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Contents

Neurocardiac Mechanisms in Sympathetically Triggered Inherited Cardiac Arrhythmias

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Introduction

Inherited cardiac arrhythmias are electrical abnormalities of the heart caused by gene mutations encoding various cardiac ion channels or their interacting proteins.[1] The main inherited cardiac arrhythmias are long QT syndrome (LQTS), short QT syndrome, catecholaminergic polymorphic ventricular tachycardia (CPVT), and Brugada syndrome. Unfortunately, sudden cardiac death, an unexpected natural death, might be the first clinical presentation in patients with inherited cardiac arrhythmias, especially in young adolescence. The diagnosis is based on clinical presentation and family history, the characteristics of the electrocardiogram during rest and exercise, and genetic analyses. Table 1 lists prevalence, electrocardiographic characteristics, underlying gene mutation, age of onset, and known triggers for each type of inherited cardiac arrhythmias. Among the inherited cardiac arrhythmias, the LQTS (particularly LQTS type 1, LQT1) and CPVT are known for their association with sympathetic triggering of life-threatening cardiac events. In this brief review, we discuss the neurocardiac mechanisms in inherited cardiac arrhythmias, focusing on LQTS and CPVT.

The Brain-Heart Connection in Inherited Cardiac Arrhythmias

Starting from embryonic stages, the autonomic nervous and cardiovascular systems are functionally interplayed with both systems affecting and regulating each other. The cardiac-projecting sympathetic neurons are in the stellate ganglia, middle cervical and second thoracic ganglia. Postnatally, the maturing heart becomes densely populated with a network of nerve terminals. Functional connections between sympathetic inputs and target cells are characterized by neurotransmitter switching. Norepinephrine (NE) released from sympathetic nerves activates adrenergic receptors in e.g. cardiomyocytes, leading to downstream positive chronotropic, inotropic, dromotropic and lusitropic effects. Disease states associated with sympathetic hyperactivity augmented released noradrenaline and/or adrenaline, involving each step in the process of arrhythmogenesis.[2]

In 1985, the concept of “neurocardiology” had been introduced by a case study of a neurologist's wife who experienced ventricular tachycardia and epilepsy.[3] The patient’s tremor and palpitation could be suppressed by propranolol and nadolol until a large intracranial glioma was found and surgically removed. With the understanding of the autonomic system and cardiac arrhythmia, neuromodulation, mostly designed to suppress the sympathetic tone and/or increase parasympathetic tone, has emerged as a therapeutic strategy for the treatment of rhythm disorder. In inherited cardiac arrhythmias, the interplay between the heart and brain has been proven by examples of syncope or death, resulting from hyperactivity of the sympathetic nervous system during extreme emotions and/or stress. LQTS and CPVT are characterized by catecholamine-induced ventricular arrhythmias and typical adrenergic triggers include intense emotional stress, physical activity, and water immersion.

Neuromodulatory Therapies in Inherited Cardiac Arrhythmias

Beta-adrenergic blockade has remained the first option in patients with inherited cardiac arrhythmias, especially LQTS and CPVT. International guidelines suggested an implantable cardioverter-defibrillator (ICD) for patients who continue to have ventricular arrhythmias despite β-blocker therapy or for survivors of aborted sudden death. However, ICD implantation in children and young adults could be challenging and concerning because of long-term complications and psychosocial drawbacks.[4] In addition, proarrhythmic effects of ICD discharge may cause more malignant arrhythmias or even electrical storm in patients with CPVT.[5] Left cardiac sympathetic denervation (LCSD), a surgical antiadrenergic intervention, has emerged as an important treatment option for select patients with hereditary cardiac arrhythmias. A meta-analysis study showed LCSD could effectively reduce occurrence of lethal arrhythmias in both CPVT and LQTS patients. In the latter, LCSD was shown to shorten heart rate-corrected QT (QTc) interval by an average of 28 ms.[6]

In vivo studies showed that the anti-arrhythmic mechanisms of LCSD included anti-adrenergic and vagotonic effects, antagonism of neurotransmitters and correction of imbalanced neuronal nitric oxide synthase.[7] Moreover, LCSD did not affect resting heart rate or chronotropic competence during exertion. On the contrary, heart rate might even increase during exercise after LCSD, which is caused by a reflex rise in right stellate ganglion activity. Another concern of LCSD is post-denervation supersensitivity, a pro-arrhythmic condition characterized by increased sensitivity of the myocardium in response to circulating catecholamines. Animal studies confirmed that LCSD did not completely deplete catecholamine stores in the myocardium[8], nor did it increase the propensity to ventricular arrhythmia upon NE administration.[9]

