

Tachyarrhythmi as

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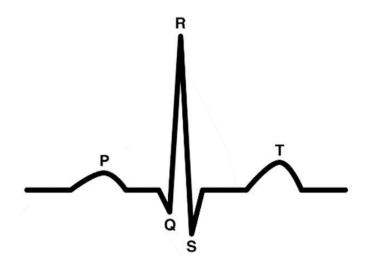
Learning objectives

- Learn the calculation of heart rate via electrocardiogram
- Distinguish between regular and irregular rhythms
- Recognize different types of tachyarrhythmias on electrocardiogram
- Understand the mechanisms of tachyarrhythmias and correlation with EKG changes.
- Learn the general principles of tachyarrhythmias treatment

Interpretation of ECG

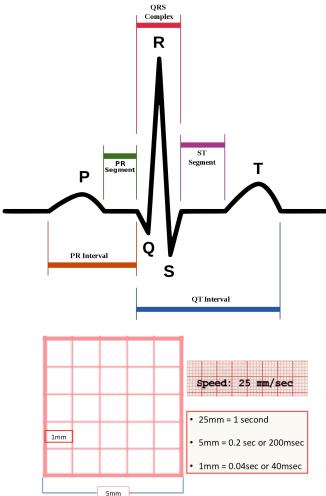
ECG waves:

- P wave: Atrial depolarization
- PR interval: time for impulse to travel from SA node to myocardium
- QRS: Ventricular depolarization
- ST segment: isoelectric period before repolarization
- T wave: Ventricular repolarization



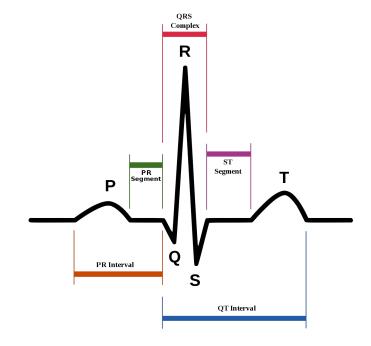
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ECG segment:	Duration:	Squares number:
Р	< 80 milliseconds	2 small
PR*	< 200 millisecond	5 small (1 large)
QRS	< 120 millisecond	3 small

^{*}PR is measured from the beginning of p wave till the R peak

ECG principles:

Wave correlation:

- P wave = atrial depolarization
- QRS = ventricular depolarization
- T = ventricular repolarization

Wave width (duration):

- absent wave = no conduction
- Long duration = slow conduction (via muscles)
- Short duration = fast conduction (via nerves)

Wave height: muscle hypertrophy

Purkinje fibers: anatomy and physiology:

SV node

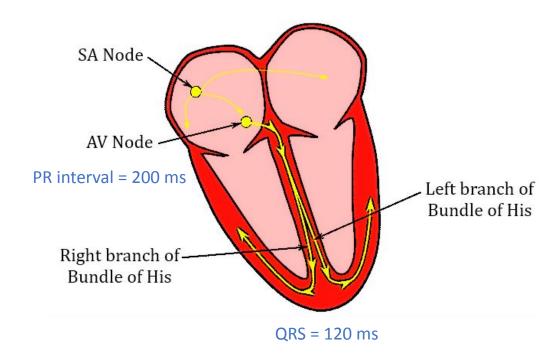
Automatic pacing

AV node

- Delay between contractions of atria and ventricles
- to allow for ventricular filling)

Buddle of his and branches

Fast depolarization of the ventricles



!! Transmission via nerves is faster than transmission though muscles

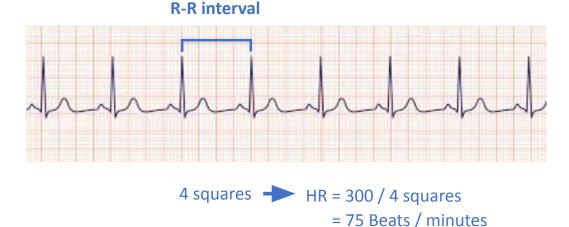
Approach to ECG:

- 1. Patient profile (name, date of birth)
- 2. ECG quality (calibration, speed)
- 3. Rate?
- 4. Rhythm?
- 5. PQRST interpretation
 - P wave
 - PR interval
 - QRS segment
 - ST segment
 - T wave

Heart Rate:

Heart rate (regular) = 300 / number of big squares between R raves

Normal Heart rate:
 60 - 100 Beats/minute



Heart rate (irregular) = Number of R waves over a 10 second period \times 6

Heart Rate:

Heart rate (irregular) = Number of R waves over a 10 second period \times 6



Beats = 14 beasts in 10 seconds Heart rate = 14 X 6 = 84 beats/min

Heart rate interpretation:

Slow <60 beat / min	Fast > 100 beats /min
Bradycardia	Tachycardia
Heart blocks	Arrhythmias
Types of bradycardia	Types of tachyarrhythmias
Sinus bradycardia 50 bpm	Sinus tachycardia usually < 130
Complete heart block 30 bpm	Atrial fibrillation Variable ventricle response
	Atrial flutter = 150 bpm
	Supraventricular tachycardia > 150 bpm
	Ventricular tachycardia Variable

Heart rhythm:

1. P-wave? (lead II , III, aVF)

2. Regularity? *(QRS)*

3. QRS rate?

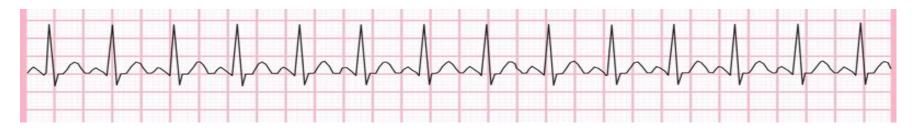
4. QRS narrow or wide?

Atrial fibrillation		
Absent p waves		
Irregular ORS		

Narrow QRS		Wide QRS	
Regular	Irregular	Regular	Irregular
- Sinus tachycardia - SVT	Atrial fibrillation (AF)	SVT with aberrancyVentricular tachycardia(monomorphic)	- AF with aberrancy - Ventricular tachycardia (polymorphic)
- Junctional tachycardia	- MAT	- Antidromic AVRT	

SVT = Supraventricular tachyarrhythmias, AF = Atrial fibrillation, VT = ventricular tachycardia, MAT = multifocal atrial tachycardia AAVRT = Atrioventricular reentrant tachycardia

Sinus tachycardia



- Sinus rate >100bpm but wave are generated from SA node
- Sinus tachycardia is a physiological response to a stimulus
- Treatment is aimed at correction of the underlying cause.
 Avoid Beta blockers except in hyperthyroidism.

Cause of tachycardia		
Exercise & excitement		
pain		
anemia		
hypovolemia		
thyrotoxicosis		
acute pulmonary embolism		
drugs		
heart failure		

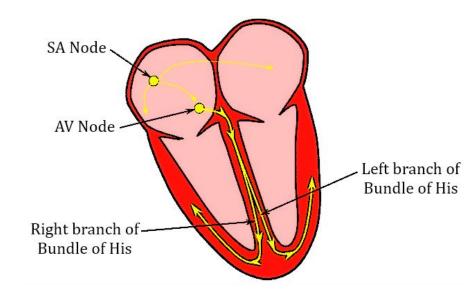
Mechanisms of wide QRS:

1. Ventricular arrhythmias

Fast depolarization of the ventricles

2. Atrial arrhythmias

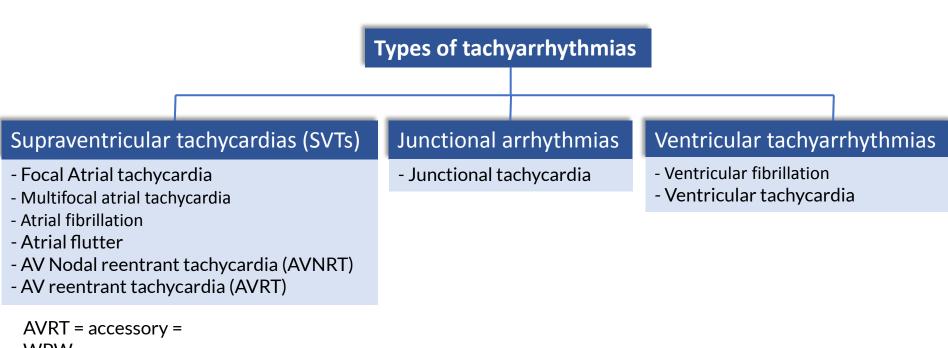
- Aberrancy or Aberrant conduction
 Conduction through the atrioventricular node with delay or block (LBBB or RBBB)
- Accessory pathway
 conduction through an accessory pathway
 (a position away from Purkinje fibers)



