APHRS Asia Pacific Heart Rhythm Society

News
Sep. 2014, No. 15

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Welcome Address to APHRS 2014!

Young-Hoon Kim, MD
President, APHRS

Dear APHRS members,

It will be a great pleasure for me to meet you again at the APHRS 2014 Scientific Session, which will be held in New Delhi, India. I would like to extend my deepest gratitude to Dr. Mohan Nair and other key members of the Indian Heart Rhythm Society, as well as APHRS Scientific Committee members including Dr. Jon Kalman, chairman of the committee for their dedicated efforts to make the utmost preparations for this year's APHRS.

The APHRS has positioned itself as a solid international organization by gaining a wide-ranging foothold after its inception in 2008. The Scientific Session 2014 will serve as a significant venue to bring together world-renowned experts and professionals in the field of arrhythmia to discuss our future. Notably, we have invited many scholars and clinicians who have produced a high level of achievements over the past year to act as our speakers at this year's Scientific Session; indeed, we will have their in-depth and well-prepared presentations delivered in an efficient and organized manner.

Also, we will offer each and every participant opportunities for the intimate interaction and networking with guru experts in different parts of this field. The APHRS is not just a body to prepare for the hosting of an annual scientific session but it is also designed to establish infrastructure for basic and clinical researches on arrhythmia in Asia-Pacific countries, provide systematic educational opportunities for young researchers and clinicians wanting to specialize in this field, and to promote multinational researches. The scientists and physicians in the field of arrhythmia are who have courage, vision, creativity, and determination to challenge the existing order and system.

The APHRS would like to encourage its members to submit their remarkable papers to "Journal of Arrhythmia," which is the official journal of the APHRS. Now, we are putting

our utmost effort to make this journal an internationally renowned one by appointing Dr. Shih-Ann Chen as its editor-in-chief who will work as co-editor with Dr. Aonuma Kazutaka from Japanese Heart Rhythm Society. Indeed, we will do everything within our capacity to make our journal a SCI-registered one within one to two years so that your researches could be shared more widely. Again, please submit your papers and thank you in advance for your kind attention to our activities and journal.

Furthermore, the APHRS is dedicated to building a regional database system by supporting the registry/joint clinical study for atrial fibrillation, inherited arrhythmia and sudden cardiac death, etc., between its member countries. To that end, we are operating a subcommittee under the control of APHRS. And we will renovate website, as well as with the development of web-based educational programs and various educational courses to make sure that participants get more opportunities for education and exchange information.

In cooperation with HRS and EHRA, we are pushing forward with the program for better training and education in the arrhythmia field for young fellows. In 2015, we will run a new program to give an educational opportunity to two to three fellows seeking to get a training course in member countries. We invite you to apply for this program.

Even though we still have a long way to go before achieving our goals, we have equipped ourselves every year to emerge as one of the world's top three heart rhythm societies thanks to your dedication and commitment. We will do our best to provide genuine support and help for you to achieve great results in your research and clinical projects.

I look forward to seeing all of you once again at APHRS 2014 to be held in New Delhi which is a very beautiful and attractive city with a long tradition and history.

Thank you.

Program-at-a-glance

Time	29th October 2014	30th October 2014	31st October 2014	1st November 2014
Morning	**Pre-Congress Symposium & Workshop**	Plenary Session Breakout Session Free Paper	Plenary Session Breakout Session Free Paper Best Oral Presentation Young Investigator Award	Plenary Session Breakout Session
Afternoon		Lunch Symposium Free Paper Plenary Session/CD Presentation/ Live Transmission Tea Break Women in EP program Breakout Session	Lunch Symposium Plenary Session Tea Break Breakout Session	Lunch Symposium Plenary Session/CD Presentation/ Live Transmission
Evening		Sponsored Symposium Faculty Dinner	Sponsored Symposium Gala Dinner	

APHRS
The 7th Asia-Pacific Heart Rhythm Scientific Session
Oct 29th to Nov 1st 2014, New Delhi, India
APHRS 2014 7th

Beating Heart of Vibrant India
Oct 29th to Nov 1st 2014

In conjunction with '10th Asia-Pacific Atrial Fibrillation Symposium'

Dear Colleagues and Friends,

It is our great pleasure to welcome you to the 7th Asia Pacific Heart Rhythm Society Scientific Session (APHRS 2014) scheduled to take place from October 29 to November 1, 2014. This year's conference is being jointly organized by the Asia Pacific Heart Rhythm Society (APHRS) and the Indian Heart Rhythm Society (IHRS) in the effervescent capital of India, New Delhi.

To uphold excellence and advancement in the diagnosis and treatment of patients with heart rhythm disorders, Asia Pacific Heart Rhythm Society has been hosting these conferences since 2008 in various regions of Asia. Not only has each Conference been outstanding from a scientific perspective, but exposure to the local culture has been rewardingly educational and deeply enriching as well.

The conference will feature more than 200 top-notch scientific sessions including Symposia, panel discussions and debates on such key areas as ECG's, Syncope, Sudden Cardiac Death, Atrial Fibrillation and Heart Failure. The 7th APHRS, 2014 is sure to be a powerhouse of creative and inspirational ideas, providing innovative educational experiences and highlighting the field of critical care for patients with heart rhythm disorders along with most up-to-date evidence-based developments in electro-cardiology.

World renowned thought leaders and nearly 250 core faculty from across the globe will bring in unparalleled diversity of perspectives at this one of the largest meeting of scientists, clinicians and allied professionals involved in the field of Arrhythmia and Electro-Cardiology.

Anticipation and excitement for this year's conference are high, and we hope you will take advantage of all that APHRS 2014 has to offer. Be sure to visit the Exhibition area which is a relaxed space designed to facilitate networking, knowledge sharing and conversation with representatives of internationally acclaimed companies displaying new tools and future developments.

We owe special thanks to the Heart Rhythm Society, European Heart Rhythm Association and the Cardiology Society of India for endorsing APHRS 2014.

New Delhi, where legend, lore, history and modernity are wrapped together in a unique amalgam will offer an inspirational and friendly atmosphere for our discussions and plenty of opportunities for leisurely and stimulating explorations. The city will be waiting with its thousand fascinating faces to make us feel at home, ready to renew old friendships and start new ones.

We are looking forward to greet you personally at 7th Asia Pacific Heart Rhythm Society Scientific Session in New Delhi!

Cordially yours,

Mohan Nair
India
Chairman, Organizing Committee
APHRS 2014

Jointly organized by:
APHRS Asia-Pacific Heart Rhythm Society | Indian Heart Rhythm Society

Endorsed by:
Heart Rhythm Society | European Society of Cardiology | Cardiological Society of India Delhi Branch

For queries write to us : secretariat@aphrsindia.com

APHRS Asia Pacific Heart Rhythm Society

Anatomy and Physiology of Pulmonary Vein

Yung-Kuo Lin, MD, PhD[1,2]; Yi-Jen Chen, MD, PhD[1,3]

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Atrial fibrillation (AF) is the most common cardiac arrhythmia, which is associated with cardiac dysfunction and strokes.[1] Thoracic veins, especially the pulmonary veins (PVs), play a key role in the genesis of AF since ectopic impulses generated from the PVs or non-PV regions could initiate AF.[2,3] Besides, PVs also contribute to the maintenances of AF. Therefore, elimination of PV arrhythmogenic foci or isolation of PVs may reduce AF occurrences of cure AF. PVs contain complex electrical activity due to distinctive anatomical and physiological characteristics. However, it is not fully elucidated how the AF risk factors can remodel PV electrical and structural properties to predispose AF genesis. It is intriguing to explore the pathophysiology of PV arrhythmogenesis.

