



**DEPARTMENT OF CARDIOLOGY AND VASCULAR MEDICINE  
MEDICAL FACULTY OF ANDALAS UNIVERSITY**

# **Certificate of Attendance**

This is to certify that

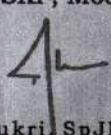
**Dr. Eka Fithra Elfi, Sp.JP**  
**as Speaker**

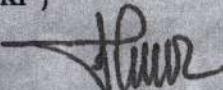
**Symposium Strengthening the Role of Physicians  
in Cardiovascular Care**

Padang, May 13-14<sup>th</sup> 2017

SKP IDI SUMATERA BARAT: No 608/IDI-WIL-SB/SK/ XII/2016  
( Participant 12 SKP, Speaker 8 SKP, Moderator 6 SKP, Committee 1 SKP )

  
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Chairman of Organizing Committee

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4. Yuniadi Y, Tondan AE, Hanafy DA. Pedoman Tatalaksana Fibrilasi Atrium. Perhimpunan Dokster Spesialis Kardiovaskuler Indonesia, 2014.
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## Aritmia Supraventrikular dan Ventrikular

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Pasien dengan gangguan irama jantung dapat terjadi dengan berbagai keluhan, namun gejala seperti palpitas, sinkop, presyncope, atau gagal jantung kongestif umumnya menyebabkan mereka mencari pengobatan. Dalam menilai pasien dengan aritmia yang telah diketahui atau diduga, beberapa informasi penting harus diperoleh yang dapat membantu menentukan diagnosis atau panduan uji diagnostik lebih lanjut. EKG adalah alat utama dalam analisis aritmia. Mekanisme aritmogenesi yaitu gangguan formasi impuls, gangguan konduksi impuls, dan kombinasi (tabel 9.1).<sup>1</sup>

**Tabel 9.1 Mekanisme Aritmogenesis**

Sumber: Lilly, Leonard S. 2007.

Disorders of Impulse Conduction		TABLE 35-4 – Mechanisms of Arrhythmogenesis	
Block	DISORDER	EXPERIMENTAL EXAMPLES	CLINICAL EXAMPLES
Bidirectional reentry	Disorders of Impulse Formation		
Unidirectional reentry	Automaticity		
	Normal automaticity	Normal <i>in vivo</i> or <i>in vitro</i> in sinoatrial node, AV nodal, and Purkinje cells	Sinus tachycardia or bradycardia inappropriate for clinical situation; possibly ventricular parasystole
Reentry	Abnormal automaticity	Depolarization-induced automaticity in Purkinje myocytes	Possibly accelerated ventricular rhythms after myocardial infarction
Competition	Triggered activity		
Interactions between automaticity and reentry	Early afterdepolarizations	Drugs ( <i>sotalol, N-acetylprocainamide, terfenadine, erythromycin</i> ), cesium, barium, low $[K^+]$	Acquired long-QT syndrome and associated ventricular arrhythmias
Interactions between automaticity and triggered activity	Delayed afterdepolarizations	Gain-of-function mutations in the gene encoding RyR2	Catecholaminergic polymorphic ventricular tachycardia

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**Tabel 9.2 Aritmia yang Umum Terjadi.**

Sumber: Lilly, Leonard S. 2007.

**Table 12-1.**

Location	Bradyarrhythmias	Tachyarrhythmias
SA node	Sinus bradycardia Sick sinus syndrome	Sinus tachycardia
Atria		Atrial premature beats Atrial flutter Atrial fibrillation Paroxysmal supraventricular tachycardias Focal atrial tachycardia Multifocal atrial tachycardia
AV node	Conduction blocks Junctional escape rhythm	Paroxysmal reentrant tachycardias (AV or AV nodal)
Ventricles	Ventricular escape rhythm	Ventricular premature beats Ventricular tachycardia Torsades de pointes Ventricular fibrillation

AV, atrioventricular; SA, sinoatrial.

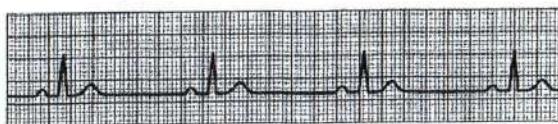
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## **Bradikardia<sup>1,2</sup>**

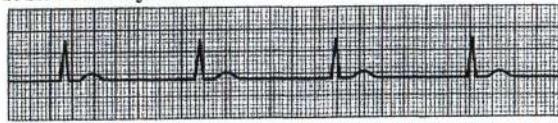
Rangsangan denyut jantung normal, akibat depolarisasi sinus nodul berulang, berkisar antara 60 sampai 100 kali/menit. Bradikardia adalah ritme dimana denyut jantungnya 60 kali/menit. Hal ini timbul dari gangguan formasi impuls atau gangguan konduksi impuls.

