APHRS NEWSLETTER

SEPTEMBER 2023 | NO.68

Contents

A Case Presentation: Ablation of Left Ventricular Summit Ventricular Arrhythmias

Wen-Han Cheng [1,2], Ting-Yung Chang [2,3], Hsuan-Ming Tsao [1,2]

[1] Department of Medicine, National Yang Ming Chiao Tung University Hospital, Yilan, Taiwan

[2] Institute of Clinical Medicine and Cardiovascular Research Institute, National Yang Ming Chiao Tung University, Taipei, Taiwan

[3] Heart Rhythm Center and Division of Cardiology, Department of Medicine, Taipei Veterans General Hospital, Taipei, Taiwan

Catheter ablation of ventricular arrhythmias originating from the Left Ventricular Summit (LVS) area is challenging, owing to the proximity to the coronary arteries and anatomic barriers[1,2]. Detailed mapping of the neighboring areas including Right Ventricular Outflow Tract (RVOT), Left Ventricular Outflow Tract (LVOT), coronary cusps, and coronary venous systems are often required[3,4].

We presented a case report demonstrating the importance of detailed mapping and alternative ablation strategies.

A 76-year-old man with hypertension, type 2 diabetes mellitus, and coronary artery disease visited the cardiac electrophysiology clinic because of recurrent LVS ventricular arrhythmias and failed Radiofrequency Catheter Ablation (RFCA) attempts in another hospital.

Based on the medical records and documented images, LVS Ventricular Premature Complex (VPC) was diagnosed and ablation lesions were ever deployed at the septal wall of RVOT, sub-valvular area of the Left Coronary Cusp (LCC), distal Coronary Sinus (CS), Great Cardiac Vein (GCV), and Anterior Interventricular Vein (AIVV). However, VPC recurred and was refractory to medications.

On admission, a 12-lead Electrocardiogram (ECG) revealed monomorphic Ventricular Premature Complexes (VPC) with inferior axis, early precordial leads transition, and RS pattern on lead I *(Figure 1)*. Twenty-four-hour Holter monitor demonstrated frequent monomorphic VPCs (19085 beats, with 18% VPC burden) and several episodes of non-sustained ventricular tachycardia. Compared with the previous 12-lead ECGs, identical QRS morphologies and axis were appreciated *(Figure 2)*. Transthoracic echocardiogram revealed mildly dilated LV with an ejection fraction of 46%. Cardiac magnetic resonance imaging showed patchy and mid-wall late gadolinium enhancement at basal anterior, basal anteroseptal, mid anterior, and mid anterolateral segments of LV.

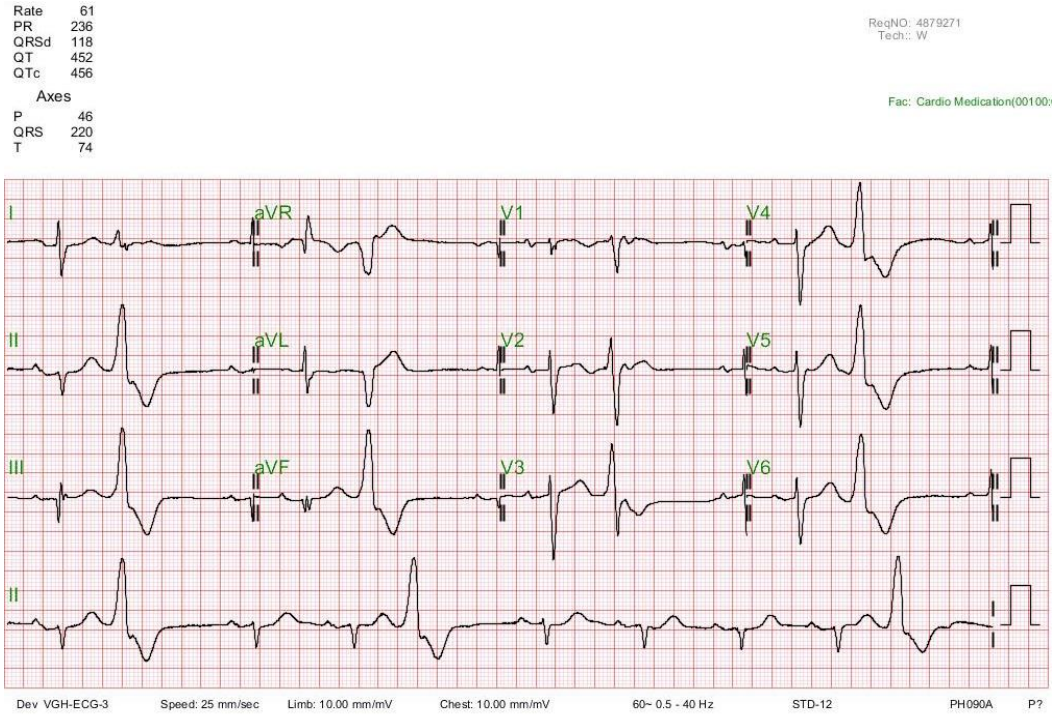


Figure 1. RS pattern on lead I

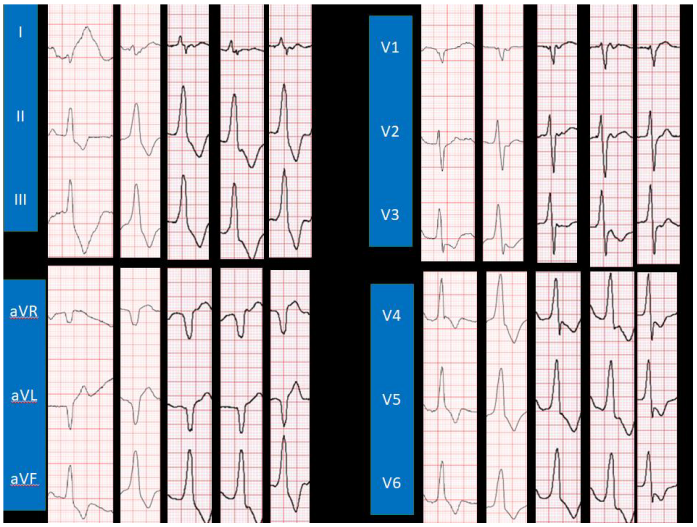


Figure 2. Identical QRS morphologies and axis were appreciated

After a thorough discussion with the patient and obtaining informed consent, another catheter ablation procedure was arranged. A 7.5 French, 3.5 mm open-irrigated tip ThermoCool® ablation catheter (Biosense Webster) and Carto 3D mapping system (Biosense Webster) were used to create anatomic and activation maps. Sequential activation mapping of RVOT, supra-valvular and sub-valvular areas of LCC, distal CS and GCV/AIVV was performed. The activation time was defined as the interval from the earliest bipolar electrogram on the distal end of the mapping catheter to the earliest onset of QRS complex on a 12-lead ECG. Among all mapped areas, GCV presented with the earliest activation time during VPC, which preceded the QRS onset by 50 ms *(Figure 3)*. The activation wavefront of VPC demonstrated an eccentric pattern, which spread out from GCV to RVOT/LCC and consequently to the LV cavity.