Anatomy of Pulmonary Vein

Despite the diversity of individual ablation strategies, catheter ablation nearby the PVs has been proved to be a highly effective treatment option for the cure of AF. Precise mapping and ablation of PVs requires proper delineation of PV structure.

Histological Studies[4,5]

As a venous structure, the pulmonary venous wall composed of a thin endothelium, a media of smooth muscle and fibrous tissue, and a fibrous adventitia with extension of the atrial myocardium beyond the venoatrial junction (Figure 1). The smooth muscle of the venous wall overlapped with atrial myocardium, which longitudinally extended from the venoatrial junction in all veins with the longest sleeves found in the superior veins. The sleeves were thickest at the venoatrial junction, with a non-uniform circumferential taper in thickness toward the lung hila. The PV myocardal sleeves were significantly thicker in the left superior veins than in the right veins and were thicker in inferior portion than in superior portion of superior veins.

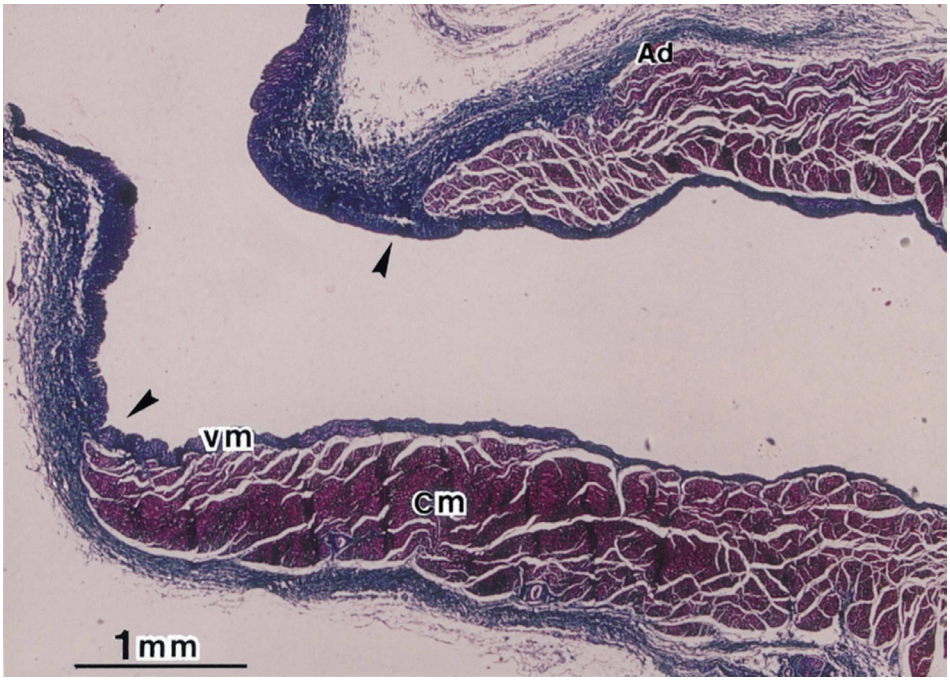

Figure 1. A longitudinal section of dog left superior pulmonary vein near the myocardial sleeve. Reproduced with permission, from Chen et al. Cardiovascular Research 2000;48:265-273. © Elsevier.

The venous wall consists of fibrous and adipose tissues with irregular shape, which interposed myocardial sleeve. Moreover, abundant nerves and ganglions of the autonomic system locate at the venoatrial junction or inside PVs. The complexity of PV myocardial structure can cause anisotropic conduction and enhance the genesis of reentrant circuits inside PVs.

Pulmonary Venous Imaging

Selective PV angiography had been used to study the details of PV anatomy.[6] The patients with paroxysmal AF initiated from the superior PVs had greater superior PV diameters than did either control subjects or the patients who had paroxysmal AF from other ectopic foci. Although the dilatation of the PV was not related to the site of the PV ectopic beats, the anatomic and geometric differences of PVs might participate in perpetuation of AF. However, the current PV angiography was not sensitive enough for detailed PV mapping or studying in PV ablation follow up. Modern 3D image modalities such as MDCT or MRI may improve the understanding of PV anatomy.

Modern 3-D Imaging Techniques

Pre-ablation Planning

MRI, MDCT, echocardiography, and trans-esophageal sonography have been used to study the PV anatomy before ablation.[7,8] Among them, MDCT and magnetic resonance angiography (MRA) are excellent tools to define the pulmonary venous anatomy and to observe patients for complications after the procedure, particularly PV stenosis.

In consistent with PV angiography, significant dilation of both superior PVs was demonstrated in PAF and CAF patients by MDCT and MRA. Moreover, MDCT had found the four PV trunks also became dilated after the patients were > 50 years old, but not in the patients aged 70 to 79 years and > 80 years.[9] Age correlated well with the four PVs with linear regression, indicates that age significantly determines PV structures (Figure 2). These findings show the important contributing effects of aging on PVs in aging-induced AF in the general population. Apart from aging effect, MDCT also been used to study the effects of myocardial ischemia on the structural changes in the PVs. Calcified coronary arteries are a marker of coronary atherosclerosis and are associated with a higher incidence of coronary events, which could be detected by MDCT presented as calcification score. A previous study has shown that in patients undergoing MDCT for general check-up, the PVs were enlarged in the presence of high calcium scores.[10] A highly calcified coronary artery may produce an abnormal PV structure and in turn facilitate AF genesis.

Post-ablation Follow-up of PV Stenosis

The incidence of PV stenosis after catheter ablation of AF ranged from 2% to 100%, which depends on the definition of PV stenosis, ablation technique, and diagnostic image modalities.[11] Real-time intracardiac echocardiography (ICE), transesophageal echocardiography (TEE), 3D CT and MRA all had been used to follow-up the post-

