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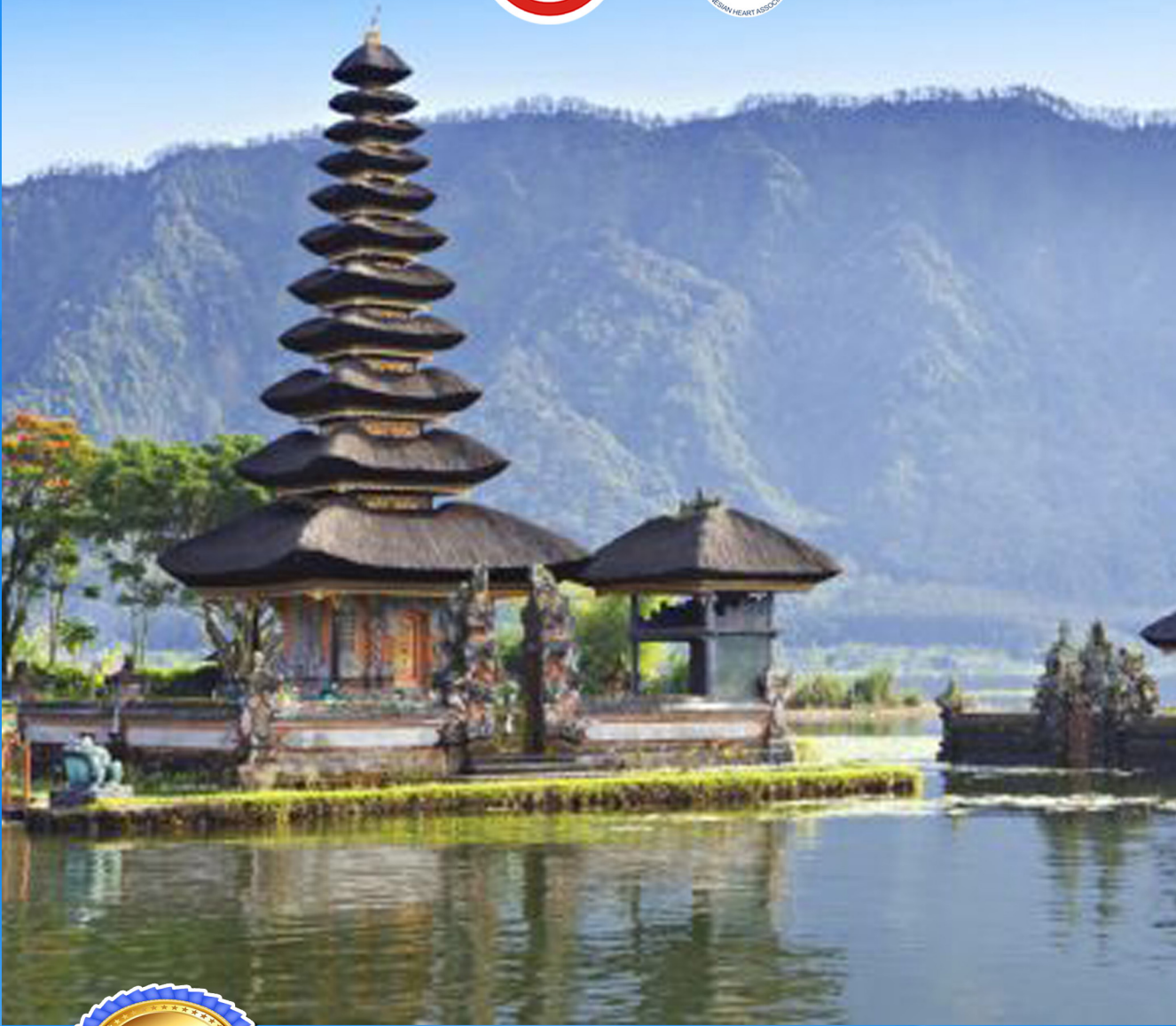


PT. Intisari Sains Medis



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Workshop on Arrhythmias

“UPGRADING SKILLS ON MANAGEMENT OF ARRHYTHMIAS IN DAILY PRACTICE”

Editor:
dr. I Made Putra Swi Antara, Sp.JP(K), FIHA

WORKSHOP ON ARRHYTHMIAS “UPGRADING SKILLS ON MANAGEMENT OF ARRHYTHMIAS IN DAILY PRACTICE”