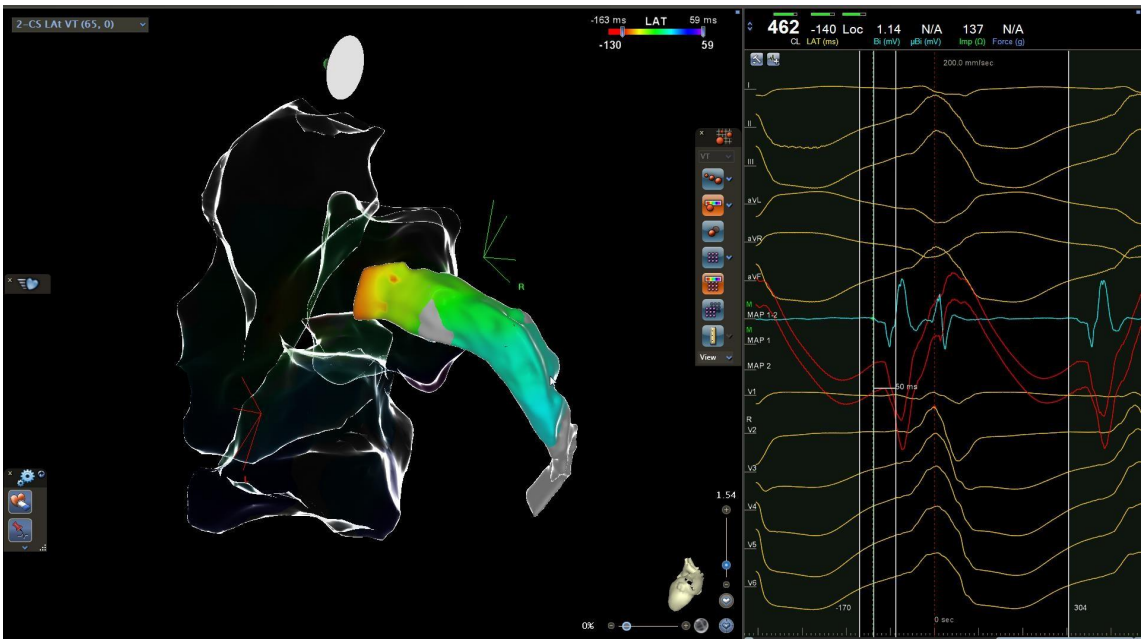


Figure 3. Among all mapped areas, GCV presented with the earliest activation time during VPC, which preceded the QRS onset by 50 ms

Before advancing the ablation catheter to GCV, a coronary venogram was performed; however, stenosis of proximal AIVV was observed, which might be related to previous RFCA in this area *(Figure 4A)*. Alternatively, a 2 French mapping catheter was introduced across this region and to the distal AIVV, which unveiled an earlier activation time, preceding the QRS onset by 57 ms *(Figure 4B)*.

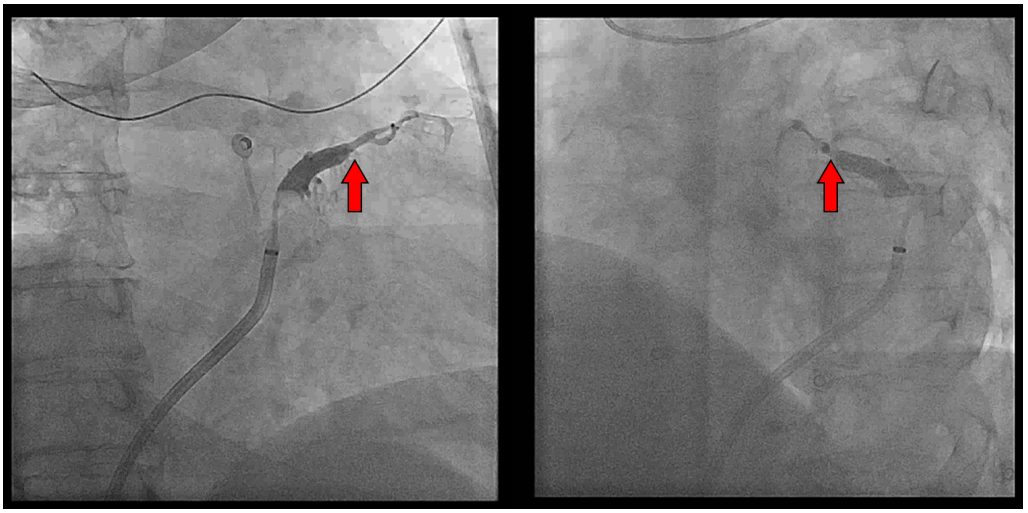


Figure 4A. Stenosis of proximal AIVV was observed, which might be related to previous RFCA in this area

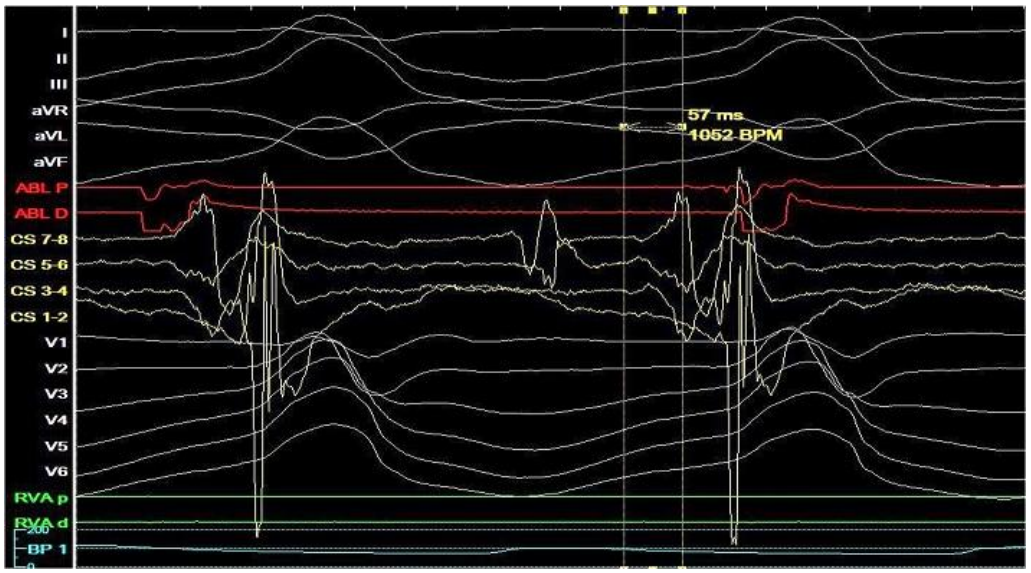


Figure 4B. An earlier activation time, preceding the QRS onset by 57 ms

Since RFCA was unfeasible due to stenosis of proximal AIVV, alcohol ablation into the coronary venous system was chosen. First, a 6 French coronary guiding catheter was introduced into the coronary sinus. Then an Alligator-clip connected coronary guidewire (ASAHI SION®) with an over-the-wire (OTW) balloon was advanced to the AIVV branch to detect the earliest activation region *(Figure 5)*. Once the AIVV branch with the earliest activation time was determined, the coronary guidewire was removed and the OTW balloon was inflated to prevent backflow. The efficacy test was performed with a trial of 3-time 1 ml cold saline at the selected AIVV branch before alcohol injection. After completion of 1 ml alcohol injection 3 times, the morphology and axis of VPC changed. Detailed mapping of the VPC was then performed and VPCs were ablated successfully at LCC sub-valvular area *(Figure 6)*. No major complications were noted. The patient remained VPC-free during the follow-up 3 months after the procedure.