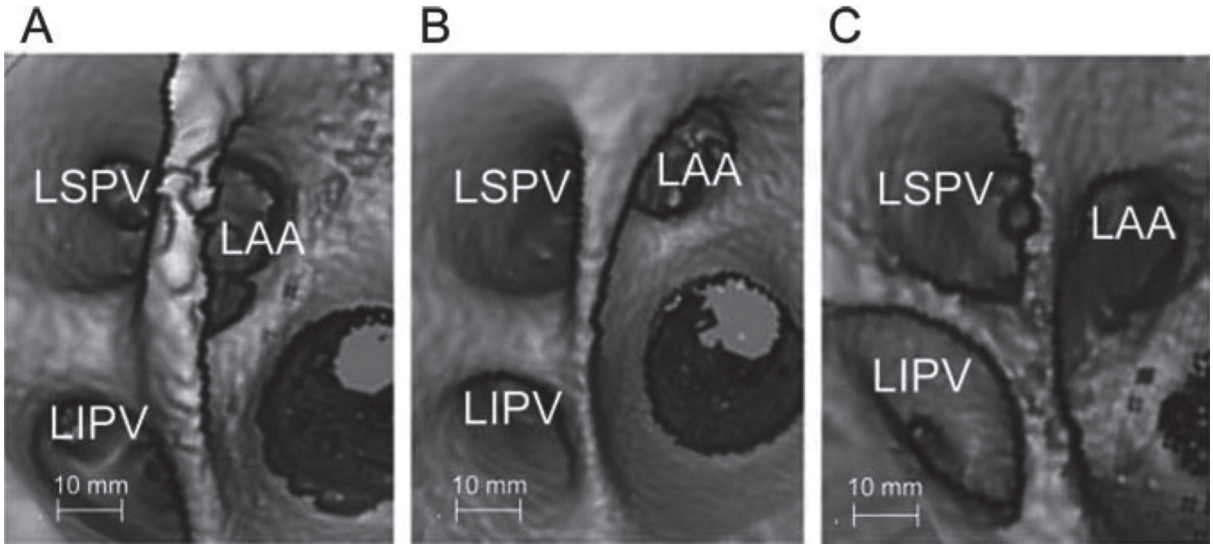

Figure 2. Intra-atrial oblique-sagittal views during MDCT from patients aged > 40 years (left, A), 50 to 59 years (center, B), and 70 to 79 years (right, C). Reproduced with permission, from Pan et al. CHEST 2008; 133:190-196. © the American College of Chest Physicians.

ablation PV stenosis. Late progression of PV stenosis during follow-up was demonstrated with up to 10–45% progressive reduction of PV diameter in the late (6–12 months) follow-up after PV isolation. Tsao et al. had used MRA to study PV and LA morphology before and 12 months after ablation of paroxysmal AF. They found that elimination of AF could decrease sizes of the superior PVs and LA, which indicates the reversibility of the structural changes in superior PVs and LA.[12] However, late recurrence of AF is associated with progressive LA dilation, although the causality is unclear. In addition to post-ablation PV stenosis, preexisting stenosis of PV had been reported occasionally. Through 3D CT or MRA to find the congenital focal narrowing or external compression by the adjacent structures in PVs may reduce the risk of ablation-related PV stenosis, which is mandatory for PV ablation strategy.

Physiology of Pulmonary Vein

PVs contain a mixture of pacemaker cells and working myocardium, which can be a subsidiary pacemaker or inducer for atrial arrhythmias. Enhanced automaticity or induced triggered activity in PV cardiomyocytes contributes significantly to PV arrythmogenesis and the genesis of AF.[13] Moreover, PVs can modulate electrical activity through mechanoelectrical feedback due to interactions between vascular structure and cardiomyocytes.[14]

Vascular Structure

PVs served more than a simple conduit for blood transportation. As a vascular structure, PVs contain endothelium and smooth muscle, which may produce nitric oxide (NO) through the enzyme of eNOS or iNOS. Our previous study had found that nitroprusside (NO donor) could decrease PV cardiomyocytes spontaneous beating, and suppress delayed afterdepolarization through inhibition of L-type calcium currents, transient outward currents and transient inward current.[15] Moreover, Chang et al. showed that stretch force dependently increased the incidence of spontaneous activity, and incidence of early afterdepolarisations or delayed afterdepolarisations in PVs.[14] Therefore mechanoelectrical feedback significantly regulates PV arrhythmogensis and exerts significant impact on the pathophysiology of AF.

Oxidative Stress

PVs receive the highest oxygen content blood in the circulatory system, which naturally make PVs bear a high oxidative stress. H_2O_2 increases PV spontaneous rate and induces PV burst firing with the genesis of early afterdepolarizations.[16] The oxidative stress related PV arrhythmogenesis can be suppressed or attenuated by pretreatment with ascorbic acid (an antioxidant) or N-MPG (a specific scavenger of the •OH free radical), which suggests the anti-AF potential of anti-oxidants. In addition, hypoxia was found to reduce PV beating rates, and reoxygenation can induce PV burst firings (Figure 3).[17] Therefore reperfusion may also induce PV arrhythmia and lead to AF genesis.

SAN-PV Interactions

In normal heart, the subsidiary pacemaker in PVs is overdrive by the faster sinoatrial node (SAN) activity. However, enhanced PV electrical activity or SAN dysfunction may facilitate the electrical effects of PV arrhythmogenesis to overdrive SAN and lead to PV or atrial tachyarrhythmia including AF. This theory has been proven from simultaneously electrical recordings in PVs and SAN through mechanical interruption of PV-SAN electrical connections.

Chen et al. nicely presented how SAN modulates PV electrical activities.[18] The isolated PVs showed more burst firing and EADs under the treatment of ATX-II as compared to those in the intact PV-SAN electrical connections. This study suggest there was mutual electrophysiological interaction between SAN and PV whenever SAN hyperactivity or dysfunction. Those findings at least in part may explain the pathophysiology of tachycardia-bradycardia syndrome, which was common feature of sick sinus syndrome.

Ionic Currents in PV Arrhythmogenesis

AF triggers in PV involved both calcium homeostasis related ion channels and non-calcium ion channels.[19] Abnormal calcium homeostasis and heterogeneous expression of potassium channels, pacemaker channels, and stretch- and swelling-activated chloride channels may modulate the electrical activity of PV and promote the occurrence of AF. Decreases in inward currents and/or increases in outward currents lead to shortening of action potential duration, which can facilitate the genesis and maintenance of AF. Different underlying diseases may yield different patterns of electrical remodeling, caused by alterations in electrical activity, ionic currents, and calcium handling, play an vital role in the arrhythmogenesis of PV cardiomyocytes.

Selective agents that could regulate the ionic currents underlying the electrical activity of AF triggers, would be the potential target therapy for atrial tachyarrhythmias. Although the molecular mechanism underlying the abnormal calcium homeostasis is not clear, anisotropic structures with abundant fibrous or adipose tissues may lead to calcium overload in PV cardiomyocytes. Moreover, increased sodium-calcium exchanger currents or abnormal calmoduline-kinase II in PVs can produce calcium leaks,[20] which is suggested to be critical to the genesis of AF.