Neurocardiac Mechanisms in a Mouse Model of Inherited Cardiac Arrhythmias

Calmodulinopathy is a relatively new entity of inherited cardiac arrhythmias associated with mutations in the genes encoding for the calcium (Ca2+)-binding protein, calmodulin (CaM). Mutations in the all three CaM-encoding genes CALM1, CALM2 and CALM3 have been linked to ventricular arrhythmias manifesting with features of CPVT and/or LQTS.[10-16] The largest calmodulinopathy registry reported that 13 out of 74 subjects had mild to moderate neurological impairments, including seizures, developmental delay, and motor and/or cognitive disability, suggesting a primary neuronal component of congenital calmodulinopathy.[11] A recent study has demonstrated that human calmodulin mutations cause neuronal dysfunction in C. elegans, including impaired chemosensing and neuromuscular signaling.[17] Neuronal, voltage-gated calcium channels Cav1.3 and Cav2.1 exhibit attenuated calcium-dependent inactivation and/or facilitation when co-expressed with certain human CaM mutations in HEK293 cells.[18] Our previously generated knock-in mouse model of a human calmodulinopathy caused by the p.N98S mutation in CALM1 exhibits a mixed CPVT/LQTS phenotype which exclusively manifests during sympathetic activation, prompting us to investigate the possibility that primary sympathetic neuron dysfunction contributes to arrhythmia induction in Calm1$^{N98S/+}$ mice.

CaM is highly expressed in neurons where it controls neurotransmitter release and electrical activity. It seems therefore plausible to hypothesize that heterozygosity for the Calm1^{N98S} allele causes sympathetic dysfunction, resulting in stress-induced cardiac NE overflow. Exteroceptive stimuli elicit signals to dorsomedial hypothalamus which in turn excite preganglionic sympathetic neurons, located in the spinal cord, and ultimately sympathetic neurons in the paravertebral ganglia, resulting in NE release in the target organ. Conceptually, electrical hyperexcitability and/or increases in the excitation - NE secretion coupling gain could each give rise to sympathetic hyperactivity which may be responsible for the -adrenergically induced potentiation of L-type calcium current that we have observed in Calm1$^{N98S/+}$ ventricular cardiomyocytes.[19] The diagram presented in Figure 1 summarizes our hypothesis on the role of primary sympathetic hyperactivity for LQTS/CPVT manifestation in the Calm1$^{N98S/+}$ mouse model.

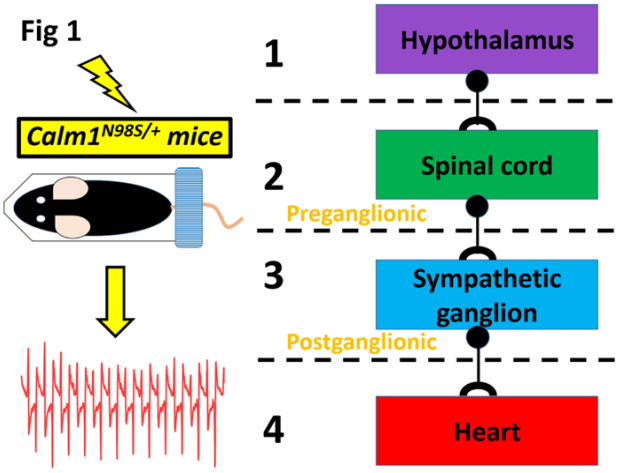

Figure 1. Schematic diagram of the neurocardiac hypothesis in Calm1N98S/+ mice.

Conclusion

Sympathetically triggered congenital arrhythmias, particularly LQTS and CPVT significantly increase the incidence of repeated syncope, lethal arrhythmic events and sudden cardiac death, especially in young individuals. Current neuromodulatory therapies, beta-blocker and cardiac sympathetic denervation significantly alleviate the symptoms. Enhanced understanding of the neurocardiac mechanisms underlying the sympathetically triggered inherited cardiac arrhythmias could potentially improve management in patients and families with inherited cardiac arrhythmias.

Arrhythmia	Prevalence	ECG characteristic	Gene/ion channel /function	Age of onset	Triggers of cardiac events
Long QT syndrome[20-23]	1/2,000	12-lead ECG with Bazett's formula QTc ≧500ms or QTc between 480-499ms with unexplained syncope	KCNQ1 (30-35%), ↓ I_{Ks} KCNH2 (25-40%), ↓ I_{Kr} SCN5A (5-10%), ↑ I_{Na}	<20y	LQT1: exercise (swimming) LQT2: acute arousal LQT3: rest
Catecholaminergic polymorphic ventricular tachycardia[21, 24-26]	1/10,000	Exercise or catecholamine-induced bidirectional VT or polymorphic ventricular premature beats or VT	RYR2 (60-65%), ↓ CASQ2 (3-5%), ↓	<15	Adrenergic stimulus (exercise/emotional stress)
Brugada syndrome[21,27]	0.5-1/1000 (South Asia)	ST elevation with type I morphology at lead V1 and V2, located at 2nd, 3rd, or 4th intercostal space spontaneously or after the challenge of class Ic drugs	SCN5A (20%), ↑ I_{Na}	30-50ys	Fever. sleep
Short QT syndrome[21,28]	0.2-1/1,000	12-lead ECG with Bazett's formula QTc≤330 ms	KCNH2 (<5%), ↑ I_{Kr} KCNQI (<5%), ↑ I_{Ks} KCNJ2 (<5%), ↑ I_{K1}	Same	Unknown, mainly at rest

Table 1 Summary of inherited cardiac arrhythmias

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GETTING TO KNOW APHRS LEADER

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APHRS Country/Region Representatives 2023 of Myanmar

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Why did you choose to enter medicine and above all, prefer to specialize in Electrophysiology?