!! Transmission via nerves is faster than transmission though muscles

Tachyarrhythmias

Definition: An abnormal heart rhythms with a ventricular rate of 100 bpm or more



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Mechanism of Tachyarrhythmias

Increased automaticity.

repeated spontaneous depolarization of an ectopic focus, often in response to catecholamines

Re-entry

The tachycardia is initiated by an ectopic beat and sustained by a re-entry circuit.

Triggered activity

It is a form of secondary depolarization arising from an incompletely repolarized cell membrane (acute coronary syndrome).

Signs and symptoms:

Symptoms:	Signs:	Mechanism:
dizziness or lightheadedness Fainting decreased level of consciousness	Hypotension	Cerebral hypoperfusion
Palpitations or fluttering	Tachycardia	Feeling heart beats
Shortness of breath	Pulmonary edema	Impaired heart contractility
Chest pain	Active pain on exam	cardia ischemia

Arrhythmias: Diagnostic approach

Hemodynamic assessment

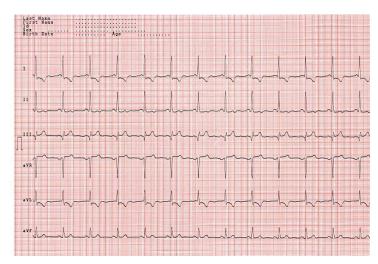
Blood pressure, heart rate, mental confusion

- Stable patient

 12 lead Electrocardiogram (ECG)
- Unstable nationt

 Cardiac monitor





Supraventricular tachyarrhythmias (SVTs)

- Focal atrial tachycardia
- Multifocal atrial tachycardia
- Atrial fibrillation
- Atrial flutter
- AV Nodal reentrant tachycardia (AVNRT)
- AV reentrant tachycardia (AVRT)

Focal Atrial tachycardia



Iultifocal atrial tachycardia



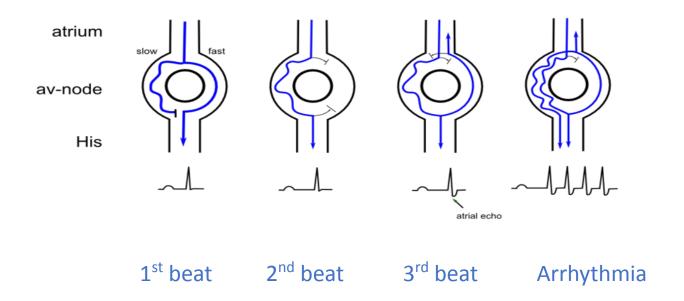
Focal Atrial tachycardia

- originating from a single ectopic focus within the atria but outside of the sinus node.
- these are usually paroxysmal and self-limited.

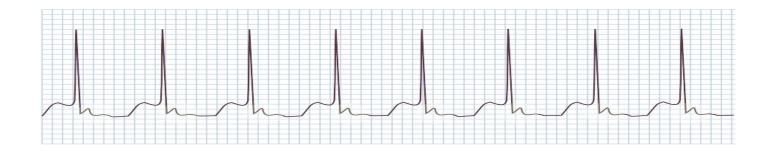
Iultifocal atrial tachycardia

- Usually secondary to underlying illness (usually pulmonary)
- Treatment is based on oxygen therapy and supportive care
- Treat the underlying cause
- Medication maybe used as an alternative therapy if the above fails

Atrioventricular nodal reentrant tachycardi (AVNRT)

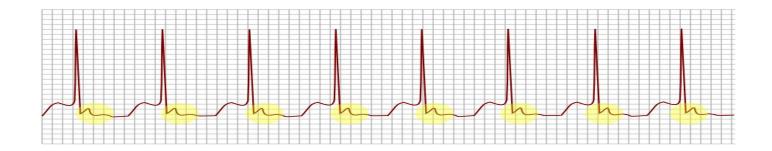


Atrioventricular nodal reentrant tachycardi (AVNRT)



- P waves are either not visible or are seen immediately before or after the QRS complex.
- QRS complex is usually of normal shape.