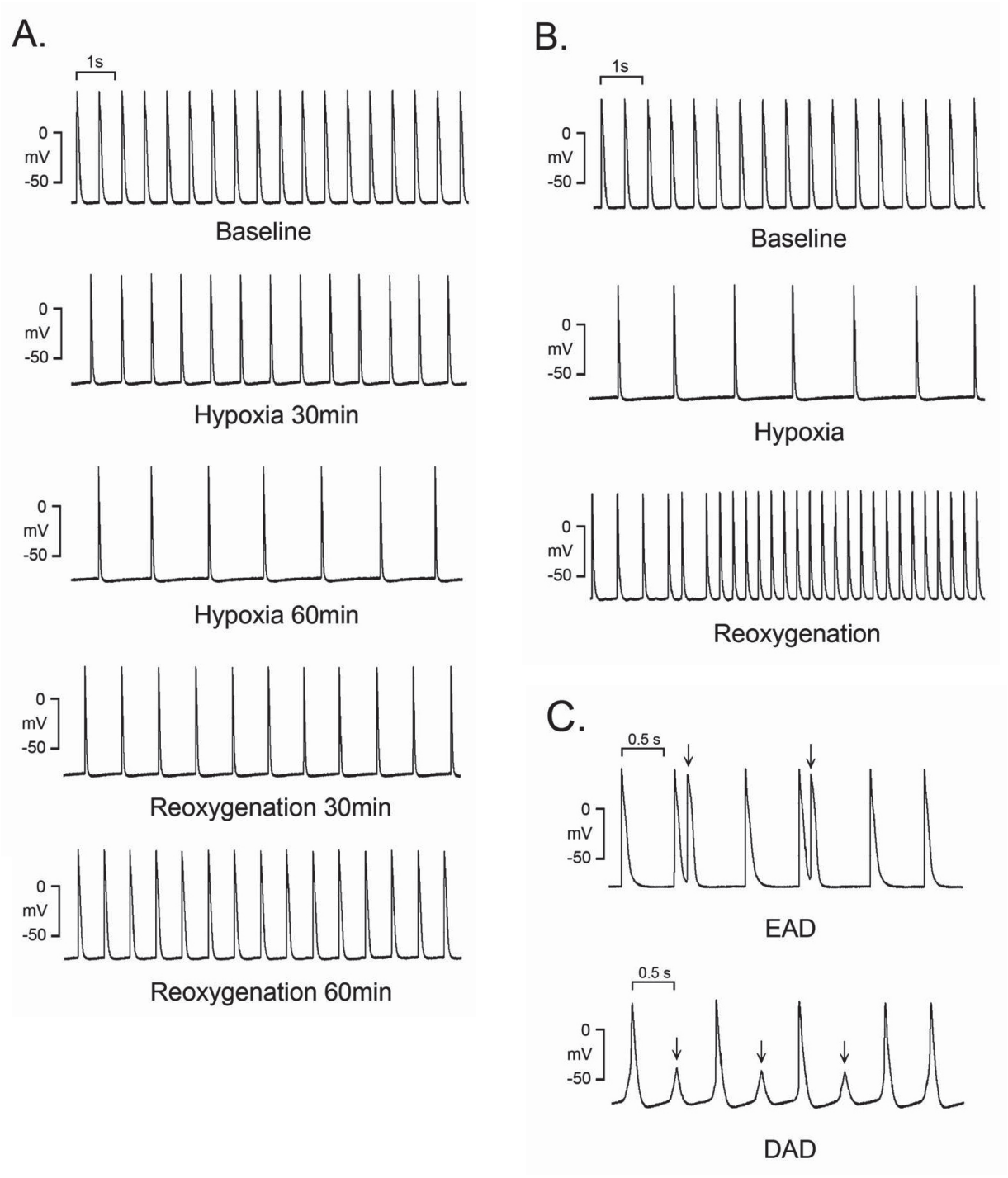

Figure 3. Effects of hypoxia/reoxygenation on PVs with spontaneous activity. Reproduced with permission, from Lin et al. 2012, Clinical Science (London, England : 1979) 2012;122(3):121-32. © the Biochemical Society.

Conclusions

PVs act a vital role in AF mechanism. PV isolation is still the core therapy of most non-pharmacological treatments. Understanding the complexity of PVs anatomy and physiology is crucial for the successful AF treatment.

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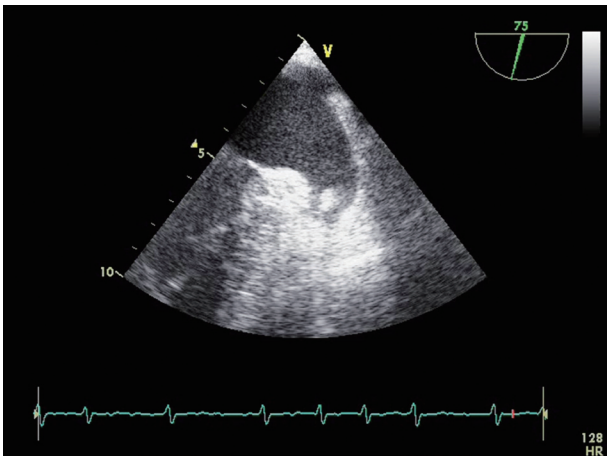
EP Image: A Clot or Not

Dr Kathy Lee, MBBS, FRCP, FACC

Cardiac Health Heart Centre, Hong Kong SAR

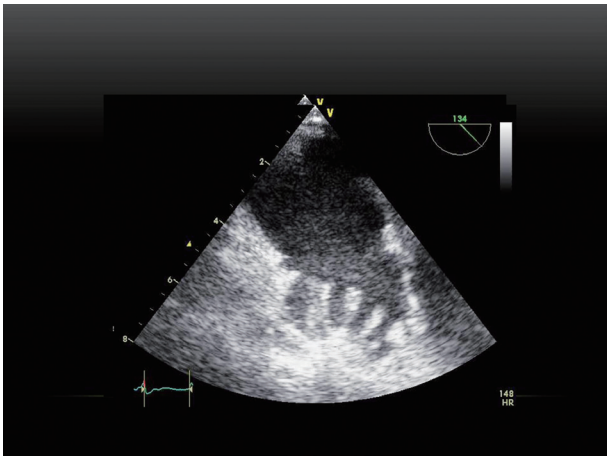
A 60 years old gentleman with drug refractory atrial fibrillation was planned for pulmonary vein isolation. He had no history of prior stroke or thromboembolism. The CHA2DS2VASc score was 1. He underwent routine trans-esophageal echocardiogram before the ablation procedure and was suspected to have a clot in the left atrial appendage (LAA).



Trans-esophageal echocardiography enables better visualization of the LAA, an important source of embolization in atrial fibrillation. However, it may sometimes over- or under-diagnose thrombus. It is well described that prominent pectinate muscle may mimic clot formation.

Differentiation between pectinate muscle and thrombus in the LAA:

	Pectinate muscle	LAA thrombus
Shape	Strand-like	Roundish
Attachment	Adherent	Pedunculated or adherent
Mobility	Not mobile	Can be mobile
Associated feature	May or may not be associated with spontaneous echo	Often associated with spontaneous echo



A better profiled LAA showing exaggerated petinate muscle in the multi-lobed left atrial appendage mimicking thrombi formation.

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ECG Quiz

The model commentary will be provided in the next issue No. 16

Meiso Hayashi, MD

Department of Cardiovascular Medicine, Nippon Medical School

A 59-year-old man presented with frequent palpitations and dyspnea, which emerged under no particular conditions and persisted several hours daily. The examinations revealed no abnormalities except that the body-surface electrocardiogram exhibited irregular incessant supraventricular tachycardias (Figure 1).

