Life-Threatening ECG Rhythms: Identification & Management

Recognize rhythms, underlying causes, and emergency treatment





MENU

- Fundamentals of ECG Interpretation
- Ventricular Fibrillation and Ventricular Tachycardia
- Asystole and Pulseless Electrical Activity
- Supraventricular Arrhythmias with Life-Threatening Potential
- Bradyarrhythhmia ie SND and CHB
- Emergency Management and Treatment Strategies

Fundamentals of ECG Interpretation


HR
LEAD
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Asystole and Pulseless Electrical Activity

Recognizing Asystole on ECG

Identification

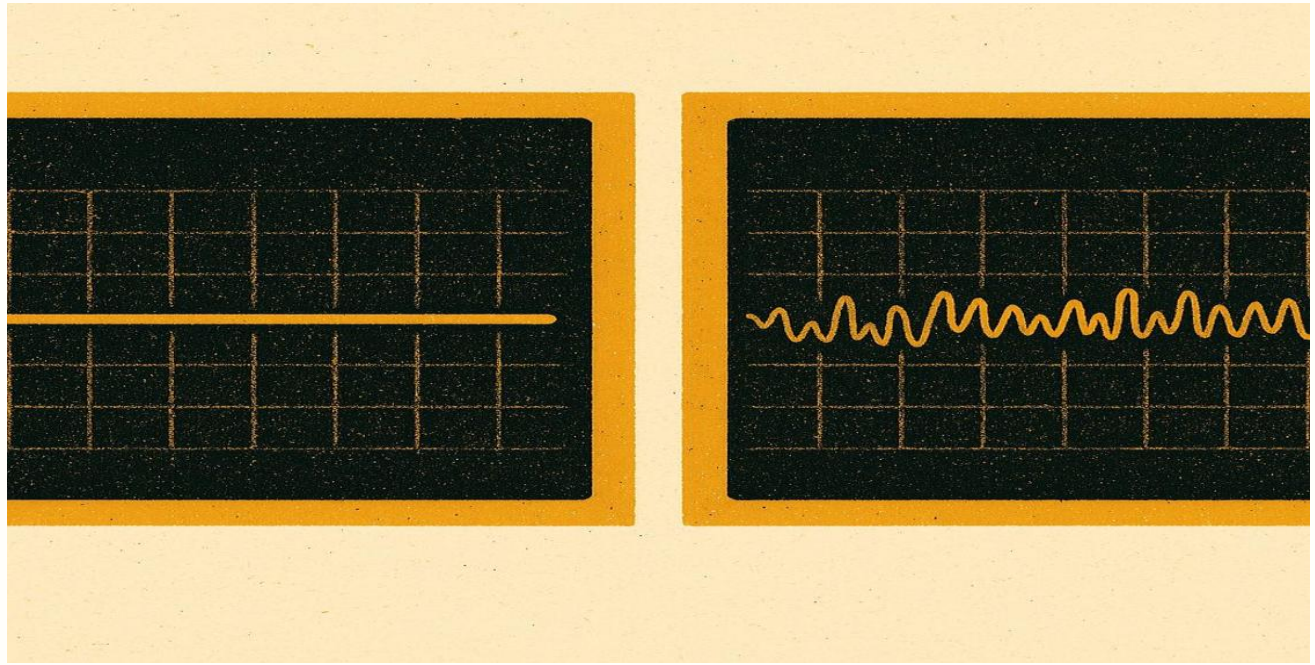
Flatline tracing with no ventricular activity and no pulse—true cardiac arrest.

Differentiate from fine VF

Fine VF shows irregular, low-amplitude undulations—optimize gain and check multiple leads.