PROGRAM BOOK

Editor:

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**Nizamuddin Ubaidillah
I Made Putra Swi Antara
Luh Oliva Saraswati Suastika
I Dewa Gde Dwi Sumajaya
AA Ayu Dwi Adelia Yasmin
Hendy Wirawan
Ni Made Ayu Wulan Sari
I Kadek Susila Surya Darma**

Penerbit :



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PT. Intisari Sains Medis

Redaksi :

Jl. Batanghari III C, No. 9

Kelurahan Panjer, Denpasar Selatan

Denpasar - Bali

Cetakan pertama : Juli 2021

2021, ix + 16 hlm, 17,5 x 24,5 cm

ISBN : 978-623-95502-4-0

Hak cipta dilindungi undang-undang

Delarang memperbanyak karya tulis ini dalam bentuk dan dengan cara apapun tanpa ijin tertulis dari penerbit

WELCOME MESSAGE

From Head of Indonesia Heart Association Branch Bali

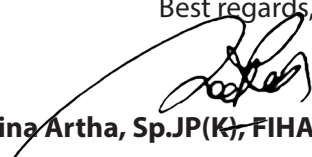
Dear Colleagues,

Cardiovascular disease is the most common cause of death worldwide including in developing countries like Indonesia. An integrative and multidisciplinary approach in the management of cardiovascular disease is necessary to obtain the expected outcomes. In the current era of National Health Insurance (JKN), general practitioners as the first liner of primary care are required to have good insight and skills to diagnose, and provide appropriate initial therapy, as well as decide when to refer patients to tertiary hospital.

In improving cardiovascular knowledge and skills for healthcare professionals, we are delighted to announce you that Indonesia Heart Association Branch Bali is going to organize our annually scientific meeting: **Workshop on Arrhythmias**, which will be held virtually from April 3 2021 until June 26 2021 through a cloud meetings. The theme that we bring in this scientific meeting is "UPGRADING SKILLS ON MANAGEMENT OF ARRHYTHMIAS IN DAILY PRACTICE" Workshop on Arrhythmias consists of one workshop. Many topics also will be presented in our workshop, include : symptomatology of arrhythmias, it's physiological effect, first line management of benign arrhythmias, and long term complication of arrhythmias.

We hope from this event will be a memorable and interesting meeting, not only for improving our knowledge but also for strengthening our relation and friendship.

Best regards,



dr. I Made Junior Rina Artha, Sp.JP(K), FIHA

GREETINGS

With every gratitude to God/Ida Sang Hyang Widhi WaSa, we proudly present our scientific meeting Workshop on Arrhythmias 2021. This year workshop which will be held virtually from April 3 2021 until June 26 2021. We have 5 series of webinars. We choose this theme of workshop to provide more information, skill and knowledge about diagnosing and treating acute cardiovascular disease. We hope this moment will guide all of us to a faster, better, and more comprehensive management in our daily practice.

We would like to thank all of the speaker for the abstract and the time to share knowledge with us in this year workshop. We also thank all of participants, sponsors, pharmaceutical industries, committee members, and all person who involve in this preparation this workshop.

We do hope this event will be a memorable and interesting meeting not only in scientific programs but also strengthening our relationship and friendship

Best Regards,
Denpasar, April 3 2021



dr. I Made Putra Swi Antara, Sp.JP(K), FIHA
Chairman of Workshop on Arrhythmias 2021

CONTENTS

WELCOME MESSAGE	iii
GREETINGS.....	iv
CONTENTS.....	v
ORGANIZING COMMITTEE	vi
WORKSHOP SCHEDULE	viii
ABSTRACT	1
SERIES 1	
Bradyarrhythmia: Physiologic or Pathologic	2
Nizamuddin Ubaidillah	
Pacemaker in Bradycardia	4
I Made Putra Swi Antara	
SERIES 2	
Malignant Arrhythmia Symptoms: Focus on Tachyarrhythmias	5
Luh Oliva Saraswati Suastika	
Sudden Cardiac Arrest Prevention	7
I Made Putra Swi Antara	
SERIES 3	
Sign and symptoms of atrial fibrillation.....	8
I Dewa Gde Dwi Sumajaya	
Stroke Prevention in Atrial Fibrillation.....	9
A.A Ayu Dwi Adelia Yasmin	
SERIES 4	
Sinus Tachycardia or Supraventricular Tachycardia: Signs and Symptoms	10
Hendy Wirawan	
Management of SVT in primary Health Care.....	12
Ni Made Ayu Wulan Sari	
SERIES 5	
PAC and PVC: Symptoms and ECG	13
Hendy Wirawan	
Non-Life-Threatening Arrhythmias: PAC and PVC, How to Treat and When to Refer?	15
I Kadek Susila Surya Darma	