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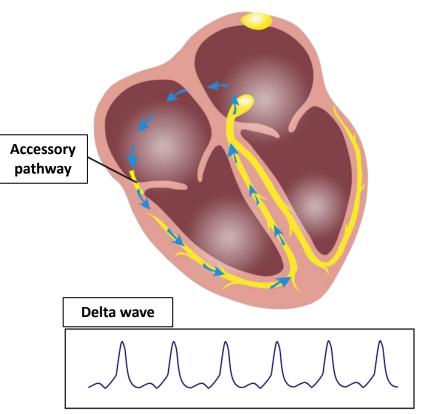


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Atrioventricular reentrant tachycardia (AVRT)

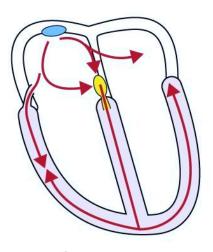
 occurs due to the presence of an accessory pathway that connects the atria and ventricles and is capable of antegrade or retrograde conduction, or both.

- Bypass consequence:
- 1- Asymptomatic with ECG changes
- 2- Regular Tachycardia and palpitation
- 3- Atrial Fibrillation



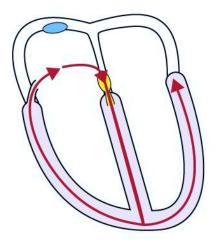
Atrioventricular reentrant tachycardia

Pre-excitation



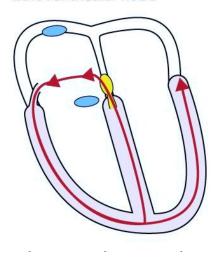
Orthodromic AVRT

Antegrade conduction through atrioventricular node



Antidromic AVRT

Retrograde conduction through atrioventricular node



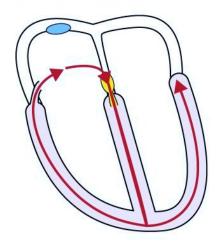
Atrioventricular reentrant tachycardia

Pre-excitation

- Short PR interval
- In this case the PR segment cannot be seen.

Orthodromic AVRT

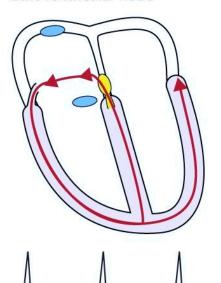
Antegrade conduction through atrioventricular node



- Normal QRS duration
- No delta wave
- Retrograde P-wave after QRS

Antidromic AVRT

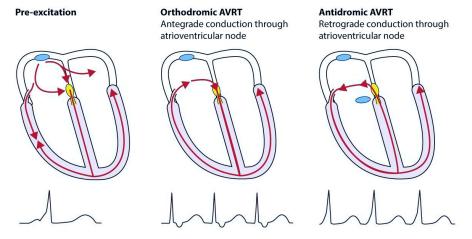
Retrograde conduction through atrioventricular node



- Wide QRS complex with delta wave
- P-wave rarely seen
- If P-wave visible, it is retrograde and occurs just before the QRS

Atrioventricular reentrant tachycardia (AVRT)

- Accessory pathway
- Accessory pathways alters the activation of atria and ventricle



Atrioventricular nodal reentrant tachycardia (AVNRT)

- Nodal reentry
- Activates both atria and ventricles at the same time



Atrioventricular reentrant tachycardia (AVRT)

Managemen t:

Unstable WPW □ DC cardioversion

Stable WPW

- Vagal maneuvers
- Procainamide are available for use in resistant cases
- 3. Involve cardiology consultant

Long-term management: Radiofrequency ablation of the accessory pathway

! Avoid use of calcium channel blocker or IV beta blocker

Atrioventricular nodal reentrant tachycardia (AVNRT)