After getting higher scores in matriculation, I chose to enter medicine according to my interest and dedication. It has been a long journey to finish a master degree in Internal Medicine, MRCP, Master in Cardiology in the UK and Doctorate in Cardiology locally. The Cardiac Electrophysiology (EP) service was far-fetched in Myanmar at the time. I joined Cardiology and there was a demand to set up an EP Lab in Yangon General Hospital. I was trained in Electrophysiology and device implantation at National Heart Center Singapore under supervision of Professor Teo Wee Siong. I became the first EP fellow to learn for the first EP set up and service in Myanmar.

What do you regard as the most significant development in Electrophysiology in the recent past?

After setting up the EP lab, Yangon General Hospital has become the first center for arrhythmia service in Myanmar for EP procedures and device implantation. We gradually improve ourselves, from doing conventional to advanced procedures. We are also collaborating with overseas EP doctors to perform complex cases like VT ablations and advanced pacing procedures like His Bundle Pacing.

Can you talk about an accomplishment that you are particularly proud of?

During these years, I have expanded to perform EP procedures in pediatric patients. I have arranged the local and overseas training for these juniors and allied professionals, so as to expand the EP service across the country. I am also providing the service for mitral valvotomy procedures and interventional procedures for adult congenital heart diseases like Atrial Septal Defect (ASD) and Patent Ductus Arteriosus (PDA).

If you could have an alternative career, what would it be and why?

I am currently having an alternative career apart from being an electrophysiologist. I am actively involved in the public health sector and leading a project in prevention of Cardiovascular Disease (CVD) under World Health Organization (WHO). I am also mentoring in Cardiopulmonary Resuscitation (CPR) training across the country, aiming to prevent sudden cardiac death in the community.
Not on that, I also lead my team in charity medical tours to remote areas in many states and divisions of Myanmar.

Who has inspired you the most in your life and why?

I have been inspired by my parents the most, who had brought me up to become a graduate and achieve a professional career. They encouraged me to serve the public in return.

At work (Yangon General Hospital)

What are your hobbies and interests outside of medicine?

I love to spend my extra time cooking, shopping and listening to music for relaxation.

In Myanmar (Bagan)

In UK (London)

What is the funniest thing that has happened to you recently?

I am afraid I cannot recount funny things in the Covid-19 era for the past few years.

What is your best life advice, motto or favorite quote?

“Forgiveness is not an occasional act, it is a permanent attitude”

What advice would you give to your younger self?

In life, difficulties, struggle and success are always accompanied. Perseverance and sustained ability are needed for better achievements. Without stress, performance might not be good.

What are your thoughts about some of the emerging technologies, and the way they will shape the future care of arrhythmia patients?

Although there are advances in technology and complex procedures for arrhythmia, as an EP physician in a developing country with limited facilities, I’ve always thought about how to simplify EP procedures and service for the benefit of patients.

“Figure-of-Eight” Subcutaneous Suture to Achieve Groin Hemostasis after Atrial Fibrillation Ablation: A Mini-Review

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Introduction

Electrophysiology study and catheter ablation for many arrhythmias have grown over the decades. These invasive approaches require the insertion of many sheaths in the femoral veins. The complication rate for these procedures varied among studies, but these occurred around 0.2 to 1.5 percent, especially in atrial fibrillation (AF) ablation.[1] The most common complication was a vascular complication, which comprised hematoma, pseudoaneurysm, arteriovenous fistula, and retroperitoneal bleeding. These complications resulted in prolonged hospitalization; the patient might require blood transfusion and/or surgical repair.[2] Many interventions were implemented to prevent these complications. One process that must be focused on is achieving adequate hemostasis after sheath removal. There were various practices to achieve hemostasis, such as manual compression, suturing, and closure devices. A temporary Figure-of-Eight (FoE) subcutaneous suture for femoral venous hemostasis has been introduced and progressively utilized. Therefore, this mini-review focuses on its origin, technique, efficacy, and safety.