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*dr. Achmad Ismail Sampurna Putra

WORKSHOP SCHEDULE

DATE	TIME	EVENT
Saturday, April 3rd, 2021		SERIES 1
	10.00-10.25	Bradyarrhythmia: Physiologic or Pathologic Dr. Nizamuddin Ubaidillah, SpJP
	10.25-10.50	Pacemaker in Bradycardia Dr. I Made Putra Swi Antara, SpJP (K)
	10.50-11.15	Q&A
Saturday, April 17th, 2021		SERIES 2
	10.00-10.25	Malignant Arrhythmia Symptoms: Focus on Tachyarrhythmias Dr. Luh Oliva Saraswati Suastika, SpJP (K)
	10.25-10.50	Sudden Cardiac Arrest Prevention Dr. I Made Putra Swi Antara, SpJP (K)
	10.50-11.15	Q&A
Saturday, May 8th, 2021		SERIES 3
	10.00-10.25	Sign and symptoms of atrial fibrillation Dr. I Dewa Gde Dwi Sumajaya, SpJP
	10.25-10.50	Stroke Prevention in Atrial Fibrillation Dr. A.A Ayu Dwi Adelia Yasmin, SpJP
	10.50-11.15	Q&A

DATE	TIME	EVENT
Saturday, June 5th, 2021		SERIES 4
	10.00-10.25	Sinus Tachycardia or Supraventricular Tachycardia: Signs and Symptoms Dr. Hendy Wirawan, SpJP
	10.25-10.50	Management of SVT in primary Health Care Dr. Ni Made Ayu Wulan Sari, SpJP (K)
	10.50-11.15	Q&A
Saturday, June 26th, 2021		SERIES 5
	10.00-10.25	PAC and PVC: Symptoms and ECG Dr. Hendy Wirawan, SpJP
	10.25-10.50	Non-Life-Threatening Arrhythmias: PAC and PVC, How to Treat and When to Refer? Dr. I Kadek Susila Surya Darma, SpJP
	10.50-11.15	Q&A

ABSTRACT

Bradyarrhythmia: Physiologic or Pathologic

Nizamuddin Ubaidillah

Head of Cardiology Department Udayana Army Hospital
Lecturer Faculty of Medicine Jenderal Ahmad Yani University

Bradyarrhythmia is defined by a heart rate below 60 beats per minute (bpm) but in a recent guideline from HRS/Heart Rhythm Society 2019; it is defined below 50 bpm. Common clinical findings are range include physiologic reaction (for example in healthy athletic persons) until pathologic condition. The clinical presentation ranges too from asymptomatic until symptomatic. The US CDC reported in 2011 15.2% of adult males and 6.9% of adult females had clinically-defined bradycardia (a resting heart rate below 60 bpm). Bradycardia and conduction abnormalities are seen more commonly identified in elderly people. Prevalence of Sick Sinus Syndrome is 0.03% in the whole population, and increases with increasing age and prevalence of atrioventricular block (based on clinical studies) are range from 0.015% to 0.1%. Mortality for untreated patients with sick sinus syndromes ranges from 5 to 10% at 1 year to 25 to 30% at 5 years. Research from ARIC (Atherosclerosis Risk in Communities) and CHS (Cardiovascular Health Study) revealed SND is the commonest founding in individuals at 7 and 8 decades. In Asia, Thailand studies recalled SND account for 2.7% and AV conduction disturbance for 1.13%. Indonesia studies in PHC (Public Health Centre/Puskesmas in South Sulawesi revealed the prevalence of bradyarrhythmia in 8.3%.