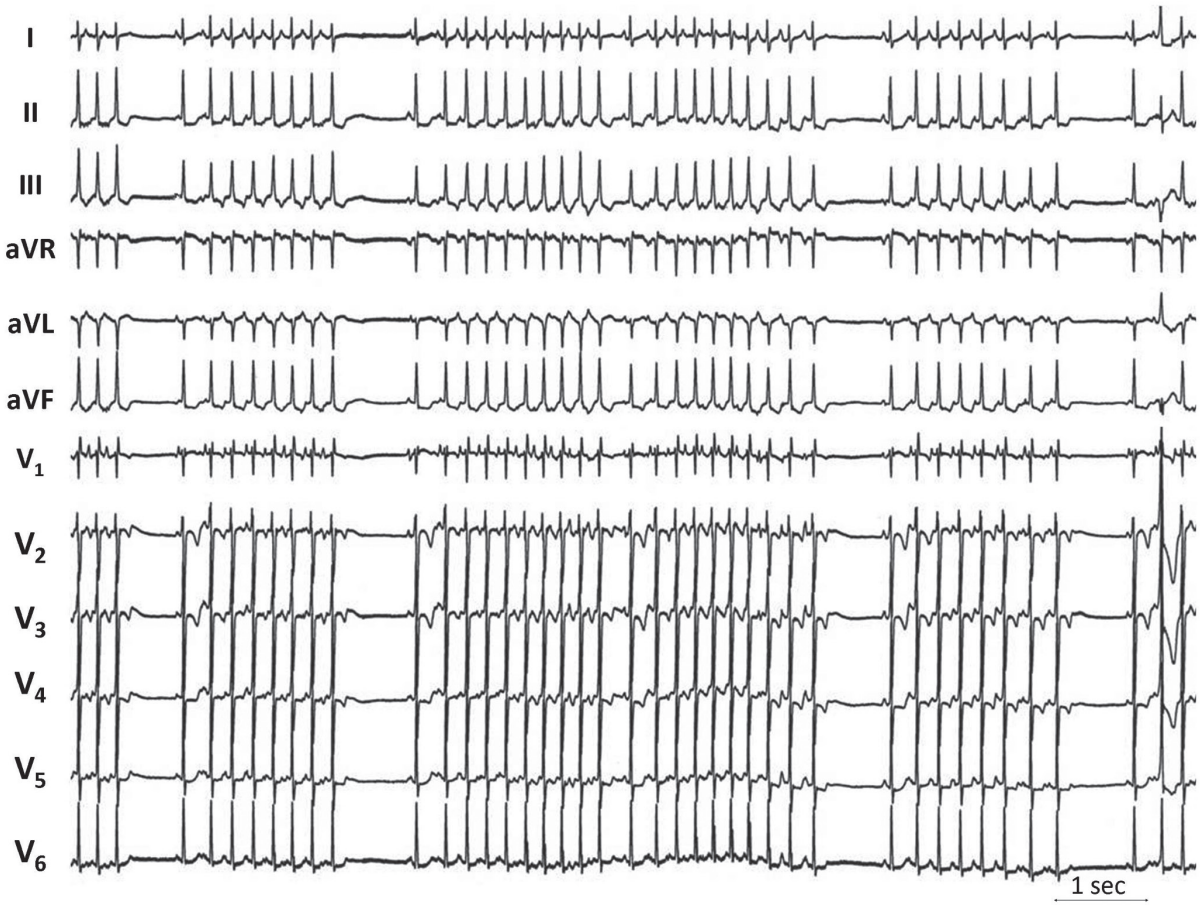

Figure 1

Question:

Q1. What seems to trigger these arrhythmias?

1) Exercise
2) Swallowing
3) Respiration
4) Excretion

Q2. What is an optimal therapeutic strategy?

1) Beta-blockers
2) Class I antiarrhythmic drugs
3) Class III antiarrhythmic drugs
4) Catheter ablation

Introduction to Renmin Hospital of Wuhan University

Congxin Huang, MD. PhD.

Renmin Hospital of Wuhan University, Hubei General Hospital

Hospital Overview

Renmin Hospital of Wuhan University

Renmin Hospital of Wuhan University celebrated its 90th anniversary in 2013. It is the oldest and one of the primier hospitals in Hubei province. It provides not only traditional medical care, but also medical education and leading scientific research.

Renmin Hospital of Wuhan University is located at the South Bank of the Yangtze River at the corner of Wuchang Jiefang Road and Wuhan Zhangzhidong Road (formerly Ziyang Road). In 2013, Renmin Hospital of Wuhan University opend east branch, which is located at the East Lake New Technology Development Zone. Since 1923, Renmin Hospital of Wuhan University has trained more than 4700 healthcare professionals and 800 postgraduates, who are now pursuing excellence in medical care, education, and research. The hospital is praised by the community for its adherence to its foundational principles, including: "All for patients, for all patients, for patients of all", "science, amicability, conscience", the innovative consciousness of unique and advanced medical technology, and the pursuit of creating a harmonious and warm recuperative environment. Furthermore, outstanding achievements have been made in research over the past ten years. Until today, the hospital has undertaken over 1000 international, national, and provincial scientific research projects. In 2012, it was ranked eleventh amongst national medical institutions based upon publications of its scientific research, and first in the World Journal Ranking Authority of medical institutions.

Renmin Hospital of Wuhan University

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Tel:	86-27 88041911/88041919
Fax:	86-27 88042292
No. of Beds:	3,000 (the main distict) & 2300 (the east branch)
No. of Staff:	4,700 employees (Including 580 highly skilled physicians)

Division of Cardiology

Academic Leader: Congxin Huang, MD
Director: Hong Jiang, MD
Number of Cardiologist: 67
(12 are specialized in cardiac electrophysiology)
Clinical Cardiac Electrophysiologist:

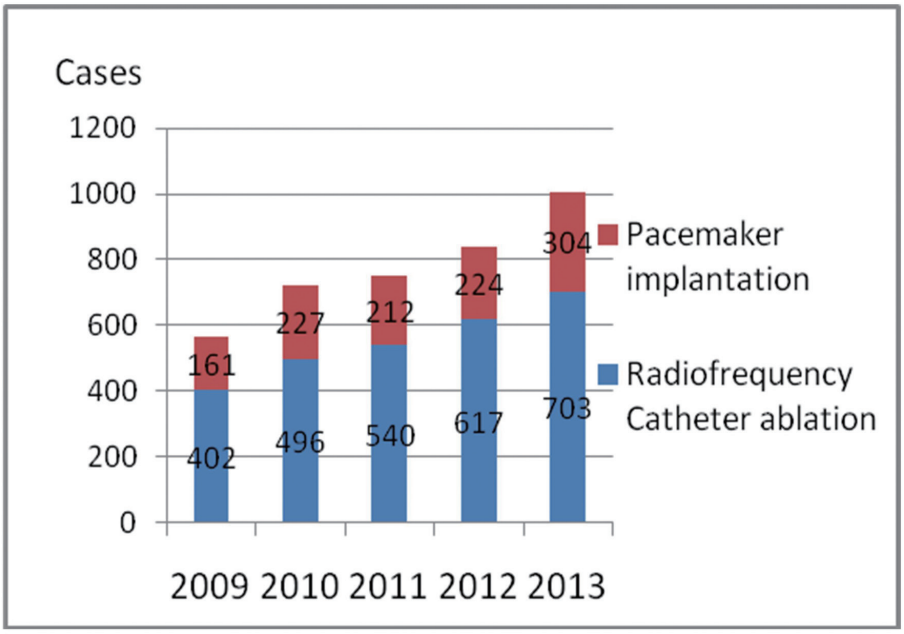
Congxin Huang	Hong Jiang, MD
Jun Wan, MD	Bo Yang, MD
He Huang, MD	Mingwei Bao, MD
Gang Wu, MD	Yuanxiu Chen, M.S.
Qingyan Zhao, MD	Zhi-bing Lu, MD
Jinjun Liang, MD	Yu Liu, MD

And, 6 electrophysiology fellow physicians

The Leading Arrhythmia Prevention and Treatment Center in China

Arrhythmia Prevention and Treatment Center of Renmin Hospital of Wuhan University, the national cardiovascular disease intervention training, has been awarded more than 20 national and provincial science and technology progress awards.