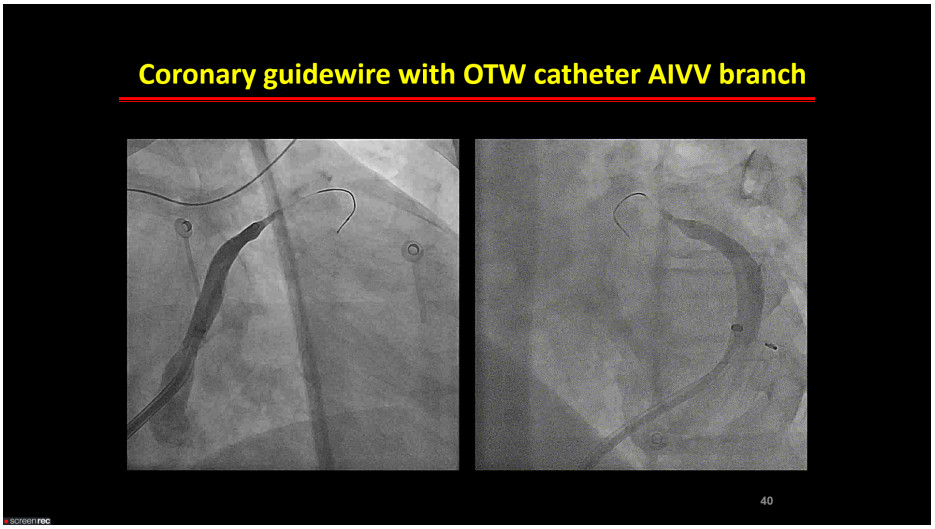


Figure 5. Coronary guidewire with OTW catheter AIVV branch

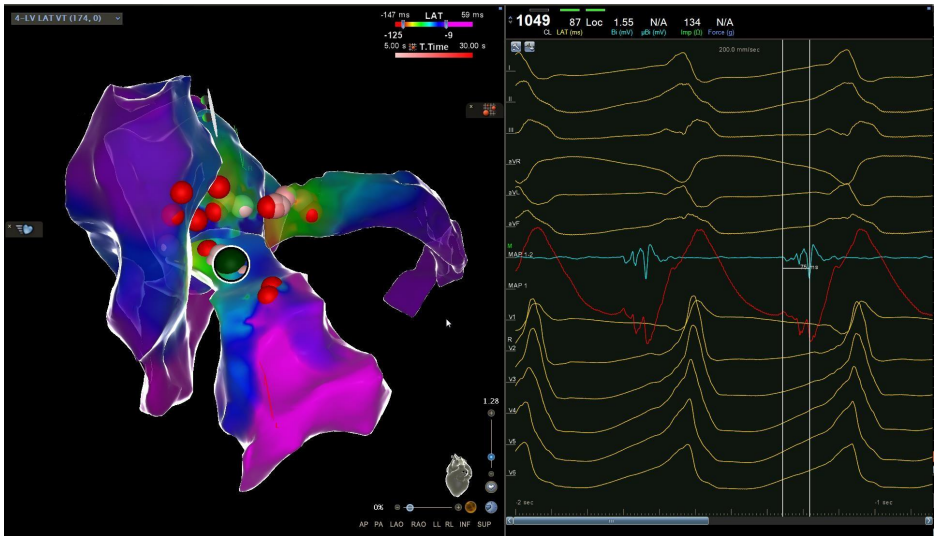


Figure 6. VPCs were ablated successfully at LCC sub-valvular area

In summary, detailed mapping of the neighboring areas around LVS is the key for successful ablation. Combination of ablation tools, including alcohol and catheter ablation, might be effective to achieve successful results for the complex arrhythmias.

References

1. Aksan G. Catheter Ablation of Left Ventricular Summit Arrhythmia in a Patient with Critical Coronary Artery Stenosis: A Sequential Approach. J Innov Card Rhythm Manag. 2020;11:4266-4271.

2. Chung FP, Lin CY, Shirai Y, Futyma P, Santangeli P, Lin YJ, Chang SL, Lo LW, Hu YF, Chang HY, Marchlinski FE, Chen SA. Outcomes of catheter ablation of ventricular arrhythmia originating from the left ventricular summit: A multicenter study. Heart Rhythm. 2020;17:1077-1083.

3. Enriquez A, Malavassi F, Saenz LC, Supple G, Santangeli P, Marchlinski FE, Garcia FC. How to map and ablate left ventricular summit arrhythmias. Heart Rhythm. 2017;14:141-148.

4. Lin CY, Chung FP, Lin YJ, Chong E, Chang SL, Lo LW, Hu YF, Tuan TC, Chao TF, Liao JN, Chang YT, Chen YY, Chen CK, Chiou CW, Chen SA, Tsao HM. Radiofrequency catheter ablation of ventricular arrhythmias originating from the continuum between the aortic sinus of Valsalva and the left ventricular summit: Electrocardiographic characteristics and correlative anatomy. Heart Rhythm. 2016;13:111-121.

GETTING TO KNOW APHRS LEADER

Kazuo Matsumoto, MD, PhD

Dean of Higashimatsuyama Medical Association Hospital, Emeritus Professor Saitama Medical University, Co-Chief Editor of Journal Arrhythmia

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

When I was in high school, I was not good at writing, and it annoyed me very much. I initially thought physicians are usually not good at writing, so I chose the medical course believing I would be able to survive. Fortunately, I passed the examination and entered Hiroshima University. When I was a medical student, I learned physiology from Professor Hiroshi Irisawa who found that the I_f channel has a role in heartbeat initiation. It is really mysterious how the heart beats by itself. During summer vacation, I visited Saitama Medical University and met Professor Dohi, a pioneer of clinical electrophysiology in Japan doing animal studies. This motivated me to choose electrophysiology for my life's work. After graduating from Hiroshima University, I chose the cardiology department in Saitama Medical University located nearby my home town.

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

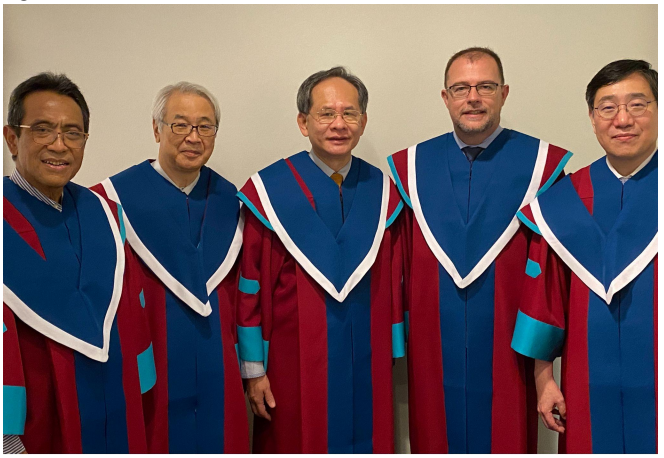
I cannot choose only 1 as there are 3 developments. 1) Development of leads implantation and ex-plantation methods were something since physician become capable in implanting pacemakers instead of the surgeon - Many doctors could then become engaged in therapy for bradyarrhythmia and to bail out from troubles by methods of ex-plantation. 2) Radiofrequency ablation method and visualization system (mapping) of electromechanical changes was one development too. Before the technique of ablation was invented, we could only do the analysis of arrhythmia mechanisms and evaluation of efficacy of antiarrhythmics. After that, we became able to cure the arrhythmias, and visualization of electromechanical changes enhanced those, and more could find new mechanisms of arrhythmias. 3) Cardiac resynchronization therapy is the third development. Pacing is unphysiological, but CRT recovers heart functions. Implantable devices become more related to heart failure matters with this development.

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

I take pride in the fact that I could work on the introduction of many new devices and new techniques into Japan and reimbursement of those devices and techniques. For the purpose of achievement of EP doctors' work, the technical fee of electrophysiological techniques must be reasonably evaluated. I worked in this field for more than ten years. I organized some societies for EP colleagues (doctors, nurses and medical engineers) to stimulate each other and grow their knowledge and up-skill for patients' safety. And I am proud of working editorial works for JOA and APHRS news, for internationalization and development of APHRS with our international colleagues.

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

When I was young, I wanted to become a high school teacher, but I was not good at writing and gave up. After that, I wanted to be a Jet Pilot, took the examination and passed. However, I was really embarrassed at the too-detailed physical check and quit. If I did not quit, I could fly all over the world, and crack jokes with beautiful flight attendants.



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

I was most inspired by Dr. Hiroshi Irisawa, who was a teacher of mine from Hiroshima University until now. His attitude to research and life is respectable. And he suggested to me how to do well as a researcher, how to teach as a teacher and how to live as a man. Also, I was inspired by Dr. Warren Sonny Jackman and Hiroshi Nakagawa. I learned a lot through them about electrophysiology, and the way of analysis of intracardiac electrocardiogram and catheter ablation.

What are your hobbies and interests outside of medicine?