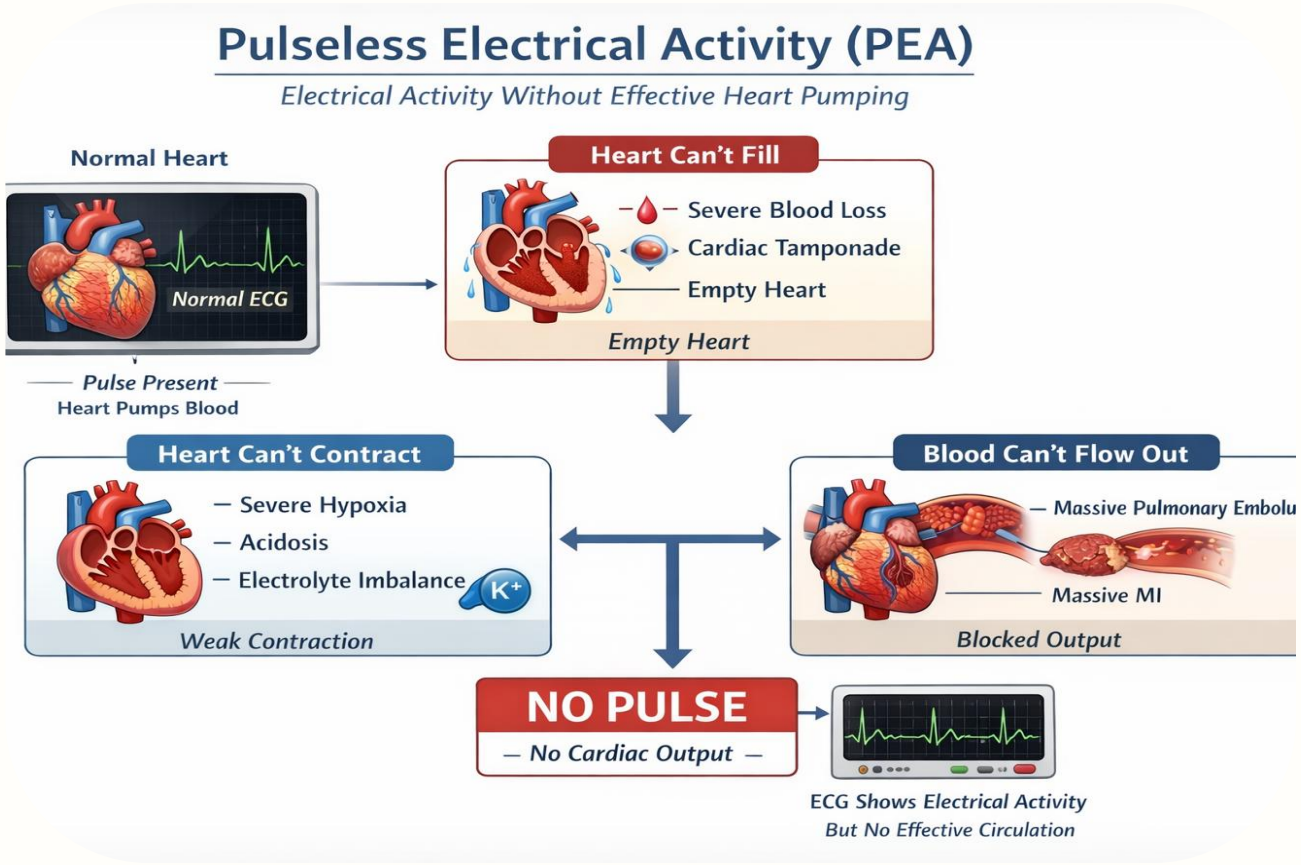
Clinical implications

Poor prognosis; treat as PEA/asystole—CPR, epinephrine, and reversible causes (Hs & Ts).



PULSELESS ELECTRICAL ACTIVITY

- Pulseless Electrical Activity (PEA) is a cardiac arrest condition where organized electrical activity is seen on the ECG, but there is no palpable pulse or effective cardiac output.In simple terms:
- the heart's electrical system is working, but the heart muscle isn't pumping blood.
- That mismatch happens when the heart **can't fill, can't contract effectively, or can't push blood out**



Understanding Pulseless Electrical Activity (PEA)

Pathophysiology, recognition, causes, and management considerations

Definition & Core Concept

PEA is organized electrical activity on ECG without a palpable pulse due to mechanical pump failure. Electrical signals do not translate into effective circulation, requiring immediate search for reversible causes.

Common Reversible Causes

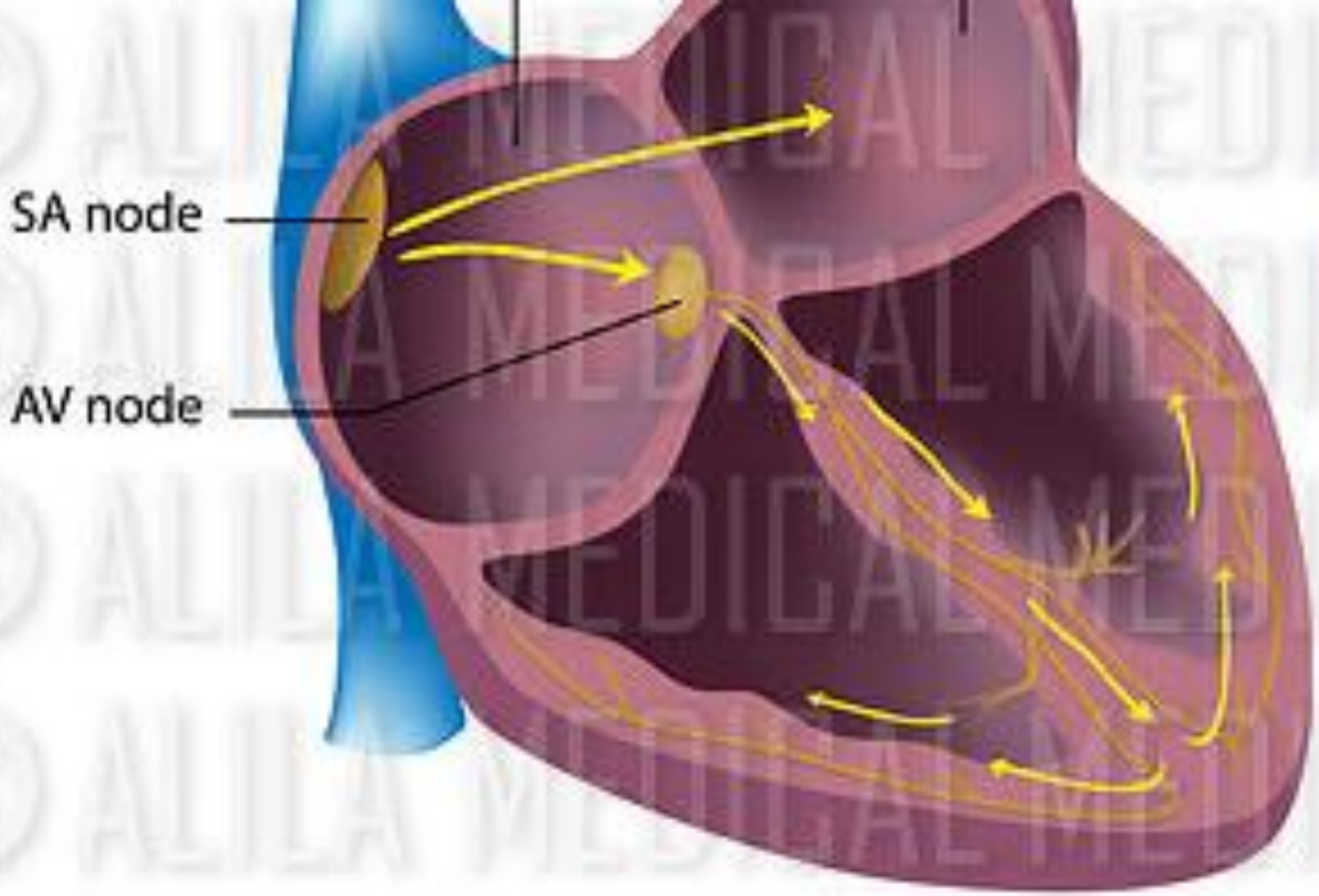
Frequent etiologies include severe hypovolemia, cardiac tamponade, tension pneumothorax, and massive pulmonary embolism. Rapid identification and targeted treatment can restore mechanical function.