The Beginning of Figure-of-Eight Suture

Manual compression to achieve hemostasis is a conventional intervention. It could be time-consuming for staff to compress until achieving hemostasis, followed by the patient's bed rest for 4-12 hours. In the past, there were some limitations for the closure devices, such as their expensive cost as well as devices being limited to sheath sizes ≤8 Fr. Mehmet Cilingiroglu, et al first published an article for the subcutaneous temporary Figure-of-Eight suture closure technique for the removal of large-caliber venous sheaths.[3] They used a 0-0 silk and a large curved cutting needle to suture deeply through the subcutaneous tissue on the caudal side of the sheath and then crossed the needle to suture back over the cranial side of the sheath, as illustrated in Figure 1. After the sheath was pulled out, the silk was knotted and the suture was tightened. Therefore, some subcutaneous tissue was bunched up to support hemostasis. A wire can be left in the vessel after sheath removal and then pulled out if the closure is successful. However, if this fails to stop bleeding, leaving a wire in place allows for sheath reintroduction and resuture can be performed. With their practice, the suture can be removed on the same day or the following morning. Moreover, the suture can be removed in 30-60 minutes in unanticoagulated patients and 4-8 hours in fully anticoagulated patients. The study reported success in achieving satisfactory hemostasis in the vast majority of patients. Other studies also provided safety and efficacy of the FoE suture for achieving immediate hemostasis after using large-caliber venous sheath access in pediatric patients.[4,5]

Figure-of-Eight Suture after AF Ablation

AF ablation is the procedure utilizing large-diameter venous sheaths, multiple femoral venous accesses, and high-intensity anticoagulation. Therefore, it can raise the chance of vascular complications. Sarah Traullé, et al conducted a prospective study to evaluate the feasibility and safety of venous FoE sutures to achieve hemostasis after AF ablation.[6] One hundred and twenty-four patients were included in this study, and 90% of the patients underwent cryoballoon AF ablation using a 15 Fr outer diameter sheath. After the ablation procedure, there were no patients who received reversal anticoagulant treatment. However, manual compression was needed in 10 patients. Hematomas were found in three patients (2.4%). Kudret Aytemir, et al. conducted another prospective study confirming the efficacy of FoE sutures.[7] This study included 200 patients who underwent cryoballoon AF ablation and were assigned into two groups. One hundred patients were assigned to the FoE suture group and the other one hundred patients were assigned to the conventional manual compression group. The median time to hemostasis and the time spent in the holding area were significantly shorter in the FoE suture group compared to the conventional manual compression group. No patient in the FoE group had a vascular complication. In contrast, 4% of the conventional manual compression group patients developed hematoma/pseudoaneurysm within 24 hours after sheath removal. However, the time to mobilization and duration of hospital stay was not different between groups.

Although the early studies reported a very low rate of vascular complication, recent studies revealed some risks of this complication. Umashankar Lakshmanadoss, et al reviewed the medical records of 284 patients who underwent AF catheter ablation.[8] There were two groups. The first group comprised 105 patients whose femoral venous hemostases were achieved by manual compressions once their activated coagulation time (ACT) was less than 180 seconds. The second group comprised 179 patients whose femoral venous hemostases were achieved by the FoE sutures, irrespective of their ACT. The major hematoma occurred in 3.9% of the FoE suture group, significantly lower than 10.5% of the manual compression group. Vineet Kumar, et al. conducted one randomized study.[9] Seventy patients were randomized to either manual compression or

FoE suture to achieve femoral vein hemostasis after undergoing cryoballoon AF ablation. The time from sheath removal to the patient leaving the laboratory room was significantly shorter in the FoE suture group. Moreover, 29% of the manual compression group required additional pressure in the recovery room. However, there was only minor hematoma or bleeding occurred in this study. Six percent of each group developed this complication. Therefore, no difference in vascular complication was found between both groups.

Yasuharu Matsunaga-Lee, et al conducted a study to assess the predictors of bleeding complications after performing the FoE suture in patients who underwent AF ablation with uninterrupted oral anticoagulants.[10] The 198 patients underwent radiofrequency ablation, and 89 patients underwent cryoballoon AF ablation. Multiple factors were included for analysis, such as patients' clinical characteristics, renal function, echocardiographic parameters, antiplatelet therapy, ablation strategy, and ACT at the end of the procedure. In the multivariate analysis with logistic regression after an adjustment for antiplatelet therapy, cryoballoon AF ablation was an independent predictor of increased bleeding complications with an odds ratio of 2.77.

Modified Figure-of-Eight Suture

Hikmet Yorgun, et al propose a new technique, modified FoE suture, to immediate venous hemostasis after cryoballoon AF ablation.[11] After suturing, both ends of the silk were passed through a three-way stopcock without knotting. The tip of the three-way stopcock was placed over the access site. Then the three-way stopcock valve was locked by turning toward 90º to the off position. They conducted a study to evaluate the efficacy of this technique. There were 75 patients allocated to use modified FoE sutures and standard FoE in the other 75 patients. Immediate hemostasis was significantly higher in the modified FoE group (100%) compared to the standard FoE group (90.7%). In-hospital rebleeding was observed in 2 patients and early local access site infection was found in 2 patients of the standard FoE group. No vascular complication was found in the modified FoE group. Therefore, this new approach is feasible and might be safe as well as time-saving. This can be an alternative technique to achieve immediate hemostasis of the femoral vein after cryoballoon AF ablation.