My hobbies at the moment are golfing, watching movies at home, especially dramas from the Chinese period, and watching baseball games of my favorite team, the "Hiroshima Carp". My most current interest now is ICD-11, which is an international classification of Diseases. I am working to introduce ICD-11 into Japanese society to make it easier to solve health problems of the world in the future and the improvement of ICD-11 itself for a more useful classification.

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

It was on the 11th of March; I flew to Fukuoka from Haneda to attend the JCS (Japanese Circulation Society) meeting. I could see Mount Fuji from the window during the flight - it was really nice. When the plane landed, I was waiting for the door to open and just behind it, a beautiful stewardess, and she told me "22!". I thought she meant "she was 22 years old". I engaged, asking with a smile that "she was beautiful but did not look so young as such. She said " No, it is not my age! It's temperature at Fukuoka, 22-degree Celsius!" with a big smile. I got outside, and finally realized it was warm in Fukuoka!

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

"Life is impermanent and fast." These are words from a novel by Souseki Natsume. Actually, I realized this recently, because I have already experienced my life for 70 years. You should not wait for a long time once you decide on things to do. And you should change yourself correspondingly to changes and do your best on it, even if it's not what you would like to do. It is a life.

How do you keep a healthy work/life balance?

I am taking vegetables first and only taking small amounts of carbohydrate everyday.

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

Artificial Intelligence will affect the way they shape the future care of arrhythmia patients a lot. With this technology, we will be able to prevent or diagnose arrhythmias more efficaciously even before it appears, and choose proper therapy methods or ways depending on the patient's conditions. For instance, AI may evaluate elderly people's condition whether they are frail or not, and applicable for ablation or not etc. Combined robotic technologies and AI may make automatic ablation possible in future.

Indian Heart Rhythm Society's DM-DNB Arrhythmia Teaching Program

Dr. Ashish Nabar
Cardiac Electrophysiologist, Mumbai, India

India has a well-established 3-year super-specialty postgraduate training program in Cardiology for many decades, now available in most states of the country. A Cardiology trainee joins this program after his 3-year postgraduate training in either General medicine, Pediatrics or Chest medicine (Pulmonology), and is awarded a DM (Doctor of Medicine, by the state or a deemed University) or DNB (Diplomate of National Board, an autonomous body under National Medical Council) degree after successful completion of the course and exit exams. In a given academic year, across the vast and varied geography of our country, at least 1500 cardiology postgraduates are in their training period. Hereafter, for further specialization in Electrophysiology and CIED implantation, a limited number fellowship / training possibilities, extending for 1-2 years, are now available under the auspices of select Universities or National Board of Examinations. Additionally, informal fellowship possibilities under the leadership of senior Indian Heart Rhythm Society (IHRS) faculty, extending for a variable duration, exist at different high-volume centers in the private set-up. In 2020, during the presidency of Dr Yash Lokhandwala, IHRS executive committee identified two lacunae in the capacity-building for practicing electrophysiologists in the country: i) not every cardiology training program had an electrophysiologist in its faculty, and ii) limited number of formal / informal EP fellowship training programs in the country. To augment the former, generate interest and train the cardiology postgraduate in arrhythmias, IHRS decided to start a once-a-month DM-DNB Arrhythmia Teaching Program.

Figure 1. A panel discussion, which includes answering questions raised by the cardiology postgraduates in the chat box. We pay our deep respects to Prof. Dr Mohan Nair (lower panel, middle), past IHRS president and organizing secretary 7TH APHRS, 2014, following his sad demise in February 2023.

For the benefit of Cardiology postgraduates training all-over India, the IHRS DM-DNB Arrhythmia Teaching Program is conducted online on a regular basis since its inception in December 2020. Early-career Cardiologists interested in Arrhythmias and Heart Failure and contemplating a fellowship in cardiac electrophysiology are also welcome to join. The program is conducted on the 1st Sunday of every month, starting at 11.30 hours and extending for 90 minutes. The content is curated such that it supplements the training that the cardiology trainee receives in his parent department. The uninterrupted continuation of the online program over more than 2 years has been possible due to the strong backing of the current IHRS executive committee headed by its President, Dr Ulhas Pandurangi. In the past, we have invited cardiology trainees from neighboring countries viz Bangladesh, Nepal, Sri Lanka and Vietnam through collegial contacts. This invitation can be formalized and extended to the HRS of other Asian countries, for their cardiology

trainees, upon emailing a request to the IHRS Secretary at: ihrssecretary@gmail.com. The program can be attended free-of-cost and recently IHRS has liaised with a sponsor, to assist with the logistics related to reaching out and broadcast of the program.

The invited Host faculty is drawn from various academic departments and private hospitals in India. IHRS members, across the country, have gladly taught in this program. The format is thematic and involves two didactic talks and 2 case presentations on a particular subject. Two experts, on the particular subject being discussed, are requested to moderate these sessions. A couple of times in the year, an international faculty is invited. Earlier, Dr Edward Rowland (London) and Dr Joe de Bono (Birmingham); and recently, Dr Abhishek Deshmukh (Rochester) and Dr Chenni Sriram (Michigan) have mentored in our program. The major emphasis is on interpretation of ECGs and intracardiac electrograms, to better the practical understanding of the postgraduate regarding cardiac arrhythmias. Hopefully, this would help them manage their patients better and update their preparation for the exit exams. A few of them might choose to become a cardiac electrophysiologist! Everyone can easily join the program through a prior e-circulated link or through the Medgami app.

Its exemplary how mentors from varied institutes, viz academic departments (AIIMS and GB Pant, New Delhi; SGPGI, Lucknow; KEM Hospital and LTMGH, Mumbai; SCTIMST, Trivandrum; SJICR, Bengaluru; CMC, Vellore), high-volume private hospital set-ups (Fortis Escorts, Medanta, Max Healthcare and FMRI, New Delhi; AIG and KIMS, Hyderabad; Amrita and Lissie, Kochi), and even practicing EP groups (Western India, Ahmedabad; Kolkata group; Pune group) have joined forces and given their time to educate the cardiology postgraduates. We feel, participating in this program, the junior faculty has been provided a stage to show-case their cases and improve their presentation skills. Serial editions of these programs have covered different aspects of Arrhythmias – Brady-arrhythmias and syncope, SVT mechanisms, wide QRS tachycardia, VT mapping, Arrhythmias in GUCH, tachycardiomyopathy and pacing-induced cardiomyopathy; as well as in CIEDs – pacemaker timing cycles, optimizing ICD programming, CRT implantation – indications and techniques, to name a few. In the future, we could involve the international Asian faculty for their expertise related to experimental electrophysiology and genetics

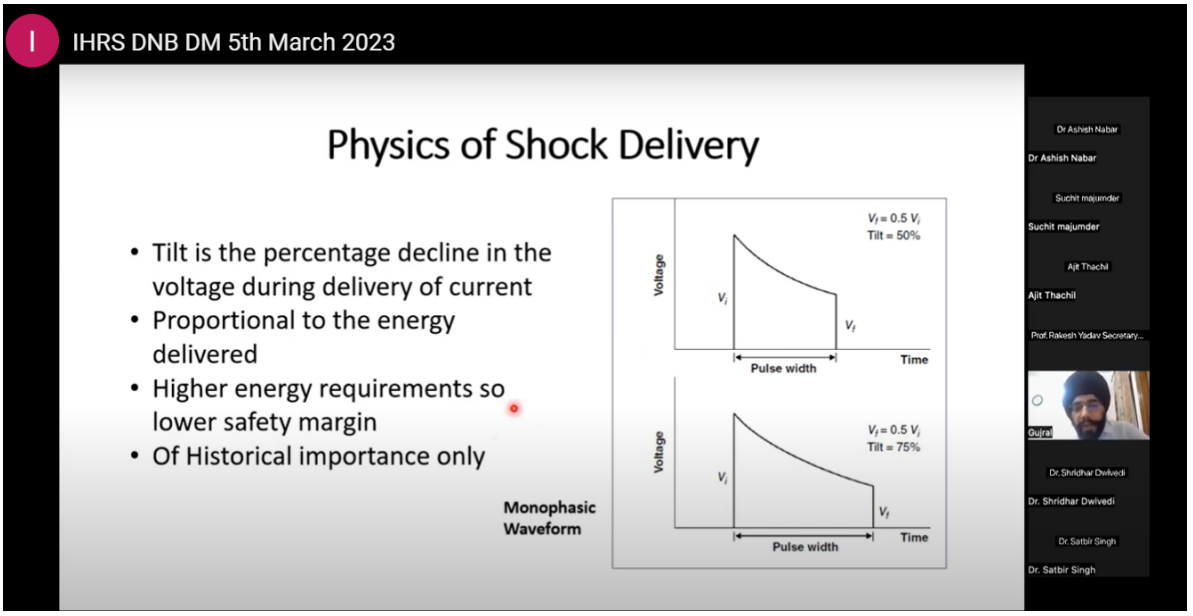


Figure 2. The participating junior faculty is trained for the bigger stage by presenting in the IHRS DM-DNB Arrhythmia Teaching Program