Physiologic Condition

There are 3 types of physiologic conditions as the cause of bradyarrhythmia: heart rate variation, sleep, and athletes. Heart rate variation depends on resting heart rates; it varies among normal individuals and upon age and level of conditioning. Resting heart rates can range from 46 to 93 beats per minute in males and 51 to 95 beats per minute in females. In adults from 20 to 90 years

of age, has the second-lowest percentile for heart rate ranged from 40 to 55 bpm depending on gender and age. In Sleep, heart rate drops below 50 bpm during sleep as a result of enhanced nocturnal parasympathetic tone which accounts for approximately 25% of men and 10% of women. During sleep, heart rates may decline by 25 beats per minute in young patients, and 15 beats per minute in the elderly. In Athletes, heart rate had a lower resting heart rate than others.

Pathologic Condition

Symptoms related to pathologic condition in bradyarrhythmias could be diverse and related to decreased cardiac output and resulting in hypoperfusion of vital organs; include specific either or non-specific and chronic such as dyspnea on exertion, exercise intolerance, fatigue, and weakness. Symptoms may be severe including syncope, lightheadedness or dizziness, palpitation, angina, or a change of mental status. The risk factors to be considered are past medical history, drug abuse, alcohol abuse, drug intoxication, and family history of SCD. The reversible causes of bradyarrhythmias include myocardial infarction, athletic training, drug intoxication, toxin overdoses, electrolyte imbalance, hypothyroid, hypothermia, and hypercarbia. The main site for disturbances is in the sinus node (SA node) and atrioventricular node (AV node). Sinus node dysfunction with accompanying symptoms include sinus bradycardia < 50 bpm, SA/Sinoatrial exit block, Sinus Pause, chronotropic incompetence; and Brady/Tachy Syndrome. The other site in the AV node includes AV Block First until Third degree.

Pacemaker in Bradycardia

I Made Putra Swi Antara

The rate of pacemaker insertion continues to increase worldwide, along with the increasing life expectancy and age of the population in developing countries. The largest proportion of pacemakers are indicated for bradycardia. Under normal conditions, the cardiac conduction system has several rhythm control points or pacemakers, starting from the sinus node, which has a basic rhythm of 60-80 beats per minute, to the Purkinje system at a rate of 20-40 beats per minute. Heart rate has a direct relationship to cardiac output in the calculation of cardiac output, so that if a person experiences bradycardia, he will experience a decrease in heart performance which is often accompanied by complaints of shortness of breath or fatigue.

The incidence of bradycardia has the greatest proportion and risk in the geriatric/elderly population. There are many etiologic causes of bradycardia, with the most common being an idiopathic degenerative process. It should be kept in mind that bradycardia can occur physiologically in professional athletes, without negatively affecting their general health. A significant risk in the elderly population is the occurrence of pre/syncope which can lead to fall trauma and bone fractures, while in the younger population it is fatigue and decreased quality of life.

Establishing the diagnosis of symptomatic bradycardia requires a correlation between the existing rhythm and the patient's symptoms. This connection can be made through long-term ECG recordings such as a holter monitor or an implantable loop recorder. Most bradycardias are benign and not life-threatening because the heart's conduction system has a different basic rhythm. In the event of AV block of degree 3 or degree 2 type 2, insertion of a pacemaker remains an indication regardless of symptomatic or not. In the rest of the population, establishing the correlation between rhythm and symptoms will determine the decision for a permanent pacemaker as the definitive treatment for bradycardia.