### **Sinoatrial Node**

#### **a. Sinus Bradycardia**

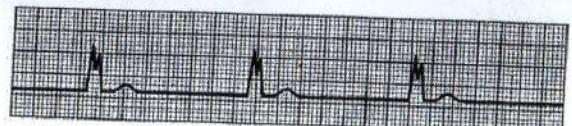


#### **b. Sick Sinus Syndrome**



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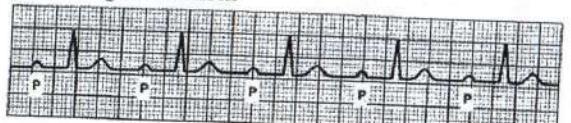
## **c. Escape Rhythms**



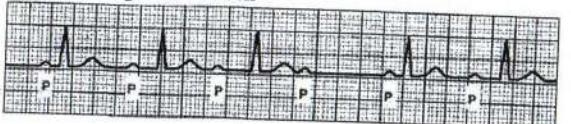
### **Atrioventricular Conduction System**

Sistem konduksi AV mencakup nodus AV, His bundle, dan kir dan kanan *bundle branches*. Gangguan konduksi antara atrium dan ventrikel bisa terjadi pada konduksi blok AV tipe tiga.<sup>1</sup>

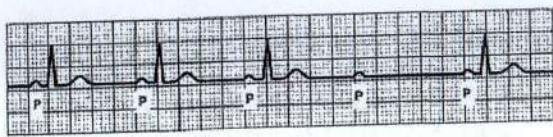
#### **a. First-Degree AV Block**



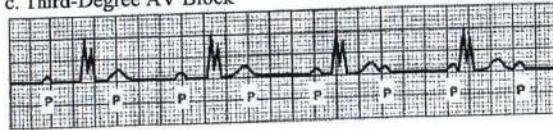
#### **b. Second-Degree AV Block**



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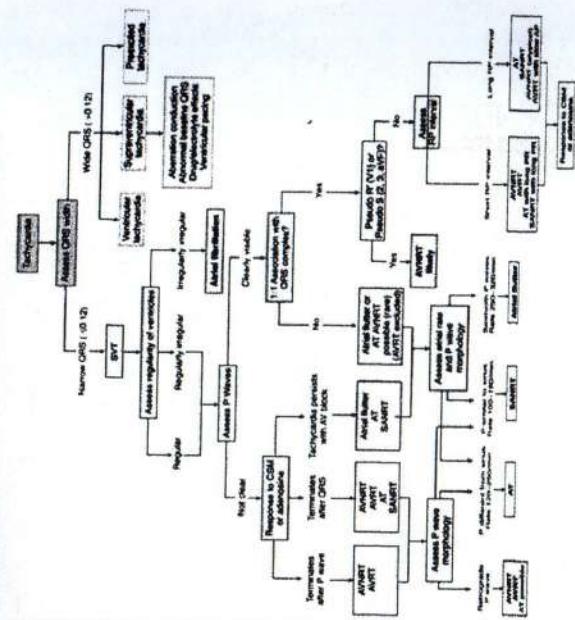


c. Third-Degree AV Block



#### Takiaritmia

Bila denyut jantung 100 bpm untuk tiga beat atau lebih, takiaritmia terjadi. Takiaritmia dihasilkan dari salah satu dari tiga mekanisme: peningkatan otomatitas, *re-entry*, atau aktivitas yang dipicu. Takiaritmia dikategorikan ke dalam yang itu timbul di atas ventrikel (supraventrikular) dan yang timbul di dalam ventrikel. Pendekatan diagnosis takiaritmia dapat dilihat pada gambar 9.3 di bawah ini.<sup>1,2</sup>

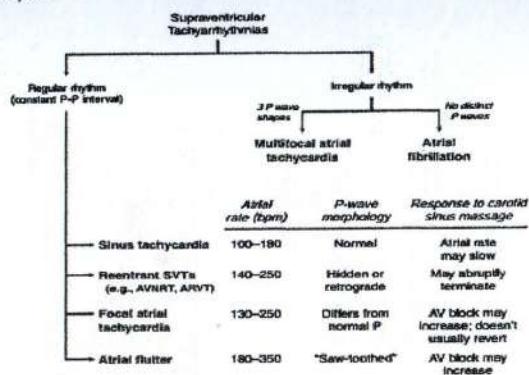


Gambar 9.3 Langkah Pendekatan Diagnosis Tipe Takikardia.  
Sumber: Lilly, Leonard S. 2007.