The ball has been set rolling; the bar needs to be set higher with every passing year. This is certainly a low-cost and far-reaching educational initiative of the IHRS. The cardiology postgraduate, from India and our Asian countries, needs APHRS to take up such a task on a round-the-year basis, completely gratis, to provide a cardiology trainee world-class training in cardiac arrhythmias. Food for thought?

Micra AV Troubleshooting - Leadless Pacemaker Programming and Troubleshooting

Dr. Jacky Kit Chan
Pro-Care Heart, Hong Kong

Introduction

Transcatheter leadless pacemaker system is associated with 63% reduction of 1-year major complications compared with conventional transvenous pacemaker[1]. However, the current Micra™ leadless pacemaker system only provides single chamber ventricular pacing. The lack of atrial sensing and tracking in the first generation leadless pacemaker systems results in atrio-ventricular (AV) dyssynchrony. The Micra™ AV leadless pacemaker (LPM) could sense atrial mechanical activities / heart sounds and achieve AV synchronous pacing up to approximately 100 beats per minute (bpm) in VDD pacing mode. The MARVEL 2 study[2] demonstrated that among patients with sinus rhythm and complete heart block, 95% of patients could achieve >70% AV synchrony in VDD pacing mode at rest (versus only 0% in VVI pacing mode). The mean percentage of AV synchrony increased from 26.8% under VVI pacing mode to 89.2% under VDD pacing mode. However, AV synchrony is more difficult to achieve during sinus tachycardia. Observational study[3] demonstrated that when sinus rate was above 80bpm, the median AV synchrony achieved with Micra™ AV LPM was only 33% (29-46%). As oversensing or undersensing of atrial mechanical activities could occur with change in physical activities, heart rate and posture, manual atrial mechanical (MAM) test to optimize atrial mechanical sensing is essential after implantation and during each clinic visit. This article summarizes the common programming and troubleshooting tips and tricks of the Micra™ AV LPM system.

Micra™ AV LPM Event Markers Nomenclature[4]

The Micra™ AV LPM utilizes a 3-axis accelerometer to detect cardiac mechanical contraction and heart sounds. The sensed atrial contractions are then followed by AV synchronous pacing in VDD mode. Each intra-cardiac mechanical signal is detected by the accelerometer in 3 vectors perpendicular to each other. The A1 signal represents the beginning of ventricular contraction (onset after QRS complex) and the closure of mitral/tricuspid valves. The A2 signal represents the end of ventricular contraction (coinciding with the end of T wave) and the closure aortic/pulmonary valves. The A3 signal represents passive atrial emptying and ventricular filling (between the end of T wave and the beginning of atrial contraction during ventricular diastole). It corresponds to the mitral E wave in echocardiogram. The A4 signal represents active atrial contraction (onset after P wave and before QRS complex of the next cycle). It corresponds to the mitral A wave in echocardiogram. There is a 100-millisecond electromechanical delay between the onset of P wave and onset of atrial mechanical contraction (A4). The atrial mechanical contraction sensed by the device in the A4 sensing window is labeled as AM in the marker channel. The A3 window refers to the interval in which the A3 signal is sensed, lying between the end of A2 and beginning of A4. Ventricular end (VE) marks the end of A3 window. During sinus or atrial tachycardia, fusion of A3 and A4 signals could occur. Differentiation of A3 from A4 could be difficult at high heart rates. Under such circumstances, the fused A3/A4 signal is labeled as A7 (A3+4=7). The A7 signal could be used as a surrogate of A4 for atrial tracking at high sinus/atrial rates.

Signal Markers	Mechanical events	Timing
A1	Closure of mitral and tricuspid valve at the beginning of ventricular contraction	Immediately after QRS
A2	Closure of aortic and pulmonary valve at the end of ventricular contraction	At the end of T wave
A3	Passive atrial emptying and ventricular filling in ventricular diastole (E wave in echocardiogram)	Between the end of T wave and QRS
A4	Active atrial contraction (A wave in echocardiogram)	Between P wave and QRS
A7	Ventricular filling phase (E/A fusion in echocardiogram)	During fusion of A3 and A4 signals at high heart rates
AM	Atrial mechanical contraction sensed by the device	In A4 window - above the A4 threshold (not seen in VVI mode)
AR	Atrial refractory event	Atrial signals detected during the PVAPR
VE	End of passive atrial emptying and ventricular filling	Ventricular end – marks the end of A3 window (not seen in VVI+ mode)

Table 1. Micra™ AV LPM intra-cardiac mechanical event timing and event markers nomenclature. Abbreviation: PVARP, post ventricular atrial refractory period.

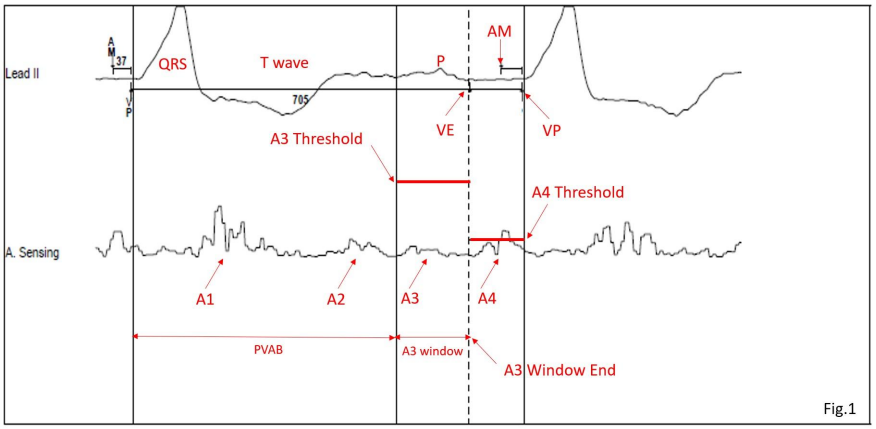


Figure 1. Micra™ AV events and timing. The upper tracing is the lead II ECG. The lower tracing represents the sensing of intra-cardiac mechanical events. A1 represents the beginning of ventricular contraction (onset after QRS complex) and the closure of mitral/tricuspid valves. A2 represents the end of ventricular contraction (immediately after the end of T wave) and the closure aortic/pulmonary valves. A3 represents passive atrial emptying and ventricular filling (between the end of T wave and the beginning of atrial contraction during ventricular diastole). A4 represents active atrial contraction (onset after P wave and before QRS complex of the next cycle). There is 100 milliseconds electromechanical delay between onset of P wave and onset of atrial contraction (A4). AM represents atrial mechanical contraction sensed by the device above the A4 threshold in the A4 sensing window. VE represents ventricular end – the end of A3 window. VP represents ventricular pacing. A3 window marks the interval in which A3 signal is sensed, lying between the end of A2 and the beginning of A4. PVAB represents post ventricular pacing atrial blanking window – it blanks the A1 and A2 signals and prevents atrial oversensing in the early ventricular diastole. A3 threshold and A4 threshold represent the sensing thresholds of A3 and A4 activities respectively.