Malignant Arrhythmia Symptoms: Focus on Tachyarrhythmias

Luh Oliva Saraswati Suastika

Malignant arrhythmias are life threatening arrhythmias (primarily ventricular) that result in cardiovascular collapse. These include bradyarrhythmias (complete heart block, sick sinus syndrome, idioventricular rhythm) and tachyarrhythmias (ventricular tachycardia (VT) and fibrillation (VF), Wolff-Parkinson-White (WPW) syndrome, Brugada syndrome and long QT syndrome). Tachycardia of atrial or ventricular origin reduces stroke volume and cardiac output particularly when the ventricular rate is greater than 160 beats/min. The stroke volume becomes reduced because of decreased ventricular filling time and decreased preload at high rates of contraction. Furthermore, if the tachyarrhythmia is associated with abnormal ventricular conduction, the synchrony and thus the effectiveness of ventricular contraction will be impaired leading to reduced ventricular ejection. Another consequence of tachycardia is increased myocardial oxygen demand that can cause angina, especially in patients with underlying coronary artery disease. Finally, chronic states of tachycardia can lead to systolic heart failure. In managing malignant arrhythmias, we should recognize the clinical consequences (hemodynamic instability), interpret the ECG rhythm and find the underlying disease/cause of arrhythmia. Clinical symptoms of malignant arrhythmias may vary depend on the ventricular rate, duration of tachycardia, and presence and extent of the underlying heart disease and peripheral vascular disease. Symptoms include palpitations, dizziness or light-headed, shortness of breath, chest pain, syncope/pre-syncope, unconsciousness, seizure, and even cardiac arrest or sudden cardiac death. Hemodynamically stable events usually resulted in milder symptoms, this may happen with atrial fibrillation (AF), VT (non sustained or sustained episodes), supraventricular tachycardia, and AV node reentrant tachycardia (AVRT) in WPW syndrome. More severe symptoms may result from unstable events of those arrhythmias, while pulseless VT and VF result

in faintness, followed by loss of consciousness, seizures, apnea, and may lead to cardiac arrest and sudden death. Malignant arrhythmias caused by cardiac channelopathies (Brugada syndrome, long QT syndrome, arrhythmogenic right ventricular cardiomyopathy, catecholaminergic polymorphic ventricular tachycardia) increase the chance of VT or VF to occur spontaneously, leading to sudden cardiac arrest. It is very essential to recognize the malignant arrhythmia from ECG and whether the symptoms are hemodynamically stable or unstable.

Sudden Cardiac Arrest Prevention

I Made Putra Swi Antara

Sudden cardiac arrest (SCA) is a clinical syndrome in which a detectable pulse loss or sudden cessation of breathing occurs. This process generally has a natural course to death in less than an hour. There are various etiologies that cause SCA, which often overlap. These causes can be grouped into structural abnormalities of the heart, electrical abnormalities of the heart, and acquired heart defects.

The risk of SCA events increases significantly with age, where age >75 years has an incidence rate of about 2-5 events per 1000 people in the general population. However, the highest incidence actually occurs in groups of high-risk patients, such as heart failure with weak heart function and post-myocardial infarction. CHD is the most common cause of SCA with an incidence proportion of up to 75%, followed by cardiomyopathy as the second most common cause at 15%.

SCA prevention can be done primary or secondary. Primary prevention includes screening and prevention of CHD risk factors in the general population, as well as standard therapy and implantation of ICD or CRT-D in some patients who meet the criteria for the CHD and Heart Failure population. While secondary prevention is given to the population of people who have experienced cardiac arrest requiring ICD installation if the SCA incident is not caused by reversible causes and has a life expectancy of more than one year. It is important to remember that the risk of SCA is not proportional to the severity of the complaint, especially in the heart failure population. The incidence of SCA actually has the highest incidence in the population with NYHA class II compared to class III or IV.

The high effectiveness of ICD in preventing SCA for high-risk populations emphasizes the importance of risk stratification to always be carried out in patients with CHD or heart failure who meet the criteria for ICD installation.

Sign and symptoms of atrial fibrillation

I Dewa Gde Dwi Sumajaya

Atrial fibrillation is one of the most common types of arrhythmias, which are irregular heart rhythms. Atrial fibrillation would induce the heart to beat much faster than normal. It is characterized with heart's upper and lower chambers which not work together (dyssynchrony). When this happens, the lower chambers do not fill completely or pump enough blood to systemic circulation. This could make symptom like feel tired or dizzy, or heart palpitations or chest pain. Blood also pools inside the heart, which increases risk of forming clots and can leads to strokes or other complications.

Atrial fibrillation can also occur without any signs or symptoms. Untreated fibrillation can lead to serious and even life-threatening complications. Sometimes atrial fibrillation goes away on its own. For some people, atrial fibrillation is an ongoing heart problem that lasts for years. Over time, it may happen more often and last longer. Treatment restores normal heart rhythms, helps control symptoms, and prevents complications. Mostly we need to control heart rate to reduce symptoms and complication by ongoing tachycardia. What utmost important is to screen whether patient at risk of developing atrial fibrillation.