Recognition & Diagnostic Approach

Confirm unresponsiveness and absent pulse with organized ECG activity. Perform rapid bedside assessment for reversible causes; electrical activity alone must not delay interventions that restore perfusion.

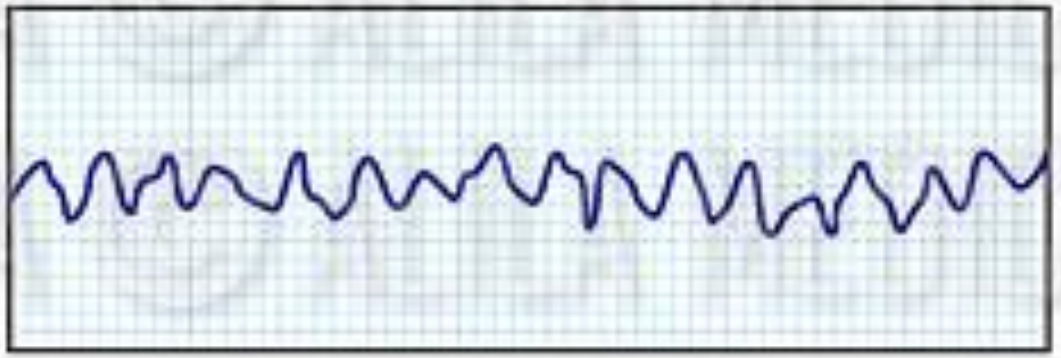
Prognosis & Management Focus

PEA carries high mortality but may be reversible with prompt, cause-directed therapy. Emphasize high-quality CPR, early epinephrine, and immediate correction of the suspected etiology.