Micra™ AV LPM Pacing Modes[4]

The device could operate in VDD, VDI, VVIR, VVI, VOO, ODO or OVO modes. In patients with intermittent AV block, when intact intrinsic AV conduction is detected, VDD automatically switches to VVI mode (40 bpm) to promote intrinsic AV conduction and minimize unnecessary right ventricular pacing. When patients develop AV block, VVI mode automatically switches back to VDD mode. When patients develop atrial tachycardia or sinus tachycardia, VDD mode automatically switches to VDIR mode. When physical activities stop, VDIR mode automatically switches back to VDD mode. During persistent sinus tachycardia, atrial sensing and tracking is difficult. Under such situation, automatic mode switch could be programmed off and the device could be programmed to VVI/VVIR mode to promote intrinsic AV conduction.

Pacing Rate and Programmable Intervals[4]

The lower rate limit nominal setting is 50 bpm. The device provides AV synchronous pacing in VDD mode. The upper tracking rate nominal setting is 105 bpm (range 80-115 bpm). The upper sensor rate nominal setting is 120 bpm (range 80-170 bpm). The device has automatic rate smoothing function. The nominal interval between atrial contraction and ventricular pacing (AM-VP) is 20 milliseconds (ms). This corresponds to sensed AV interval of 120 ms, as there is an electromechanical delay of 100 ms between P wave and atrial contraction (AM). Although AM-VP interval could be programmed up to 200 ms, long AM-VS is not recommended as it will affect the ability to track atrial activities at higher heart rate. In patients with intermittent AV block, promotion of intrinsic AV conduction should be achieved by AV conduction mode switch (e.g., changing from VDD mode to VVI/VVIR mode) rather AM-VP interval prolongation. The post ventricular atrial blanking (PVAB) blanks A1 and A2 signals and prevents atrial oversensing in early diastole. The nominal PVAB is 550 ms (range 450-600 ms). The device has automatic post ventricular atrial refractory period (PVARP) function (range 500-750ms) to prevent sensing of retrograde atrial signals. The tracking check algorithm automatically extends PVARP and checks for atrial oversensing when atrial tracking is occurring at high rates.

Recommended Parameters and Automatic Atrial Sensing Setup at Implantation[5]

The recommended optimal values for R-wave amplitude, impedance and pacing threshold at device implantation are ≥5 millivolt (mV), 400-1500 ohms and 1 volt (V) respectively. The automatic atrial sensing setup automatically starts 3 minutes after removal of programming head/telemetry. It should not be started while the tether is attached to the leadless pacemaker, as the mechanical stretch on the device by the tether may affect sensing of intrinsic cardiac mechanical activities. The atrial sensing setup automatically selects the optimal parameters including the sensing vector, A3 threshold, A4 threshold, A3 window end, minimum/maximum A3 window end. It collects 5 minutes of A3 and A4 amplitude data in each dual and triple vector combination in VDI mode (sum of 2 combination or sum of 3). It operates in VDI mode for 2 minutes and VDD mode for 2 minutes for tuning the final optimal setting.

Manual atrial mechanical (MAM) test[5]

The MAM test should be performed before discharge after device implant and during each device clinic visit. It collects electrocardiogram (ECG), accelerometer waveform, markers, and marker intervals data from the live waveforms. The accelerometer signals collected could be reviewed and the most ideal cardiac cycle for adjusting atrial sensing parameters could be selected. An ideal cardiac cycle should have clear P waves in diastole, clear and identifiable A3 and A4 signals. The A3 window end, A3 and A4 threshold values are superimposed on top of the rectified accelerometer signals displaced on the cardiac cycle tracings. The parameters could be manually adjusted and optimized. After optimization, the MAM test should be repeated to test the new parameters before final reprogramming.

Troubleshooting of oversensing and undersensing[5]

A4 undersensing[5]

A4 undersensing could be caused by high A4 threshold, long A3 window end, low A4 amplitude, posture change, exercise or sinus tachycardia. The device algorithm automatically measures A4 signals in each accelerometer vector and adjusts A4 threshold below the A4 signal to ensure accurate A4 sensing. It automatically adjusts threshold at ±0.1m/ s^2 intervals after every 8 beats. The nominal A4 threshold is 1.2 m/ s^2 (range 0.7 - 8.0 m/ s^2). The optimal amplitude of the A4 signal should be at least 1.5 times that of the A4 sensing threshold.

A4 undersensing due to suboptimal A4 threshold or low A4 amplitude

In case of A4 undersensing secondary to high A4 threshold *(Figure 2)* or low A4 amplitude, the threshold should be decreased (increasing sensitivity). However, the A4 threshold should be maintained higher than the A3 amplitude to prevent oversensing of A3 (except under the situation of A7 tracking in sinus tachycardia). The A4 amplitude should also be re-checked in different vectors. If A4 amplitude is too low in 1 vector, other different vectors or combination of vectors with the most optimal A4 amplitude should be chosen.

A4 undersensing due to suboptimal A3 window end

The device algorithm automatically adjusts A3 window end at intervals of ±15ms every 8 beats within the programmed upper and lower limits (Maximum A3 window end and minimum A3 window end). The target A3 window end is calculated from the median values of the last 8 beats. In VDD operation, the measurement of A3 window end is set at 1/3 of the interval between the end of A3 and peak of A4. At least 100 ms separation between A3 and A4 is needed for accurate A3 window end assessment. In VDI operation, the A3 window end is set at the end of A3 signal plus 50 ms. The recommended optimal programming for minimum and maximum auto A3 window end are optimal A3 window end plus and minus 50 ms respectively. The nominal setting for A3 window end is 775 ms (range: 600-1000 ms). The nominal minimum and maximum auto A3 window end values are 750 ms and 900 ms respectively. Too long A3 window results in A4 undersensing *(Figure 3)*. This should be managed by shortening A3 sensing window to allow appropriate sensing of A4. However, the A3 sensing window should be kept long enough to exclude A3 to avoid A3 oversensing.

A4 undersensing due to postural change, exercise or sinus tachycardia

In sinus tachycardia, shortening of R-R interval could result in A3 and A4 fusion. The A3 and A4 signals are sometimes sensed as a merged A7 signal. It could be difficult to differentiate A3 from A4 under such setting. In patients with sinus tachycardia and consistent A7 sensing, the auto A3 auto threshold function could be turned off and the device could be programmed to sense the merged A7 signal as a surrogate of A4, to allow atrial tracking at higher heart rate. If oversensing is suspected to be caused by posture change or physical activity, postural sensitivity test and MAM should be conducted both at rest and after exercise.