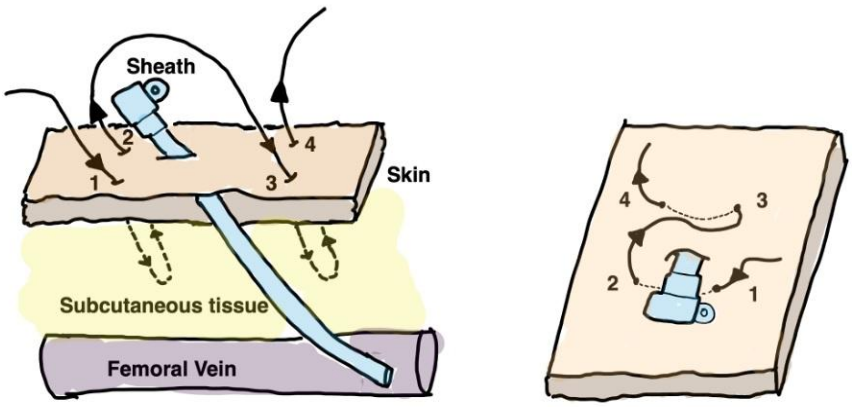

Figure 01. Steps of Figure-of-Eight Suturing

Figure-of-Eight Suture vs Vascular Closure Device

Recent studies showed the efficacy of achieving femoral venous hemostasis and shorter bed rest time with vascular closure devices after AF ablation. These studies demonstrated the results with vascular closure devices compared to manual compression. Moghniuddin Mohammed, et al published the insight from VAscular Closure for Cardiac Ablation Registry.[12] A total of 434 patients were divided into three groups according to the hemostasis techniques. There were 156, 203, and 75 patients in the manual compression, FoE suture, and vascular closure device group. The study revealed a significantly shorter time to hemostasis in the vascular closure device group compared to other groups. There was no difference in time to ambulation between the FoE suture and vascular closure device groups but significantly higher in the manual compression group. However, there was no difference in the length of stay. Ten patients developed minor vascular complications, which were not significantly different. These occurred in four patients in the manual compression group, three patients in the FoE suture group, and three patients in the vascular device closure group.

Conclusion

A “Figure-of-Eight" suture is a safe and effective technique for achieving immediate femoral venous hemostasis after AF ablation. There is growing evidence that this approach decreases the risk of vascular complications and procedure time. Using the "Figure-of-Eight" suture as a first-line approach is practical to achieve hemostasis after AF ablation.

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WEBINAR SUMMARY: EP JOURNAL CLUB 2023 (Part 1)

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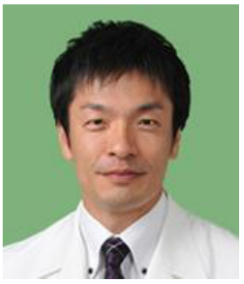
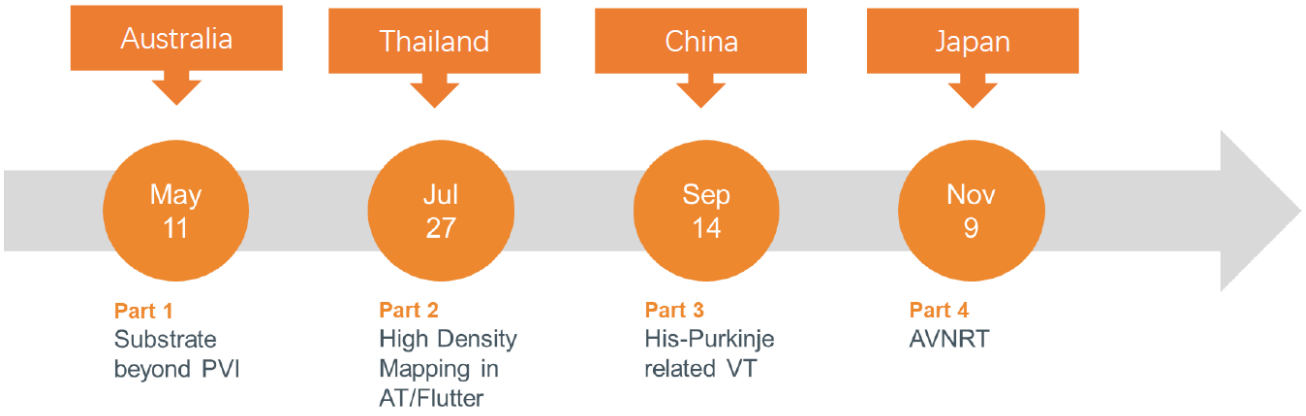
APAC EP Journal Club