Managemen

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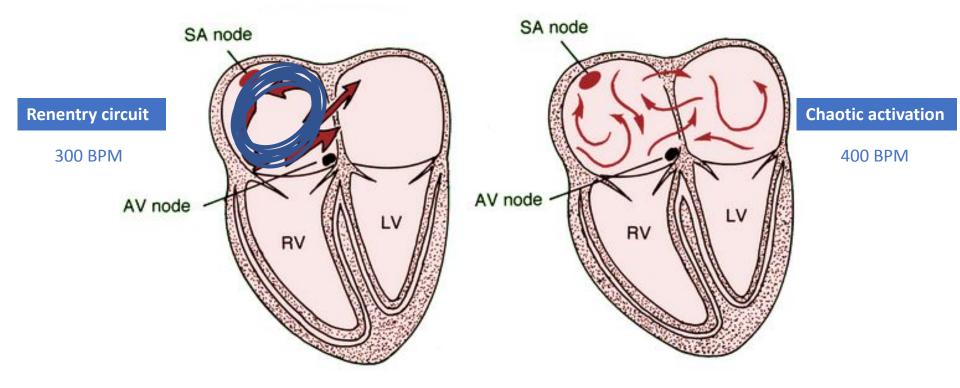
Unstable SVT □ DC cardioversion

Stable SVT

- 1. Vagal maneuvers
- 2. IV adenosine
- 3. IV calcium channel blocker or IV beta blocker

Atrial flutter

Atrial fibrillation



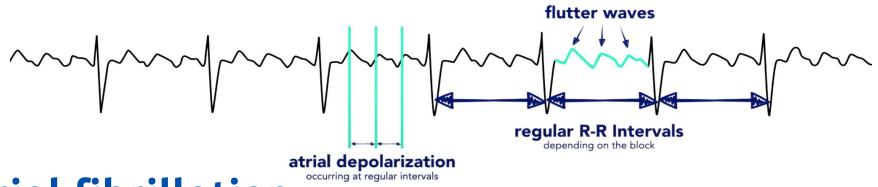
Atrial flutter



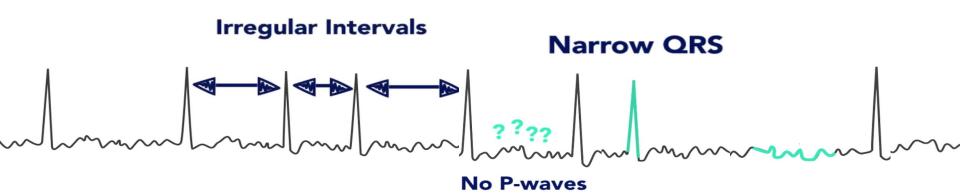
Atrial fibrillation



Atrial flutter



Atrial fibrillation



Atrial flutter management

- Our initial approach to the management of patients with atrial flutter is the same as our approach for atrial fibrillation.
- Managetreia of the little on one an agement of the need for cardioversion.
- Ventricular rate slowing therapy, and anticoagulation therapy.

Risk factors of atrial fibrillation

- 1. Hypertension
- 2. Age (risk factors)
- 3. Coronary artery disease (including acute MI)
- 4. Valvular heart disease, especially rheumatic mitral valve disease
- 5. Sinoatrial disease
- **6.** Hyperthyroidism
- 7. Alcohol
- 8. Cardiomyopathy
- 9. Congenital heart disease
- 10. Pulmonary embolism ,Chest infection
- **11.** Idiopathic (lone atrial fibrillation)

Types of Atrial fibrillation

• First diagnosed:

Not diagnosed previously, irrespective of duration or presence of AF-related symptoms

Paroxysmal AF = intermittent

characterized as sporadic episodes. Self-terminating, in most cases within 48 hours, but could last up to 7 days.