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Courtesy of Jason E. Roediger, CCT, CRAT
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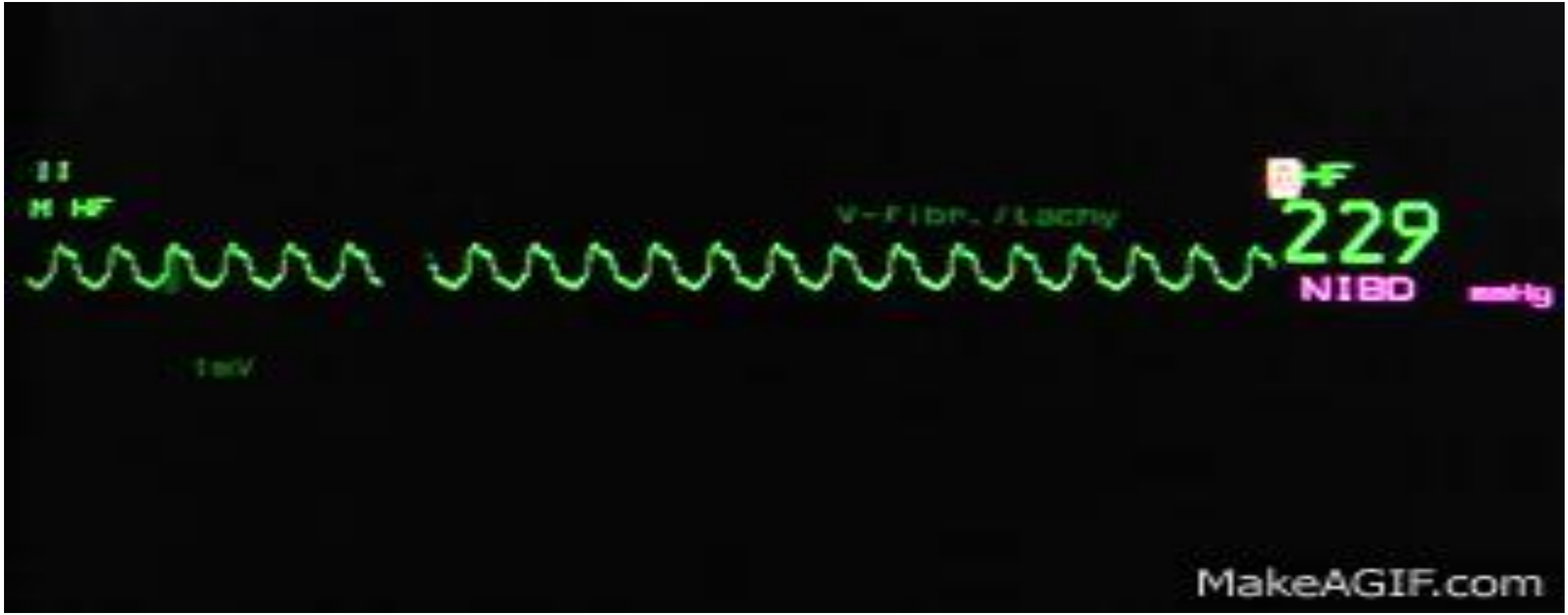
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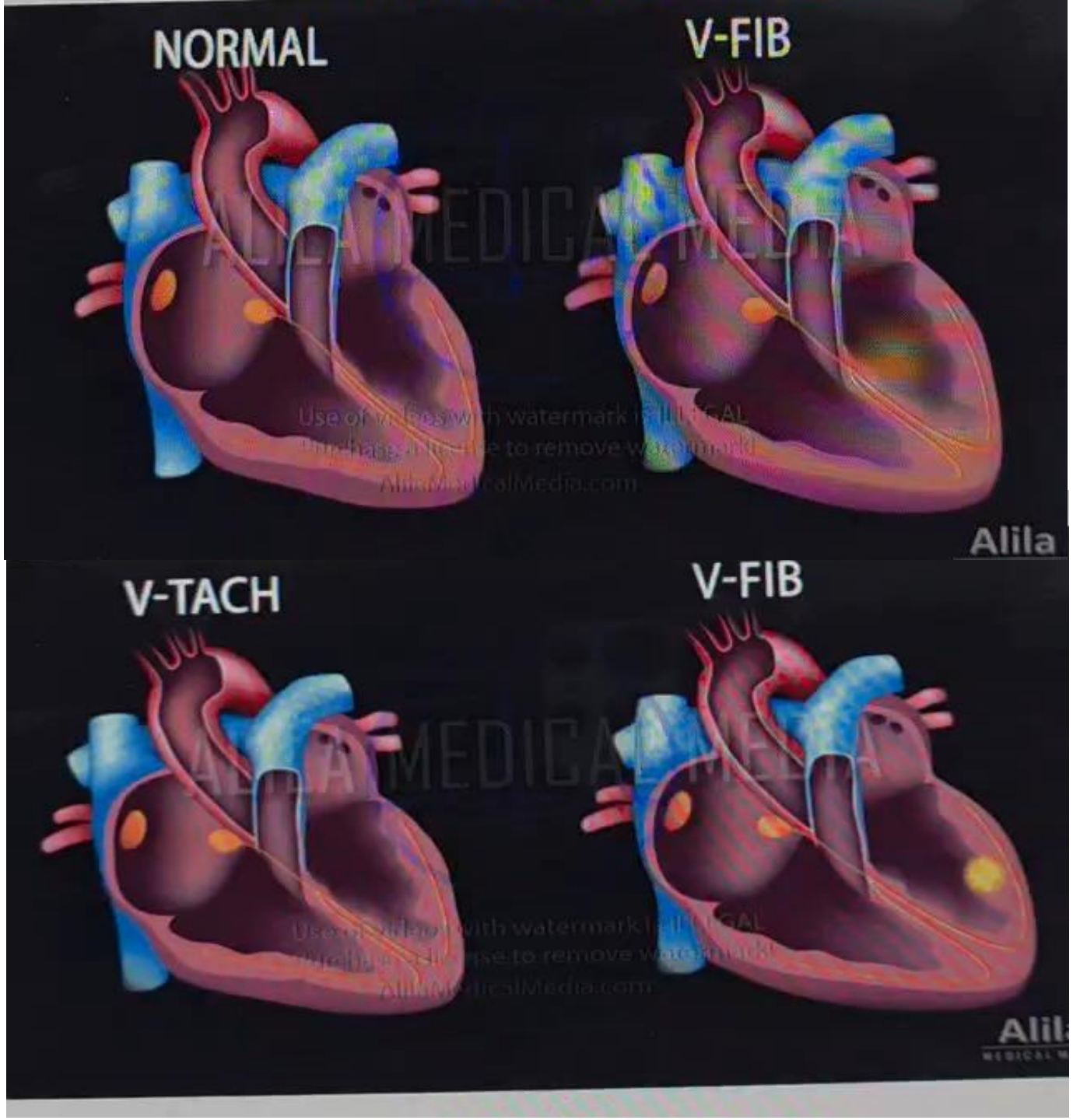
NORMAL
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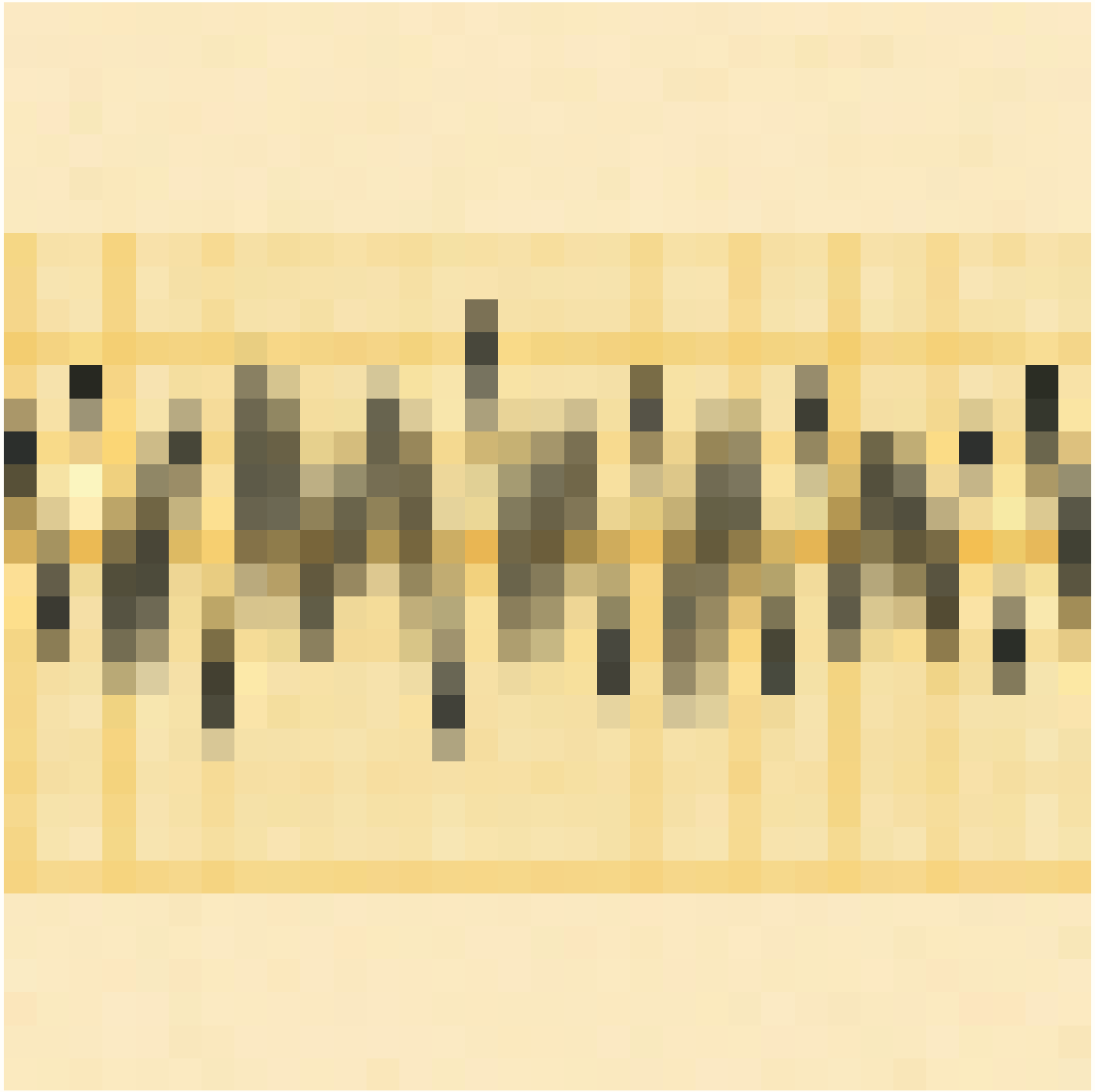