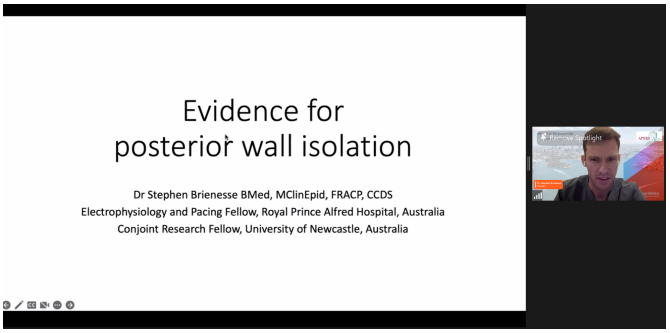
A series of educational sessions

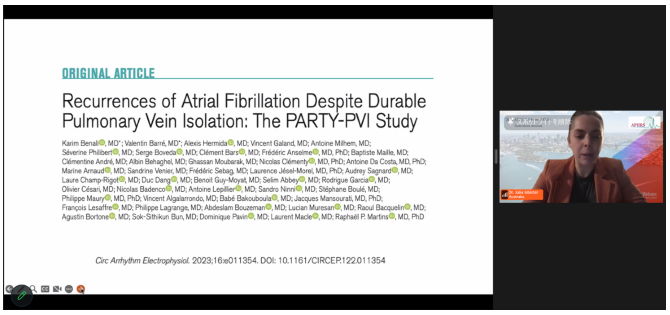
Faculty– Part 1	Faculty – Part 2	Faculty – Part 3	Faculty – Part 4
			
Prof. Raymond Sy	**Prof. Sirin Apiyasawat**	**Prof. Minglong Chen**	**Dr. Akira Mizukami**
University of Sydney	Ramathibodi Hospital	The First Affiliated Hospital of Nanjing Medical University	Kameda Medical Center



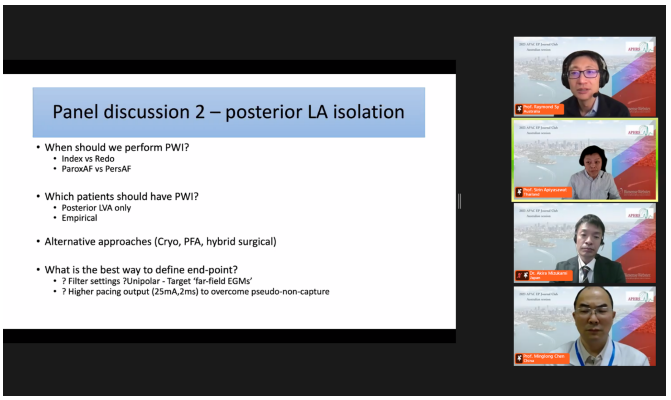
On May 11, 2023, the first session of EP Journal Club (EPJC) 2023 was hosted by the electrophysiology team led by Professor Raymond Sy, Professor Sy practices electrophysiology at the Royal Prince Alfred Hospital in Sydney, Australia. Prof Sy is an academic faculty member at the University of Sydney and is a director of arrhythmia services at the Concord Hospital also in Sydney. EPJC is a professional education event initiated by Biosense Webster, Johnson & Johnson, with Electrophysiological experts from China, Australia, Japan and Thailand. The experts from different countries share electrophysiology literature on a rotating basis, categorized by different topics, they are responsible for one topic each and lead the audience through the literature progress and share their hands-on experience. This course is aimed at Electrophysiologists with 1-5 years of experience, this event targets an audience in the Asia-Pacific region and has been endorsed by APHRS this year.

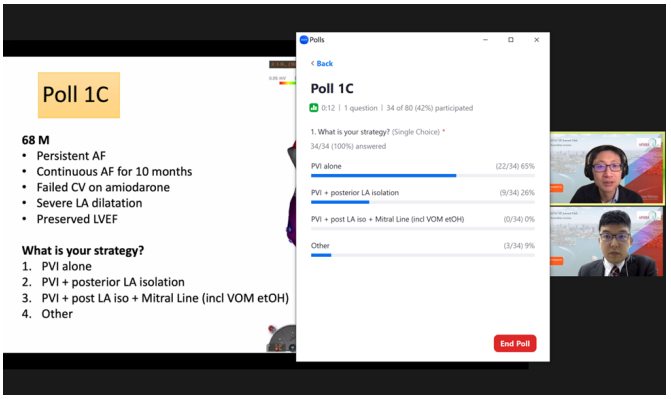
The theme of this event was "Substrate beyond PVI". Prof Sy led the Australian faculty which included Dr Stephen Brienesse and Dr Julia Isbister, both from the Royal Prince Alfred Hospital in Sydney, Australia. They shared their knowledge using practical case experience when dealing with the challenges of substrate beyond PVI. Low voltage areas, posterior wall isolation and linear mitral line ablation were discussed. Rationale, evidence, techniques and limitations were discussed by the faculty which led to excellent Q & A from our audience. The presentation utilized a case based approach with a selection of recent seminal journal publications.

