 6:40pm
New frontiers at device indications and implantation to avoid desynchrony

HRS-APHRS Joint Symposium Room4 14 November 2:40pm ~ 3:50pm
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