Characteristics of Ventricular Fibrillation

ECG Appearance

Chaotic, irregular, disorganized waveforms with no identifiable QRS complexes; variable amplitude and frequency.

Hemodynamic Consequence

No effective cardiac output due to absence of organized ventricular contraction.

Clinical Context & Action

Most common cause of sudden cardiac arrest; immediate defibrillation is essential. Incidence increases with ischemic heart disease and cardiomyopathies.

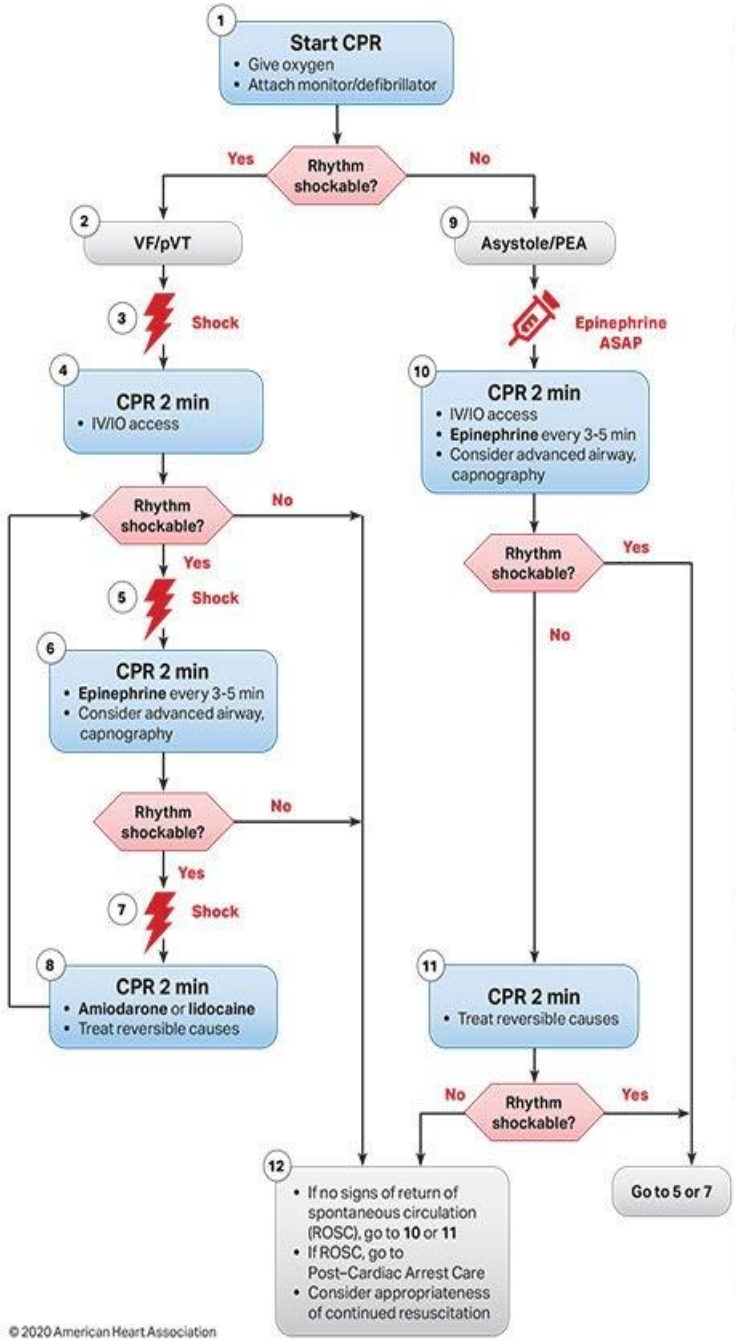
Identification and Significance of Ventricular Tachycardia

>100 bpm

Heart rate threshold for diagnosing VT on ECG

Wide QRS tachycardia from ventricular foci; prompt recognition prevents hemodynamic compromise and deterioration to VF.

Adult Cardiac Arrest Algorithm (VF/pVT/Asystole/PEA)



CPR Quality

- Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
  - If P_{ETCO_2} is low or decreasing, reassess CPR quality.