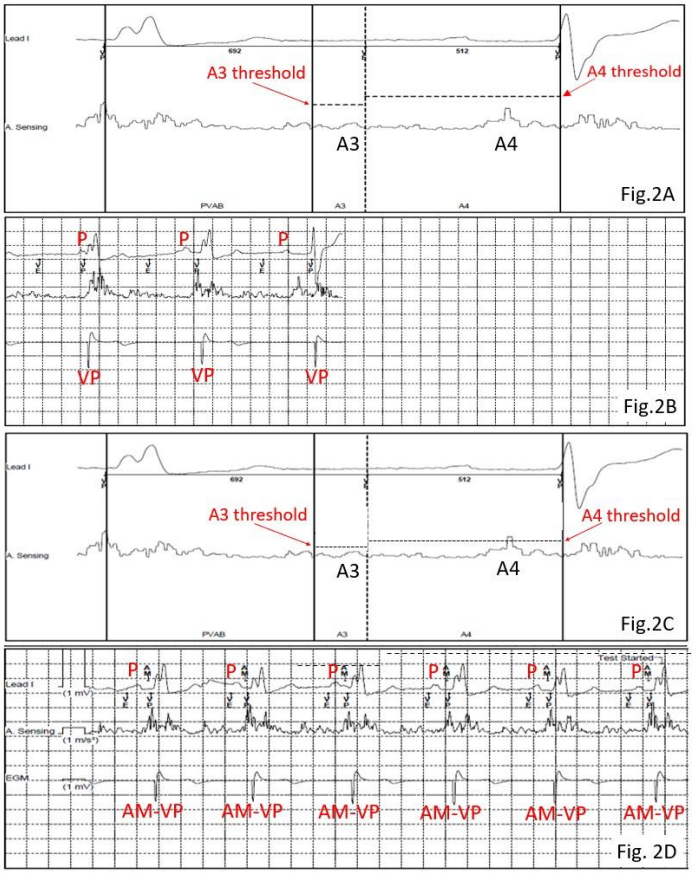


Figure 2. A4 undersensing due to high A4 threshold. 2A: Before manual atrial mechanical (MAM) test, A4 threshold was higher than the A4 amplitude, resulting in A4 undersensing. 2B: A4 signal undersensing resulting in loss atrioventricular synchronous pacing. There was no AM event after each P wave. The ventricular pacing was not atrial synchronized. 2C: After increasing the sensitivity of A4 threshold (from 5.5 m/ s^2 to 2.0 m/ s^2), the A4 events were sensed. It is imperative to set the A4 threshold lower than the A4 amplitude but higher than that of A3, to avoid oversensing of A3. 2D: After decreasing the A4 threshold, the atrial events were sensed (AM) and tracked, achieving AV synchronous pacing was achieved (AM-VP). Abbreviations: PVAB, post ventricular atrial blanking; AM, atrial mechanical contraction sensed by the device; VE, ventricular end - the end of A3 window; VP, ventricular pacing; AM-VP, AV synchronous pacing.

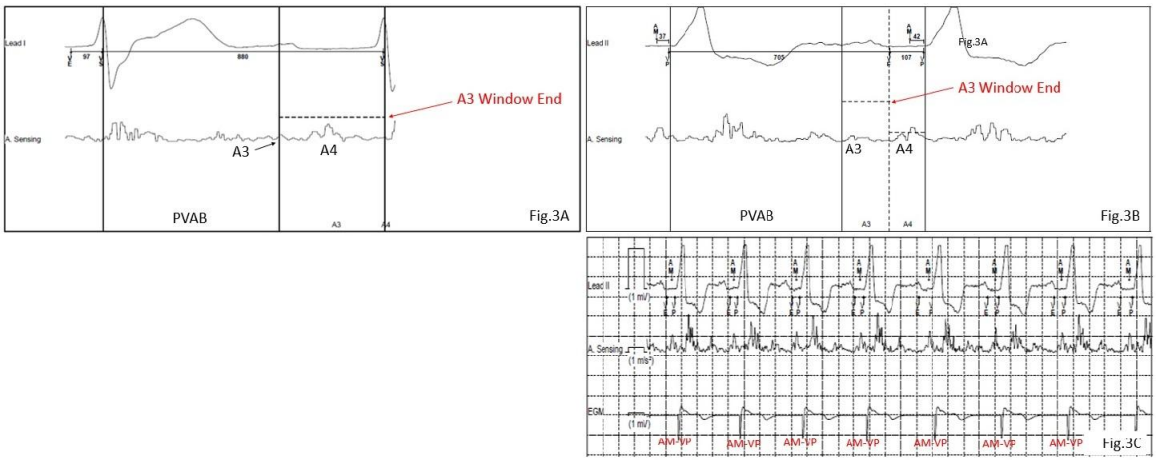


Figure 3. A4 undersensing due to long A3 window end. 3A: Before manual atrial mechanical (MAM) test, A3 window end extended beyond A4 signal, resulting in A4 undersensing. 3B: The A3 window end was shortened so that the A4 could be sensed – the A3 window end should land before beginning of A4. 3C: After shortening of A3 window end, the atrial events were sensed and tracked, achieving AV synchronous pacing (AM-PV0. Abbreviations: PVAB, post ventricular atrial blanking; VP, ventricular pacing; AM, atrial mechanical contraction sensed by the device; VE, ventricular end - the end of A3 window; AM-VP, AV synchronous pacing.

A3 oversensing

A3 oversensing can occur under the following circumstances: Low A3 threshold, short A3 window end, postural change, exercise or sinus tachycardia. The device algorithm automatically measures the A3 signal in each accelerometer vector and adjusts the A3 threshold (above the A3 signals) to prevent oversensing of A3 as A4. It automatically adjusts threshold at ±0.1m/ s^2 intervals after every 64 beats and evaluates the threshold after every 8 beats. The maximum of A3 signal is calculated using the median value from the last 8 beats. The recommended target A3 threshold is A4 threshold plus 1.5 times the maximum amplitude of A3. The nominal A3 threshold is 4m/ s^2 (range 1.0-10.0 m/ s^2).

A3 oversensing due to low A3 threshold

In case of A3 oversensing (as A4) secondary to low A3 threshold (Figure 4), the threshold should be increased (decreasing sensitivity).

A3 oversensing due to suboptimal A3 window end

Too short A3 sensing window results in A3 oversensing (oversensing of A3 as A4). This could be managed by prolonging A3 sensing window, but it should be kept long enough to exclude A4 to avoid A4 undersensing.

A3 oversensing due to postural change, exercise or sinus tachycardia

The management is the same as that mentioned in the previous paragraph for A4 undersensing.

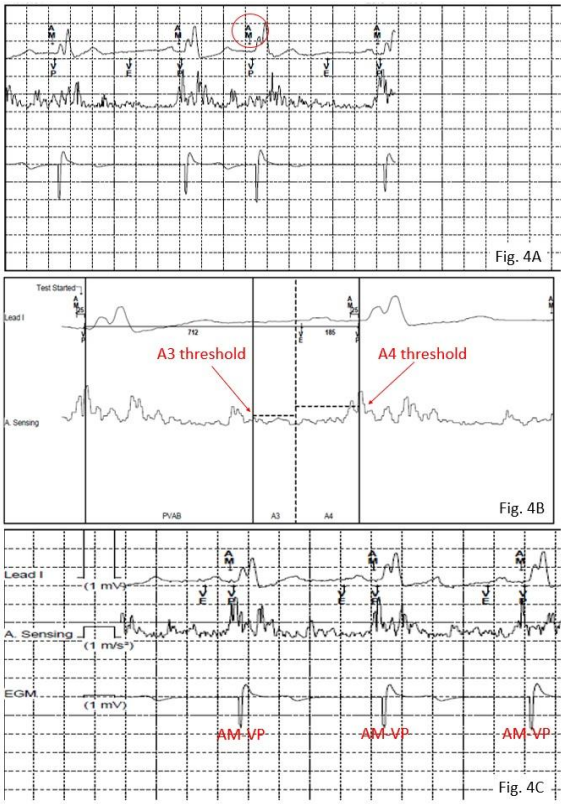


Figure 4. A3 oversensing due to low A3 threshold. 4A: There was an atrial over-sensed event (AM in red circle) in the absence of visible P wave in ECG. The A3 threshold was set to low (too sensitive). 4B: Reprogramming of A3 threshold to be less sensitive (higher than A3 amplitude). 4C: Restoration of AV synchronous pacing (AM-VP) with no more A3 over-sensing after reprogramming A3 sensitivity. Abbreviations: PVAB, post ventricular atrial blanking; AM, atrial mechanical contraction sensed by the device; VP, ventricular pacing; VE, ventricular end - the end of A3 window; AM-VP, AV synchronous pacing.