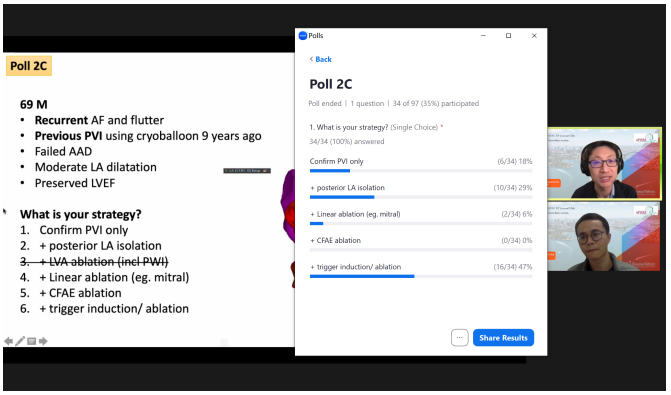




In addition to the excellent lectures and sharing by Prof Raymond Sy and the Australian faculty the evening had moderators, Prof. Minglong Chen from China, Dr. Akira Mizukami from Japan, and Prof. Sirin Apiyasawat from Thailand. They shared their valuable experiences during the discussion sessions. Through the wonderful lectures, various interactions, polling, case reviews, Q&A discussion, an audience of more than 120 people attended this event and expressed that they had a deeper understanding of substrates beyond PVI.







Virtual webinars have become a great platform to bring our experts and new electrophysiologists in the field together to share and learn from each other. We look forward to future sessions of the APAC EPJC, next session will be on July 27th 2023, please look out for registration information in the coming weeks.

Clinical Viewpoint: Minimizing Access Site Related Complications for Improved Patient Care by Using Suture Mediated Closure and Repair (SMCR) System in my Ablation Cases

Dr. Emily Kotschet
Cardiologist and Electrophysiologist, Monash Heart Rhythm, Victoria Heart Hospital, Australia

In contrast to conventional techniques for vascular closure, a Suture Mediated Closure and Repair (SMCR) system offers significant advantages in minimizing patient discomfort and the duration of the procedure. In addition, it may decrease the likelihood of complications such as bleeding, pseudoaneurysm, and hematoma formation. This article focuses on Suture Mediated Closure and Repair (SMCR) system and how they enable improved patient and procedural outcomes. I will discuss my viewpoints and personal experience of including Suture Mediated Closure and Repair (SMCR) system in my workflow for catheter ablation, and how it has helped further minimize complication rates, thereby facilitating patient and physician satisfaction.

Femoral venous access site complications are one of the most common complications seen with percutaneous catheter ablation procedures and can be associated with increased length of stay and reduced patient satisfaction, given groin pain and bruising, as well as significant resource consumption, particularly with large-bore sheaths for Atrial Fibrillation (AF) ablation. Although venous access may seem to have a lower risk of complications, compared with arterial access, patients are anticoagulated with heparin for the procedure, thus potentially having increased bleeding complications. For patients having redo procedures, with more fibrous change in the groin, the complication rate is higher in patients than those with no prior femoral venous access.

Initially, we used manual compression for closure, which is time intensive for staff, then switched to the Figure-of−eight Suture (FO8) and were quite satisfied with the results. The FO8 provides good hemostasis in most patients, with a small learning curve. However, it still requires the patient to lie flat in bed for several hours, and subsequently for suture removal four hours post-procedure. Patients cannot mobilize until it is removed. Therefore, when we read about the Suture Mediated Closure and Repair (SMCR) system for use with femoral venous access, we decided to try it for our catheter ablation patients, given that they are day case procedures, and may aid early mobilization and discharge, potentially with fewer complications. From a learning standpoint, deployment of the Suture Mediated Closure and Repair (SMCR) system is straightforward. There is a step-wise approach with numbering on the device to remind you of the steps. It is easily learned in 3 - 4 cases. There are explanatory videos, and a realistic simulation model to practice before your first case. I would recommend watching a colleague use the system, to see its simplicity, then use it with 7 or 8Fr sheath cases initially, gaining confidence in deployment. Thereafter you can learn to close larger bore sheaths. Patient satisfaction is an important procedural outcome. The patient can benefit most in terms of minimal bruising, and early mobilization, despite high dose heparin for AF ablation procedures. The femoral venous access closure is achieved on the table, at the end of a case, with no ooze. Even with an Activated Clotting Time (ACT) >300ms, the patient can mobilize two hours later.

I have a lot of positive feedback from the nursing staff being relieved of the significant burden of manual pressure, or ongoing assistance and monitoring of FO8 suture for multiple hours post-procedure. With our growing healthcare burden, this advanced closure protocol can help save time for the physician and the nursing staff, leading to the most efficient usage of their time. The study (Del Prete et al., 2020) discusses the benefits of suture mediated closure device, how they promote early ambulation and can significantly improve patient comfort by reducing the length of hospital stay, thereby translating into cost-benefits for the healthcare service, along with the clinical benefit to the patient. These benefits prompted us to learn more about the vascular closure device and then integrate it into our practice.

A Suture Mediated Closure and Repair (SMCR) system can be advantageous in cases where FO8 suturing is difficult such as in patients with high body mass index (BMI). Whilst it can be challenging using the suture mediated closure in obese patients, it does provide better hemostasis by mechanical closure of the venotomy, allowing for primary intention healing to occur. Our colleagues from the Cardiology Department have been using the system routinely and I soon learned that it could be used with equal efficiency in larger bore sheath sizes as well (Kar S et al., 2018).