Shock Energy for Defibrillation

- **Biphasic**: Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic**: 360 J

Drug Therapy

- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus. Second dose: 150 mg.
  or
  Lidocaine IV/IO dose: First dose: 1-1.5 mg/kg. Second dose: 0.5-0.75 mg/kg.

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
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- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

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Adult Tachycardia With a Pulse Algorithm

Assess appropriateness for clinical condition.
Heart rate typically ≥150/min if tachyarrhythmia.

↓

Identify and treat underlying cause

- Maintain patent airway; assist breathing as necessary
- Oxygen (if hypoxemic)
- Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
- IV access
- 12-lead ECG, if available

↓

Persistent tachyarrhythmia causing:

- Hypotension?
- Acutely altered mental status?
- Signs of shock?
- Ischemic chest discomfort?
- Acute heart failure?

Yes → **Synchronized cardioversion**

- Consider sedation
- If regular narrow complex, consider adenosine

No ↓

Wide QRS? ≥0.12 second

Yes → **Consider**

- Adenosine only if regular and monomorphic
- Antiarrhythmic infusion
- Expert consultation

No ↓

- Vagal maneuvers (if regular)
- Adenosine (if regular)
- β-Blocker or calcium channel blocker
- Consider expert consultation

If refractory, consider

- Underlying cause
- Need to increase energy level for next cardioversion
- Addition of antiarrhythmic drug
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Doses/Details

Synchronized cardioversion:
Refer to your specific device's recommended energy level to maximize first shock success.

Adenosine IV dose:
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NEW: AHA recommends referring to your specific device's recommended energy level to maximize first shock success.

Initial recommended doses:

- Narrow regular: 50-100 J
- Narrow irregular: 120-200 biphasic or 200 J Monophasic
- Wide regular: 100J
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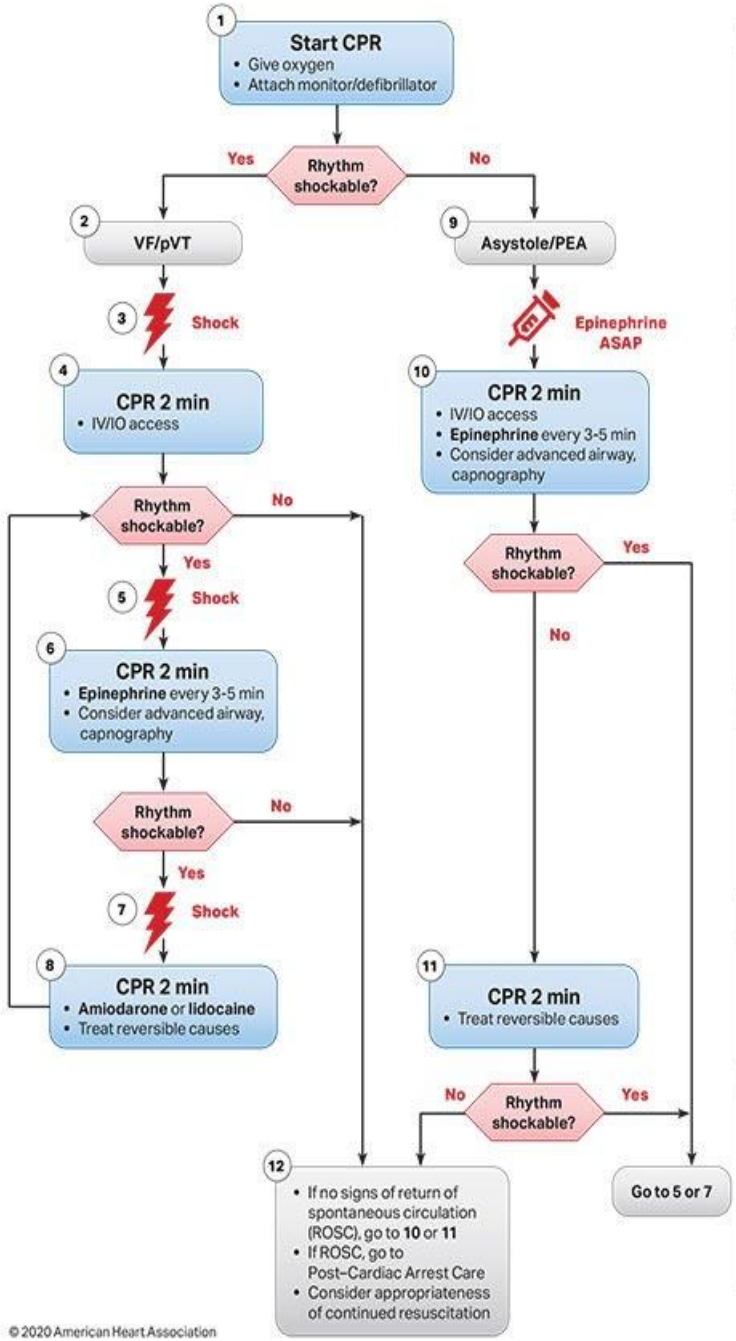