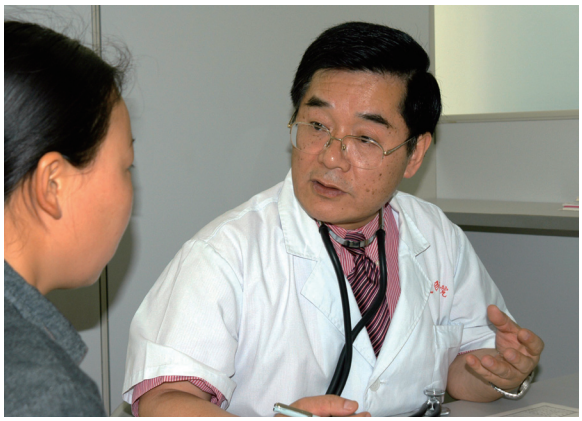
Arrhythmia Prevention and Treatment Center consists of a team of talented, highly trained, and well experienced professionals. Dr. Congxin Huang and Dr. Hong Jiang are pioneers in catheter ablation for arrhythmias in China. Dr. Congxin Huang is currently the president of Chinese Society of Pacing and Electrophysiology (CSPE). The center provides comprehensive treatment options with a high level of success for patients with arrhythmias. Furthmore,



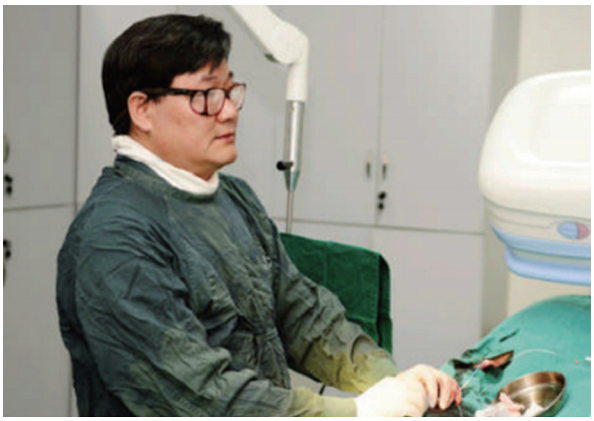
it provides high-quality treament in complicated and difficult arrhythmias. Unipolar mapping technology, transradial catheter ablation, and the understanding of the cardiac-cardiac reflex pathway have all come from the Arrhythmia Prevention and Treatment Center and have been internationlly recognized to improve outcomes in pathway ablation and AF ablation. Newly proposed advanced technologies, such as renal denervation and left atrial appendage occlusion, have been recently introduced into the clinic as well. In 2013, radiofrequency catheter ablation and pacemaker implantation have been performed in 703 and 304 patients, respectively.

In addition to clinical medical care, all physicians and medical professionals at Renmin Hospital of Wuhan University have aggressively dedicated themselves to undertaking research and investigation in this first-class advanced institute in China. The hospital has mulitple research interests including electrocardio-biology, basic electrophysiology, clinical electrophysiology, genomics, and proteomics. Over the last 4 years, the institute has made a contribution of 554 original papers,including 170 SCI papers. These articels are published in the highest ranking medical journals, including Circulation, PNAS, Hypertension, and Cardiovascular Research.

As in the past, the hospital will continue to adhere to the crede; "take the patient as the center, take the quality as the core." It will work to strengthen personnel training and progress, scientific and technological innovation steadily forward.



Dr. Congxin Huang



Dr. Hong Jiang

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Electrophysiology in Malaysia

Dr. Razali Omar

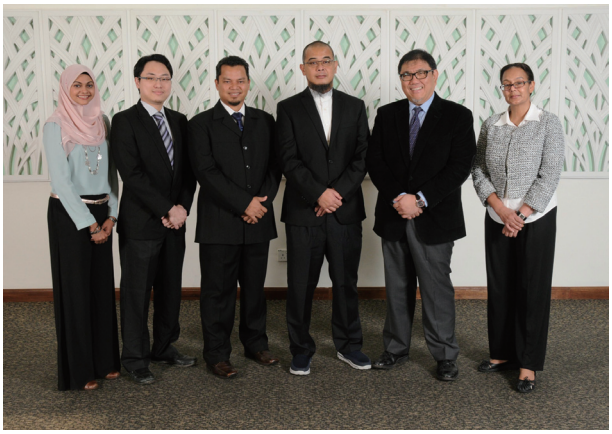
Senior Consultant Cardiologist, Director, Electrophysiology Unit, Department of Cardiology, National Heart Institute, Malaysia

Malaysia is a multi-ethnic and multi-cultural country with a population of 30 million people. It has land borders with Thailand, Singapore, Indonesia and Brunei. The official language is Bahasa Malaysia but English is widely written and spoken.

Healthcare is provided by both public and private hospitals. Every state in the country have at least one hospital which provides cardiac services including catheterization lab and cardiac surgery.

National Heart Institute (NHI), Kuala Lumpur

National Heart Institute (Institut Jantung Negara) was established in 1992 to be the national referral center for cardio-thoracic related ailments. It started as a 274 bedded hospital with 4 consultant cardiologists, one of whom was an electrophysiologist. NHI was the only hospital providing EP related services in the country for the next 6 years until a private hospital offered ablation procedures, but it remained the sole provider for EP services in the public sector until 2012 when a government hospital started offering EP services. Today NHI has expanded to 461 beds, 106 of which are critical care beds. There are 7 cath labs including a hybrid lab. The 2 EP labs are equipped with either a Carto or Navx 3D mapping systems.

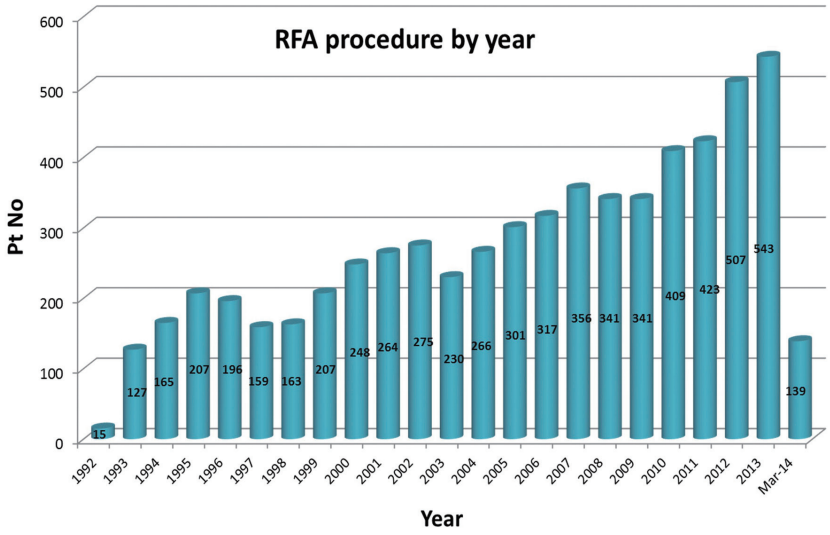


NHI EP unit. L-R; Dr Barveen Aisha, Dr Koh Kok Wei, Dr Zulkeflee Muhammad, Dr Azlan Husin, Dr Razali Omar, Dr Surinder Kaur.