Stroke Prevention in Atrial Fibrillation

AA Ayu Dwi Adelia Yasmin

Atrial fibrillation is the most common cardiac rhythm disorder worldwide, and most frequently encountered both in outpatient and emergency settings. Atrial fibrillation may lead to a prothrombotic state, which predisposes the patients to stroke, the most devastating and most common complication of thromboembolism. Stroke is the most common complication of atrial fibrillation, and associated with an increased morbidity and mortality. Data from various stroke registries show that both unknown and untreated or undertreated atrial fibrillation has an important role for most of the ischemic stroke occurrences, which are often fatal and debilitating. Although many improvements have been made in both primary and secondary stroke prevention in patients with atrial fibrillation, the long-term risks for stroke recurrence and bleeding complications arose from antithrombotic treatment remain substantial. Stroke prevention is one of the cornerstones of management in atrial fibrillation. Stroke risk assessment and determination of appropriate thromboprophylaxis is emphasized in guidelines, as well as bleeding risk assessment. Guidelines also emphasize the importance of reassessment of stroke risk at certain periodic intervals and address potentially modifiable bleeding risk factors for improving comprehensive management in patients with atrial fibrillation. Patients frequently need support from physicians and various other sources to start thromboprophylaxis treatment, and to ensure compliance to and persistence with treatment in the long term.

Sinus Tachycardia or Supraventricular Tachycardia: Signs and Symptoms

Hendy Wirawan

Tachyarrhythmia was defined as heart rate greater than 100 beats per minutes. It was occurred by various mechanisms, such as abnormal automaticity, triggered activity, or re-entry. Tachyarrhythmia was divided into two groups, narrow QRS complex tachycardia and wide QRS complex tachycardia. Both of these groups were also divided into 2 groups, irregular and regular tachycardia. Narrow QRS complex tachycardia was defined as heart rate greater than 100 beats per minute and QRS complex less than 120 ms, while wide QRS complex tachycardia has QRS complex more than 120 ms.

It is important to distinguish between sinus tachycardia and supraventricular tachycardia. Both of these tachycardia are regular narrow QRS complex tachycardia. Both sinus tachycardia and supraventricular tachycardia has different signs and symptom. The 12-lead electrocardiogram is helpful in distinguish which one of regular narrow QRS complex that was occurred.

Sinus tachycardia has gradual accelerating and decelerating of heart rate. It can be occurred from physiologic and pathologic mechanisms. Underlying condition, physiologic and pathologic, and pharmacological effect can cause physiologic sinus tachycardia. On the other hand, such conditions can lead to pathological sinus tachycardia, like sinoatrial re-entry, postural orthostatic tachycardia syndrome (POTS), and inappropriate sinus tachycardia.

Supraventricular tachycardia has various forms, such as atrial tachycardia, atrial flutter with fix atrioventricular conduction, and supraventricular tachycardia with re-entry (AV nodal re-entry tachycardia (AVNRT) and AV re-entry tachycardia (AVRT)). Both of these forms also have specific electrocardiogram morphology and mechanism.

Sinus tachycardia has different onset, symptoms, underlying condition, and electrocardiogram morphology compared to supraventricular tachycardia. The knowledge about it, is helpful to make the diagnosis.

Management of SVT in primary Health Care

Ni Made Ayu Wulan Sari

The term 'SVT' literally indicates tachycardia [atrial rates >100 beats per minute (b.p.m.) at rest], the mechanism of which involves tissue from the His bundle or above. In the general population, the SVT prevalence is 2.25/1000 persons and the incidence is 35/100 000 person-years. Women have a risk of developing SVT that is two times greater than that of men. Persons aged >_65 years or have more than five times the risk of developing SVT than younger individuals.

Clinical presentation are palpitations (22%), Fatigue, Light-headedness, Chest discomfort, Dyspnoea, Altered consciousness, In older patients, symptoms may be more extreme—with dizziness, presyncope, and syncope.

Initial evaluation of the patient with supraventricular tachycardia divided into standard evaluation and optional evaluation.