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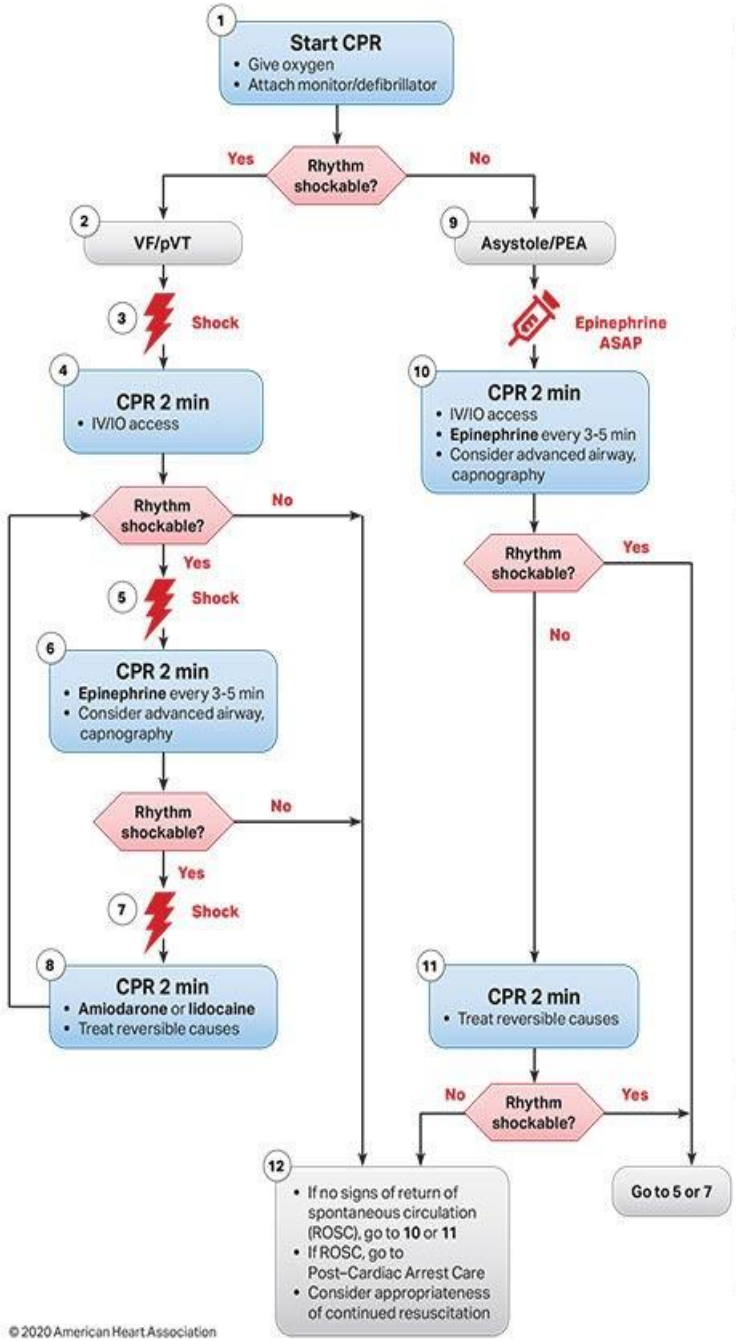

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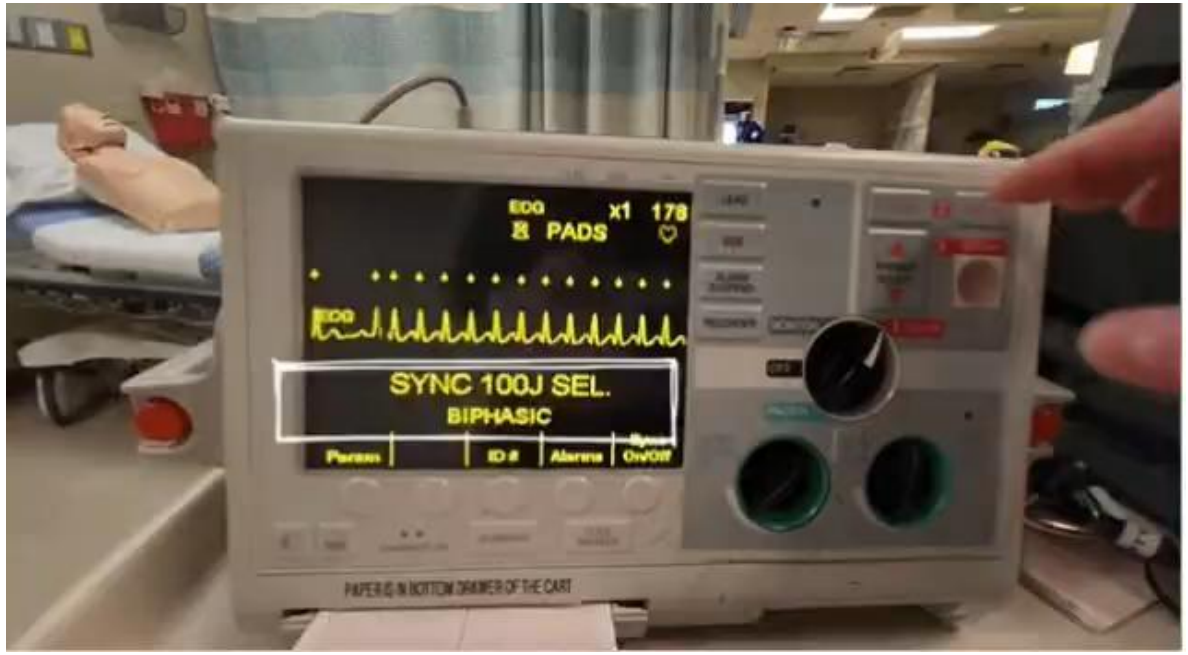
stant Clips