Postural sensitivity test[5]

The sensing of intra-cardiac mechanical motion is affected by postural change and physical activities. In patients who are physically active or who experience symptoms of pacemaker syndrome upon change of posture or physical activities, the postural sensitivity test could be performed to assess the sensing of atrial contraction under these conditions. The postural sensitivity test includes a 30-second test in right decubitus posture, 1-minute test in supine position, 30-second test in left decubitus posture, 1-minute test in sitting position, 1-minute test after walking followed by 1-minute test in supine position. The physical activity counts could be analyzed in all 3 vectors. At least >8 counts of separation should be allowed between rest and activity test. Low A4 amplitude could occur in some postures, resulting in A4 undersensing. The most optimal vector or combination of vectors with most optimal sensing and best reflection of the activity counts in different postures / during physical activities could be chosen for programming optimization.

Assessment of the efficacy of AV synchronous pacing

The percentage of AV synchronous pacing is displayed as AM-VP percentage in the pacemaker interrogation report. However, one should bear in mind that the reported AM-VP percentage does not always equate to the actual percentage of AV synchronous pacing, as oversensing of noise or A3 followed by inappropriate tracking could also be labeled as AM-VP by the device. Manual atrial mechanical test is essential in each device clinic visit to ensure appropriate atrial mechanical sensing and atrial tracking.

Proposed troubleshooting algorithm

A systematic approach to troubleshooting of common undersensing / oversensing problems is summarized in the proposed troubleshooting algorithm in Figure 5.

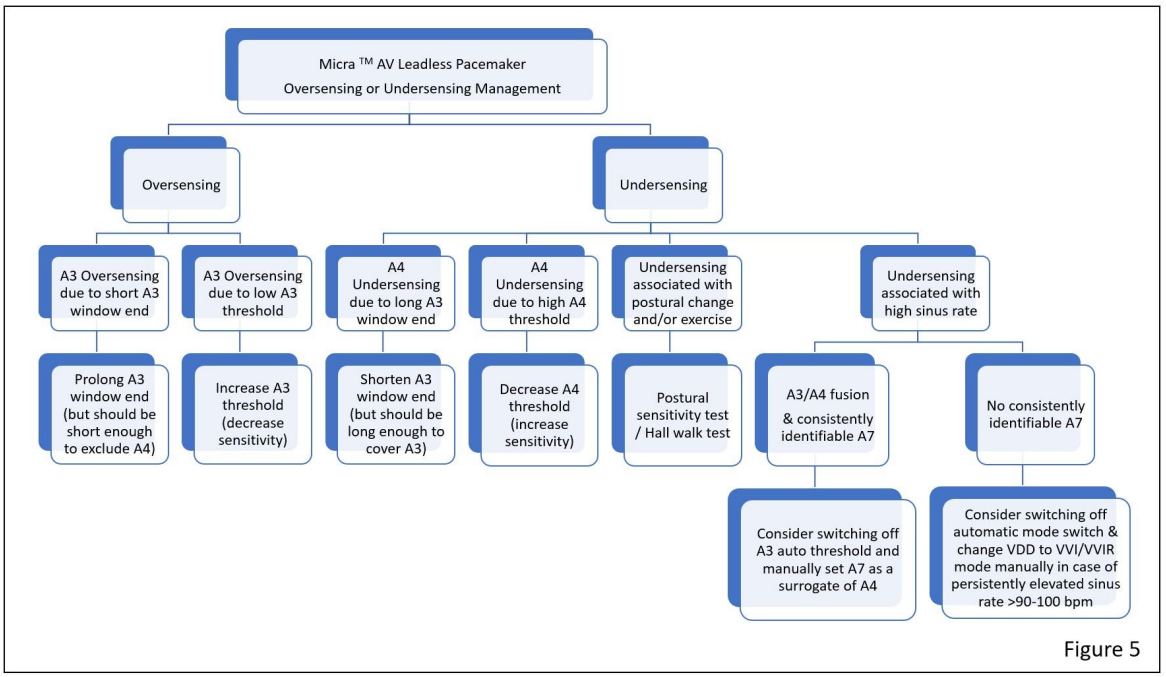


Figure 5. Proposed management algorithm for Micra™ AV LPM oversensing/ undersensing.

Conclusion

Leadless pacemaker with AV synchronous pacing function is a viable alternative to transvenous pacemaker, in reducing lead-related complications and promoting AV synchrony. However, efficient AV synchronous pacing could be limited by inappropriate detection of atrial mechanical motion during posture change, physical activities and sinus tachycardia. Manual atrial mechanical test, posture sensitivity test and a systematic, algorithmic approach to troubleshooting are imperative in optimizing the AV synchronous pacing function of leadless pacemaker system.

Acknowledgement

The author thanks Mr. Wallace Ming Kin Lam from Medtronic Hong Kong for his contribution to the pacemaker tracings.

References

1. El-Chami MF, Al-Samadi F, Clementy N, et al. Updated performance of the Micra™ transcatheter pacemaker in the real-world setting: A comparison to the investigational study and a transvenous historical control. Heart Rhythm. 2018; 15(12):1800–1807.
2. Steinwender C, Khelae SK, Garweg C, Chan JYS, Ritter P, Johansen JB, Sagi V, Epstein LM, Piccini JP, Pascual M, Mont L, Sheldon T, Splett V, Stromberg K, Wood N, Chinitz L. Atrioventricular Synchronous Pacing Using a Leadless Ventricular Pacemaker: Results From the MARVEL 2 Study. JACC Clin Electrophysiol. 2020;6(1):94-106.
3. Neugebauer F, Noti F, van Gool S, Roten L, Baldinger SH, Seiler J, Madaffari A, Servatius H, Ryser A, Tanner H, Reichlin T, Haeberlin A. Leadless atrioventricular synchronous pacing in an outpatient setting: Early lessons learned on factors affecting atrioventricular synchrony. Heart Rhythm. 2022;19(5):748-756.
4. Medtronic Micra™ AV MC1AVR1 Device Manual.
5. Medtronic Micra™ AV MC1AVR1 internal training material.

Medtronic

The leader in leadless

Introducing the next generation of Micra™ leadless pacemakers

Pioneering what's possible for bradycardia patients:

- The world's smallest pacemakers[1] now have median projected longevities of nearly 16–17 years.[2]
- Micra AV2 improves automatic AV synchrony at faster heart rates (80–100bpm),[2] and the upper tracking rate limit is now 135 bpm.

Micra AV2

Micra VR2

Establishing the rhythm.
Pioneering what's possible.

References

[1] Leick A. Micra vs Leadless and Transvenous Pacemaker Size Comparison. March 2023. Medtronic data on file.

[2] Sheldon T, Escalante K, Fagan D. Device Longevity and AV Synchrony Algorithm Modeling of a Leadless Pacemaker Family: A Virtual Patient Analysis. January 2023. Medtronic data on file.

SAVE THE DATE

APHRS 2024 SYDNEY

26 September – 29 September 2024
International Convention Centre, Sydney

26th - 29th September 2024

International Convention Centre Sydney, Australia

Over 2,500 attendees from 40 countries worldwide

ORGANISING COMMITTEE

Professor Prashanthan Sanders
The University of Adelaide & Royal Adelaide Hospital

Professor Jonathan Kalman
Royal Melbourne Hospital

Professor Peter Kistler
The Alfred Hospital & Baker IDI Heart and Diabetes Institute

Professor Rajeev Pathak
The Canberra Hospital & Canberra Heart Rhythm Centre

Doctor Melissa Mideldorp
The University of Adelaide & Royal Adelaide Hospital

Professor Christopher Semsarian
RPAH, Australian Genetic Heart Disease Registry & University of Sydney

Professor Jeroen Hendriks
Flinders University & The University of Adelaide

Associate Professor Martin Stiles
NZ Waikato Hospital & The University of Auckland

Associate Professor Han Lim
Austin Hospital, Northern Hospital & The University of Melbourne

Associate Professor Belinda Gray
The University of Sydney

Doctor Julia Isbister
The Centenary Institute at The University of Sydney

Associate Professor Andreas Pflaumer
Royal Children's Hospital Melbourne (RCH)

Eddy Kizana
Associate Professor The University

www.aphrs2024.com