The EP community in Malaysia is very small comprising of only 9 EPs across the country of which 4 are at the NHI – Dr. Razali Omar, Dr. Azlan Hussin, Dr. Surinder Kaur and Dr. Zulkeflee Muhammad.

Electrophysiology Procedures

The main bulk of catheter ablation and device implants for the country are done at the NHI. In 2013, we performed around 500 ablations and 500 implants. Ablations are performed for the whole spectrum of simple and complex cases. Most simple cases include AVNRT, AVRTs, idiopathic VTs, atrial flutters. The complex cases are done using 3D maps using either CARTO or NAVX. These includes AF ablation, atypical flutters and scar-related VTs.



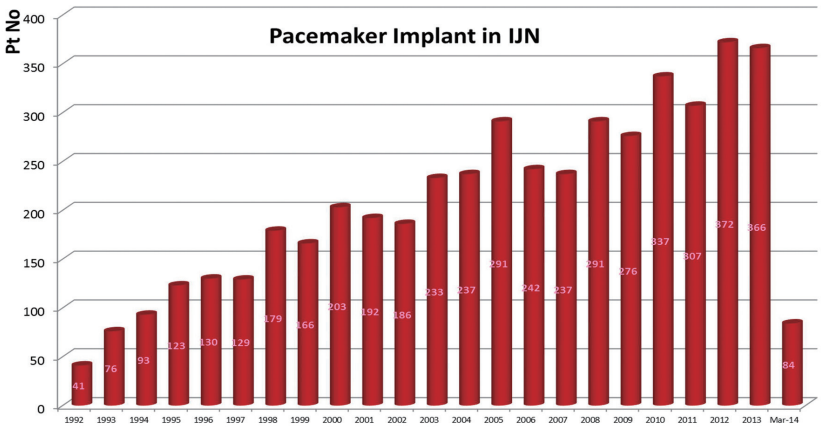
Device implants include pacemakers, implantable defibrillators and cardiac resynchronization devices. Since 2010, we have started our left atrial appendage occluder program and we are now one of the top center for the Watchman device in Asia.

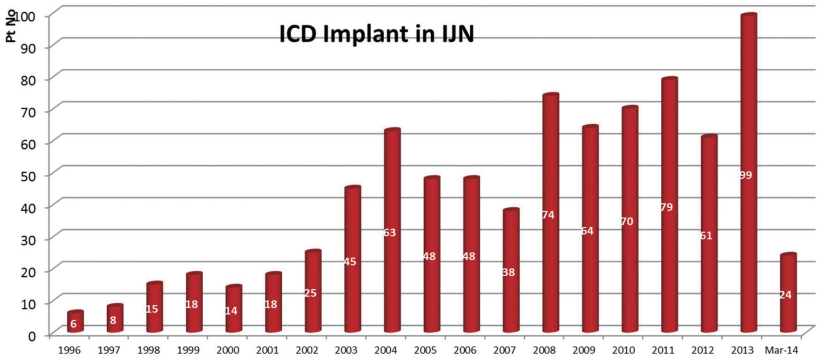
Training Center for Electrophysiology

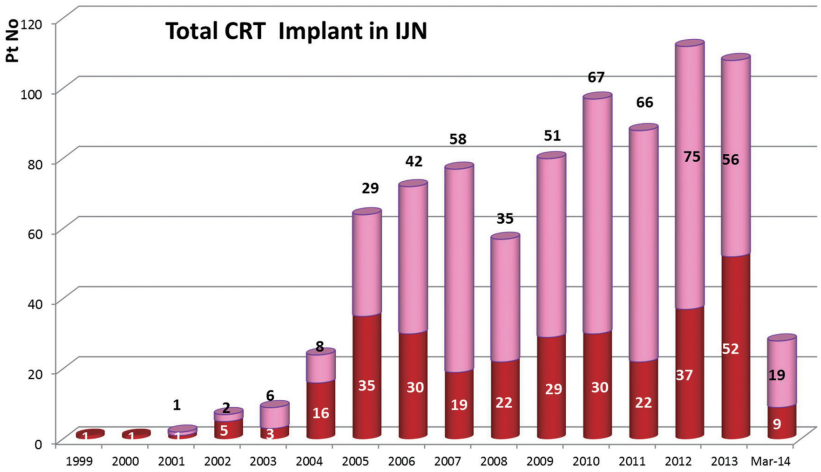
The NHI has been involved in training both local and foreign electrophysiologists since year 2000. To date we have trained 14 electrophysiologists who have returned to their own country to set up their own units.

Since 2006, we have been conducting CRT "hands on" workshops. What started from a request by Japanese colleagues to learn the tips and tricks of CRT implantation, have grown into a popular workshop with nearly 200 implanters from all over Asia including Australia who have benefited from these workshops.

In 2012 we started workshops for Watchman implants and have also proctored new labs embarking on this therapy.







Research

The NHI and in particular the EP unit have been involved in many multicenter trials. Of note were the PACE, SAFE, ASSERT, RELY, MULTISENSE and SIMPLE trials. Most of these have been published in peer reviewed journals. Recently, we had the privileged of being the first center in Asia to implant Micra a leadless pacemaker (Medtronic) as part of a multicenter clinical trial.

Accreditations and Awards

The NHI has in calculated a quality improvement culture that started with its Joint Accreditation award (JCI).

The NHI was awarded the Prime Ministers Innovation Award for 2011 and is a Joint Commission International accredited hospital.

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ECG Commentary Related to the Quiz in the No. 14 Issue

Paul C Y Lim, Eric T S Lim, Kah Leng Ho, Boon Yew Tan, Chi Keong Ching, Wee Siong Teo

National Heart Centre Singapore

Answer:

4. Type 1 Brugada pattern ECG – Fever sensitive Brugada

Commentary

The mechanism of fever triggered arrhythmias in Brugada syndrome is not well understood. Certain SCN5A mutations are known to temperature sensitive – L325R missense mutation[1], F1344S missense mutation[2], R535X nonsense mutation1, T1620M missense mutation[3], and V1340I mutation[4].

One underlying mechanism for the electrographic changes seen in Brugada syndrome has been hypothesised to be due to a repolarisation disorder arising from loss of function of the Na channels leading to action potential alterations to different degrees in epicardial and endocardial cells. I_{to} repolarization channels densities are higher in the right ventricle than left ventricle, and in the epicardium compared to the endocardium. This, together with the loss of I_{Na} due to sodium channel mutations creates a transmural voltage gradient during the early phase of repolarisation. Voltage differences at various epicardial sites results in dispersion of repolarization within the epicardium[5,6].