The initial approach to acute management tends to be non-drug-based, with escalation to intravenous (i.v.) drugs or electrical cardioversion in the absence of early correction. Immediate direct-current (DC) cardioversion is the first choice in haemodynamically compromised patients with narrow QRS tachycardia. Vagal manoeuvres can be used to terminate an episode of narrow QRS SVT. Vagal manoeuvres include the Valsalva manoeuvre, carotid sinus massage, facial immersion in cold water or forceful coughing. Adenosine, an endogenous purine nucleoside (6-18 mg i.v. bolus) is the first drug of choice. Calcium channel blockers (verapamil/diltiazem i.v.) and beta blockers (e.g. esmolol and metoprolol i.v.) are of value, particularly in patients with frequent atrial or ventricular premature beats.

Long-term management is contingent on underlying mechanism, frequency of symptoms, patient safety and preference. Catheter ablation a particularly desirable option as first line therapy for all SVTs for improvements in quality of life, reduced hospital attendances and cost burden.

PAC and PVC: Symptoms and ECG

Hendy Wirawan

Department of Cardiology and Vascular Medicine,
Sanglah General Hospital Denpasar

Arrhythmia is abnormalities of the electric rhythm, result from alteration of impulse formation, impulse conduction, or both. Premature atrial contractions (PAC) and premature ventricular contractions (PVC) are among the most common forms of arrhythmias. Both conditions are caused by premature electrical activation in heart. This results in premature heart muscle contractions, which trigger extra heartbeats which disrupt heart's normal rhythm.

The incidence of PVCs and PACs increase with age and common in healthy individual. In the general population may be as high as 80% in healthy young adults.

Management of PVCs and PACs include discernment of associated heart disease and other predisposing conditions prior to the decision to specifically treat the PVCs and PACs. The decision of whether to treat PVCs and PACs depend on symptoms and structural heart diseases. In the absence of symptoms and structural heart disease, no treatment is needed, and reassurance is recommended.

Typically, only patients with symptomatic PVCs and PACs require treatment. After appropriate identification of triggers or underlying structural cardiac conditions, therapy starts by reassuring patients that PVCs and PACs are typically benign and can be controlled by avoiding triggers. In patients with persistent symptoms despite avoidance of triggers, further therapy is indicated, starting with medications.

Attempts should always be made to control with conservative non-pharmaceutical measures first. Primary healthcare providers may provide initial reassurance and medical treatment; pharmacologic management can be achieved using beta-adrenergic blockers at low doses are the relatively safe and first-line treatment in symptomatic patients if conservative measures fail. Whereas, in advance condition of the premature ventricular contraction, additional antiarrhythmic drugs may be needed such as flecainide, propafenone, sotalol, and amiodarone. However, refractory cases or use of antiarrhythmic medications consider for ablation.

Non-Life-Threatening Arrhythmias: PAC and PVC, How to Treat and When to Refer?

I Kadek Susila Surya Darma

Premature atrial contractions (PAC) and premature ventricular contractions (PVC) are among the most common forms of arrhythmias. Both conditions are caused by premature electrical activation in heart. This results in premature heart muscle contractions, which trigger extra heartbeats. These extra heartbeats disrupt heart's normal rhythm.

The incidence of PVCs and PACs increase with age and common in healthy individual. In the general population may be as high as 80% in healthy young adults. PVCs are more common in men, in African-Americans, and in individuals with underlying heart disease, hypertension, hypokalemia or hypomagnesaemia.

PVC and PAC are frequently encountered in the primary care setting. The clinician is often faced with the dilemma of finding PVCs and PACs in a patient and deciding whether the PVCs and PACs should be treated. The decision of whether to treat PVCs and PACs depend on symptoms and structural heart diseases. In the absence of symptoms and structural heart disease, no treatment is needed, and reassurance is recommended.

Typically, only patients with symptomatic PVCs and PACs require treatment. After appropriate identification of triggers or underlying structural cardiac conditions, therapy starts by reassuring patients that PVCs and PACs are typically benign and can be controlled by avoiding triggers. In patients with persistent symptoms despite avoidance of triggers, further therapy is indicated, starting with medications.

Attempts should always be made to control with conservative non-

pharmaceutical measures first. Primary healthcare providers may provide initial reassurance and medical treatment; pharmacologic management can be achieved using beta-adrenergic blockers as first-line treatment in symptomatic patients if conservative measures fail. However, refractory cases or use of antiarrhythmic medications should be referred to cardiologists.

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ISBN 978-623-95502-4-0



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