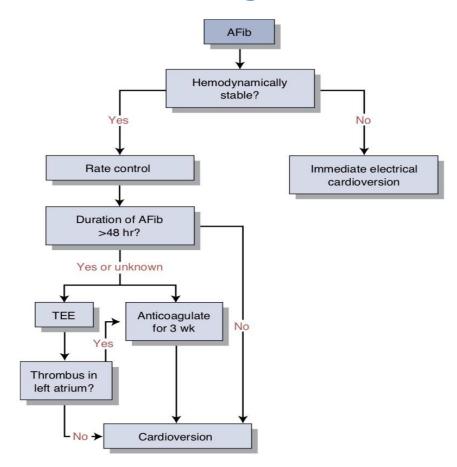
Persistent AF:

Lasts longer than 7 days and unlikely to resolve without treatment

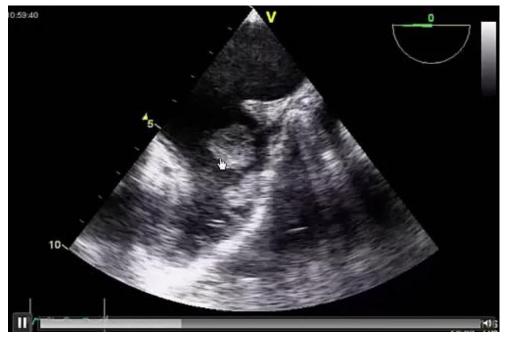
Permanent:

Can not be reverted to normal sinus rhythm despite treatment

Acute management:



A.Fibb increase the risk of thrombus formation



There is a risk of stroke when performing chemical/electrical cardioversion

Pharmacological cardioversion

When attempting pharmacological cardioversion, use is based on duration of symptoms.

For A-fib> 7 days:

- 1st line: dofetilide (class III antiarrhythmic agent)
- 2nd line: amiodarone or ibutilide

For A-fib< 7 days:

- 1st line: dofetilide, ibutilide,flecainide or propafenone (previously, dronedarone*)
- 2nd line: amiodarone (Exception: If< 48 hours and poor cardiac function, amiodarone is 1st line.)</p>

Chronic A.fib Management

1) Rate control:

- with a B blocker or Calcium channel blocker

2) Anticoagulants:

to prevent cardioembolic cerebrovascular accident (CVA), risk stratify patients with CHA2DS2-VASc score.

- For patients with a score >1 anticoagulation is generally indicated unless high bleeding risk.

Types of anticoagulants:

- A) warfarin: anticoagulant of choice in patients with valvular disease, mechanical valves, or ventricular assist devices.
- B) direct oral anticoagulants (DOACs): factor Xa inhibitors (apixaban), and direct thrombin

CHADS-VASC

Letter	Risk factor	Score
С	Congestive heart failure/LV dysfunction	1
Н	Hypertension	1
A_2	Age ≥75	2
D	Diabetes mellitus	1
S_2	Stroke/TIA/thrombo-embolism	2
V	Vascular disease*	1
Α	Age 65-74	1
S	Sex category (i.e., female sex)	1
	Maximum score	9

Congestive heart failure/LV dysfunction means LV ejection fraction ≤40%. Hypertension includes the patients with current antihypertensive medication. *Prior myocardial infarction, peripheral artery disease, aortic plaque. LV: left ventricular, TIA: transient ischemic attack

CHA₂DS₂VASC

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Radiofrequency Ablation

Radiofrequency ablation of the AV node with subsequent permanent pacing

is a treatment for patients with refractory Afib and for those who cannot tolerate the meds needed for rate or rhythm control, HFrEF, & high Afib burden.

Radiofrequency ablation, or isolation of the pulmonary veins

Is a procedure becoming increasingly popular in treating recurrent, drug refractory, symptomatic A-fib, although it is not yet established as 1st line therapy

Postoperative A.Fib

- Is usually has good prognosis and is short-lived
- For patients undergoing cardiac surgery, give an oral beta-blocker to prevent postoperative Afib (unless contraindicated).
- For those who develop postoperative Afib, achieve rate control with AV nodal blocking drugs (beta-blockers, calcium channel blockers, or digoxin).
- Routine postoperative amiodarone is not indicated for the prevention of atrial fibrillation