I believe the main barriers in using a Suture Mediated Closure and Repair (SMCR) system are lack of awareness of this therapy for femoral venous closure, and overall resistance to learning a new technique, when we have an existing, reasonably simple therapy. I learned about the system from a colleague, who started using it post-AF ablation with high ACT measurements and achieved hemostasis immediately after deploying the SMCR system at the end of the procedure with no manual pressure required. The nursing staff recovering the patient reaped the benefits immediately, with a significant reduction in nursing time required, and enabled the change in workflow. Often the uptake of a new technique or technology is limited by concern regarding its safety and efficacy. We are conducting a randomized controlled trial comparing Suture Mediated Closure and Repair (SMCR) system with FO8 standard care to assess procedural safety and outcomes and patient experience. I know of several electrophysiologists across Asia and Australia who have adopted this modality of closure routinely, for better patient care, and it has been used by other leading electrophysiologists globally, to improve their procedural outcomes (Verma S., 2019).

In summary, Suture Mediated Closure and Repair (SMCR) system has facilitated a reduction in complications, while enhancing the procedural satisfaction and convenience in my patients undergoing catheter ablation. The system is safe to use, easy to learn, and has significant benefits in reducing recovery time. Additional benefits include early mobilization and reduced nursing resource time, which leads to potential cost savings for hospitals.

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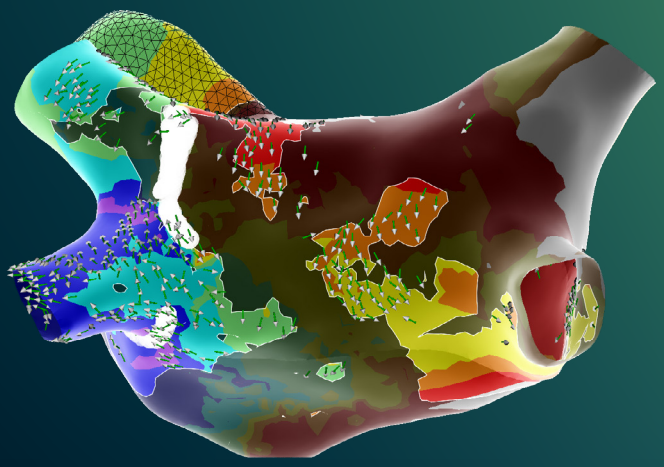

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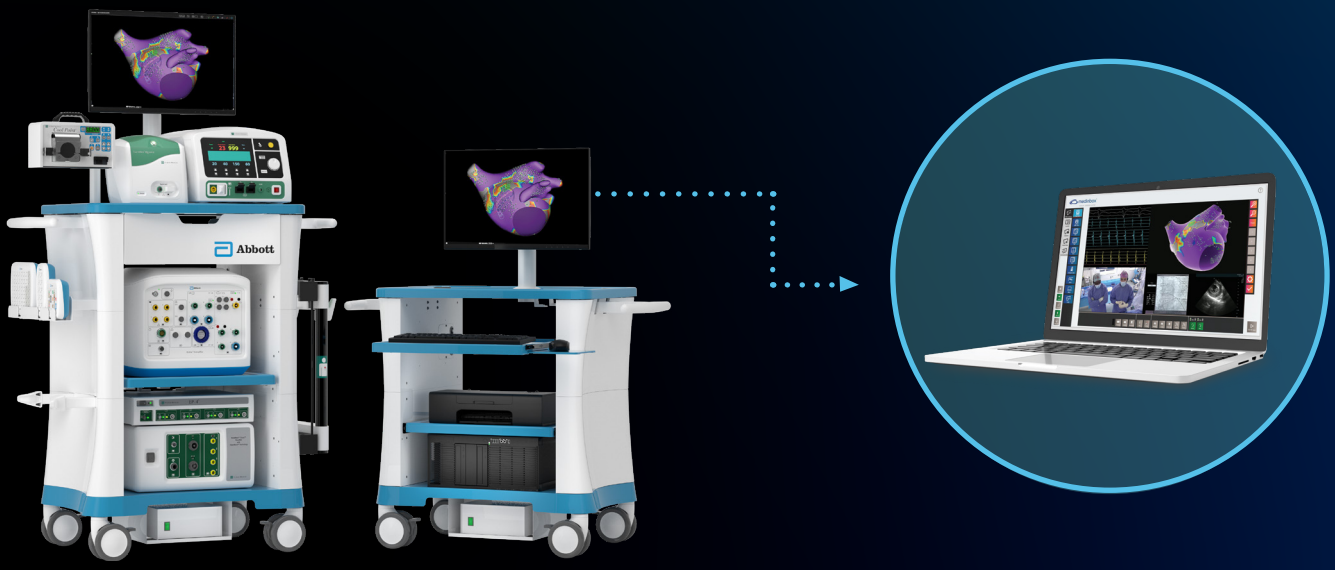

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