#### Supraventricular Arrhythmias<sup>1,2</sup>

Gambar 9.6 di bawah ini menunjukkan skema untuk

membantu menyusun takiaritmia supraventrikular yang umum terjadi.



**Gambar 9.6 Perbedaan Takiaritmia Supraventrikular Yang Umum Terjadi**

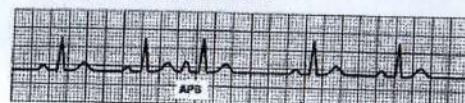
Sumber: Lilly, Leonard S. 2007.

#### a. Sinus Tachycardia

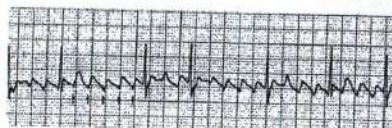


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#### b. Atrial Premature Beats



#### c. Atrial Flutter



#### d. Atrial Fibrillation



#### e. Paroxysmal Supraventricular Tachycardias

f. AV Nodal Reentrant Tachycardia

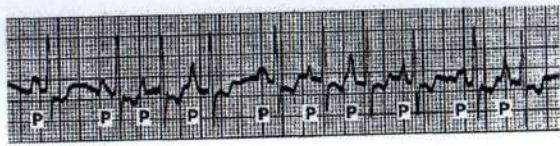
g. AV Nodal Reentrant Tachycardia

h. Concealed Accessory Pathways

i. Focal Atrial Tachycardia

j. Multifocal Atrial Tachycardia

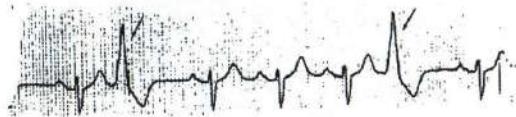
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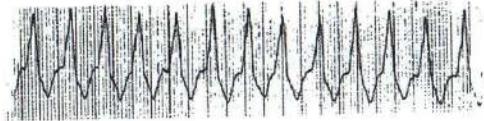
#### Aritmia Ventrikular<sup>1,2,3</sup>

Aritmia ventrikular yang umum adalah ventricular premature beats (VPBs), (2) ventricular tachycardia (VT), and (3) ventricular fibrillation (VF). Aritmia ventrikular biasanya lebih berbahaya dibanding supraventrikular. Gangguan yang bertanggung jawab atas sekitar 300.000 kematian jantung mendadak setiap tahun di Amerika Serikat.<sup>1</sup>

##### Ventricular Premature Beats

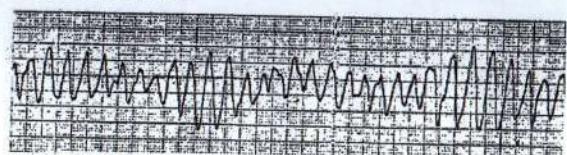


##### Ventricular Tachycardia

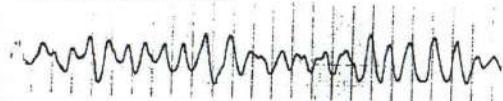


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#### Torsades de Pointes



#### Ventricular Fibrillation



#### Obat Antiaritmia<sup>2</sup>

Tabel di bawah menjelaskan klasifikasi obat antiaritmia, mekanisme dan contoh obat.

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Tabel 9.3 Klasifikasi Obat Antiaritmia

Tabel 9.3 Klasifikasi Obat Antiaritmia		
Class	General Mechanism	Example
I	Na <sup>+</sup> channel blockade Moderate block (↓ phase 0 upstroke rate; prolonged AP duration)	Dquinidine Flecainide Disopyramide Lidocaine Mexiletine
	Mild block (↓ phase 0 upstroke rate; shortened AP duration)	Flecainide Propafenone Propanolol Esmolol Metoprolol
II	K <sup>+</sup> channel blockade (↓ phase 0 upstroke rate; no change in AP duration)	β-Adrenergic receptor blockade
	Ca <sup>++</sup> channel antagonists	Amlodipine Diltiazem Sotalol Thiazide Dofetilide Verapamil
IV	AP, action potential.	

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#### Kepustakaan

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