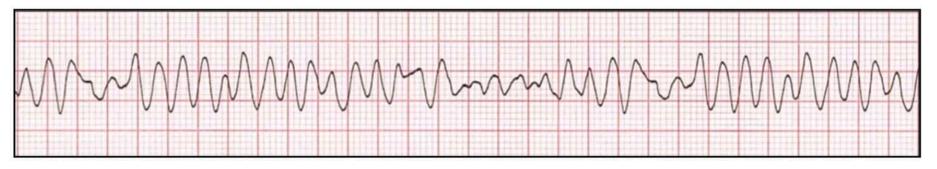
Atrial flutter with variable AV block



Ventricular tachyarrhythmias

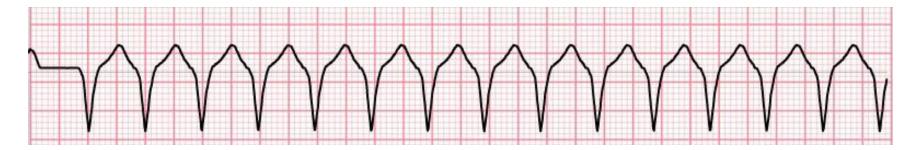
- Ventricular fibrillation
- Ventricular tachycardia

Ventricular Fibrillation



- Rhythm: chaotic & disorganized waves
- Patient is unconscious (lethal)
- It is an emergency situation, requires CPR
- Shockable rhythm requires defibrillation (unsynchronized DC shock)

Ventricular tachycardia



- Types: monomorphic Vs polymorphic (poor prognosis)
- Rhythm: wide complex tachycardia
- Patient stability varies (stable, unstable, unconscious)
- Management varies depending on clinical stability

Management of ventricular fibrillation:

Immediate assessment of patient stability takes precedence over any further diagnostic evaluation.

- Unresponsive or pulseless: cardioversion (without sedation) + epinephrine +/- amiodarone
 (ACLS*)
- Unstable but conscious: immediate synchronized cardioversion (with sedation)
- Stable patient: a focused diagnostic evaluation may proceed to determine the etiology of the arrhythmia and guide specific therapy.

^{*}ACLS = advance cardiac life support

Management of ventricular fibrillation:

For sustained stable monomorphic VT:

Give IV <u>amiodarone</u>.

Treat according to diagnosis:

VT specifically with acute MI: Most use amiodarone first. IV lidocaine can be useful.

Do not use CCBs for wide-complex tachycardias. (fear of hemodynamic collapse, hypotension and cardiac arrest)

- RVOT VT can be terminated acutely with adenosine. Beta-blockers/calcium channel blockers (CCBs) can be used for long-term management.
- Patients with NSVT without structural heart disease have good prognosis, and they do not require further management.

^{*}ACLS = advance cardiac life support

Unstable patients: Defibrillation improves mortality



Healthcare setting:

Cardioversion

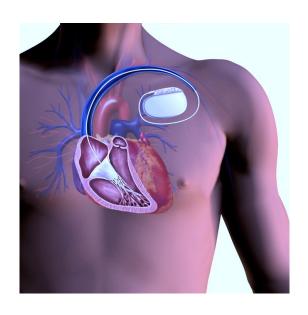


Ambulatory setting:

automated Eternal Defibrillator (AED)

Immediate Synchronized cardioversion + medications

Dealing with future V.tach



- Ideally all patient with sustained VT should undergo placement of Implantable Implantable cardioverter-defibrillator (ICD)
- It has longer benefits

Monomorphic ventricular tachycardia



Polymorphic ventricular tachycardia



Treatment of Polymorphic ventricular tachycardia

In addition to immediate defibrillation, further therapy is intended to treat underlying disorders and to prevent recurrences.

- QT prolongation + V.tach = Torsades de pointes.
 - Intravenous magnesium sulfate is first-line therapy, as it is highly effective for both treatment and prevention of recurrence.
 - Correct metabolic abnormailities including electrolytes
 - Other treatments: ATP pacing, beta-blocker (congenital)
- V.tach + no QT prolongation = the most likely cause is myocardial ischemia
 - Betablocker (if BP tolerates)
 - Amiodarone to prevent recurrence
 - Angiography and revascularization
 - Intravenous magnesium sulfate is ineffective

Thank you