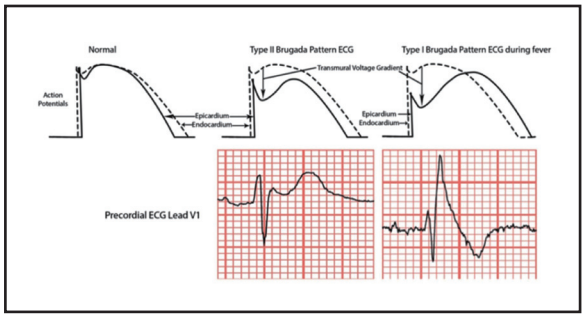

Figure 1: *Increased transmural voltage gradient due to decreased I_{Na} current in the early repolarisation phase of the action potential. Corresponding surface ECG changes are seen.*

In Brugada syndrome patients, sodium channel function can be temperature sensitive. In vivo studies on cells with the T1620M mutation showed progressive decay of the T1620M current at increasing temperatures and more rapid inactivation kinetics of I_{to} at 36°C than at room temperature[3]. Accelerated inactivation of sodium channels in elevated temperature is proposed to be due to failure of expression of sodium channels or from early conformational changes of the sodium channels leading to early closure[7]. These dynamic voltage gradients manifest as the characteristic electrographic changes seen during febrile episodes with resolution during apyrexia (Figure 1).

Temperature sensitivity is illustrated by the series of electrocardiogram changes of our patient where the coved shaped ST elevations appear prominently at the highest recorded temperature (Figure in the former issue), with some improvement during low grade pyrexia (Figure 2), and only subtle changes on follow up electrocardiograms when our patient was well (Figure 3). Early identification of febrile states and administration of antipyretics is important to such patients.

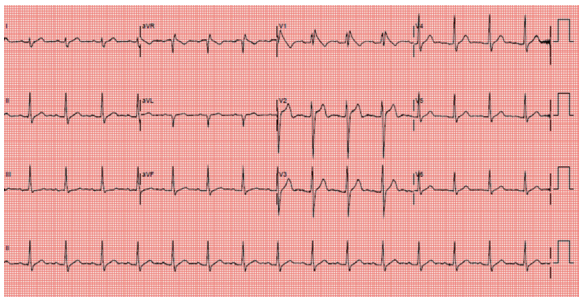

Figure 2: *ECG performed on 29/10/2010 when his fever was resolving.*

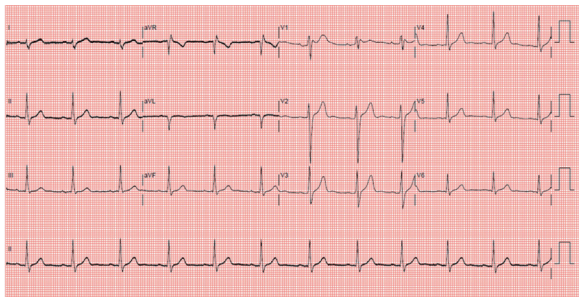

Figure 3: *ECG performed post discharge on regular clinic follow up when he was well.*

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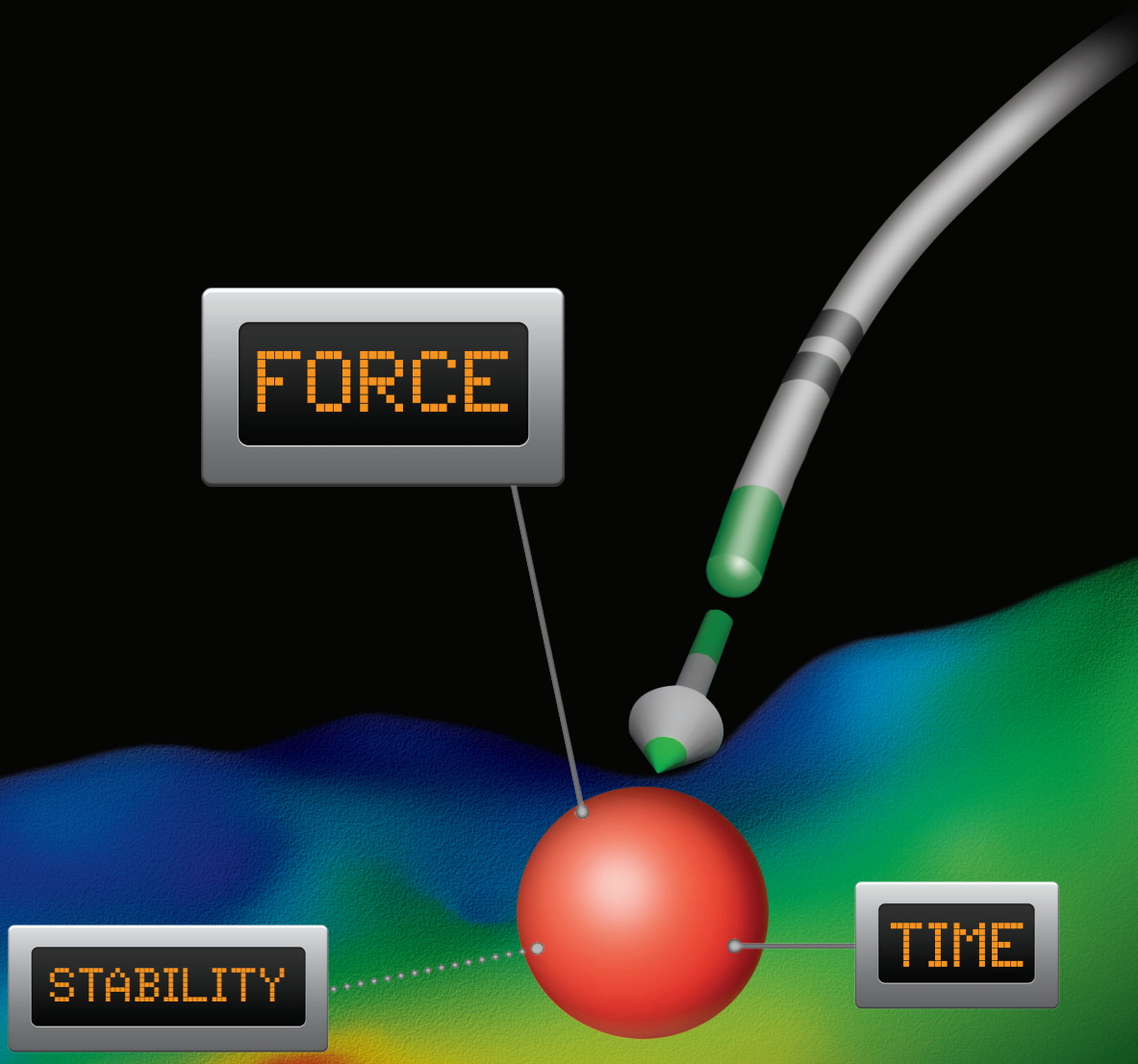

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