SYNC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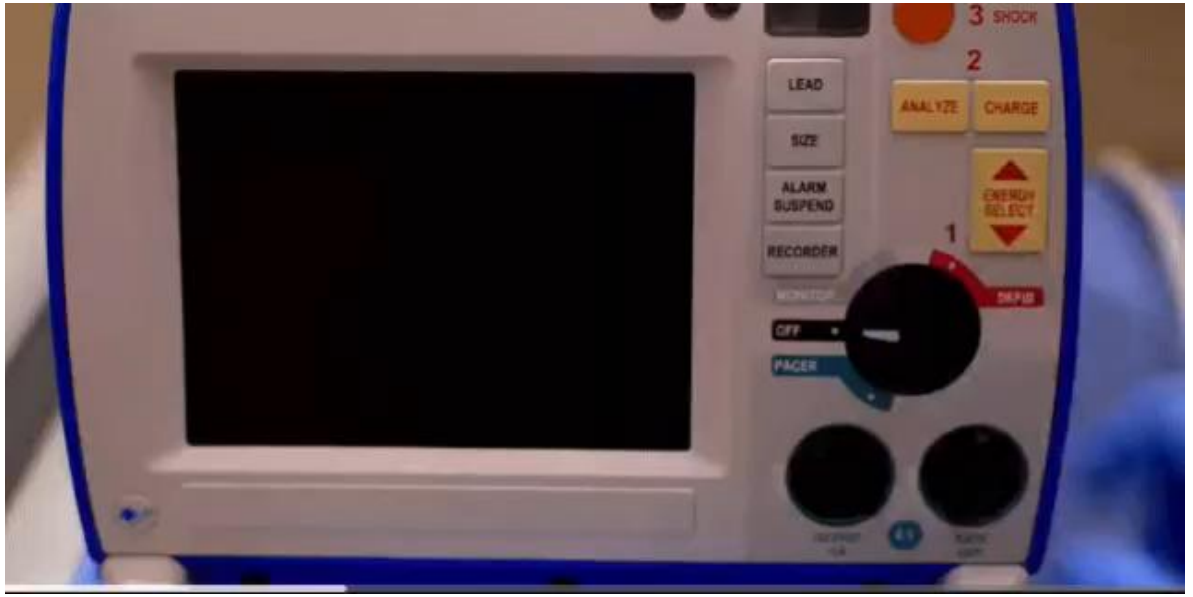
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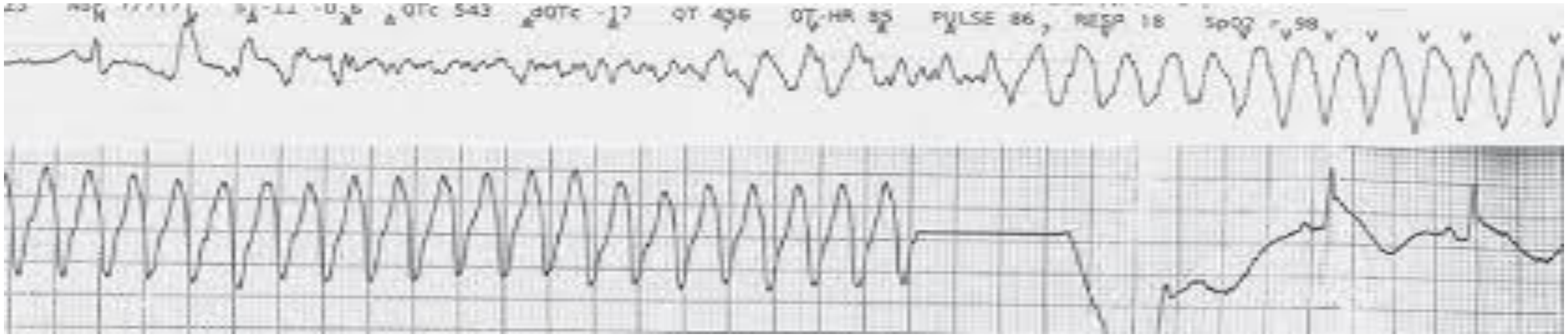
Acute Management

- Immediate defibrillation/ CV + CPR.
- Correct hypoxia, acidosis, and electrolytes.
- IV amiodarone or lidocaine for refractory VT/VF.
- Beta-blockers post-stabilization.

📚 Guidelines: ESC 2022 VA/SCD Class I; AHA/ACC 2017, Class I.


QTc 543
ΔQTc 17
QT 496
QT-HR 85
PULSE 86,
RESP 18
SpO2 98

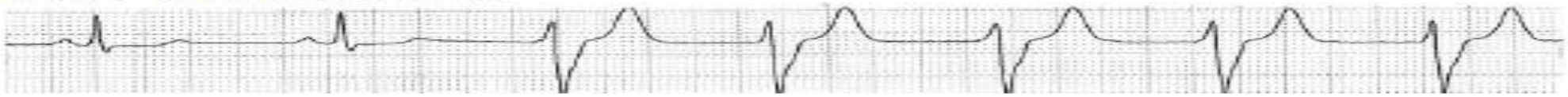
ECG MASTERCLASS

a Ventricular escape rhythm.

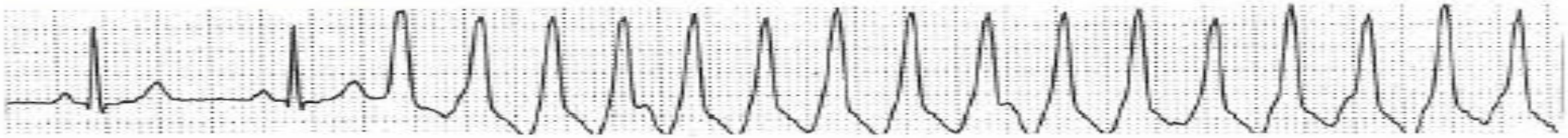
II



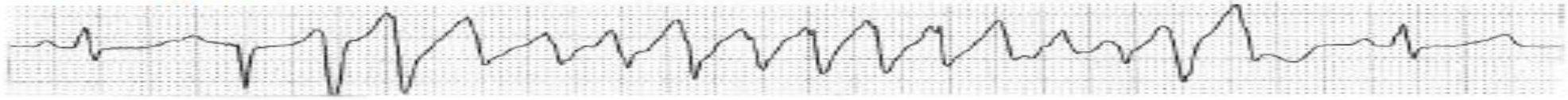
b. Accelerated idioventricular rhythm.



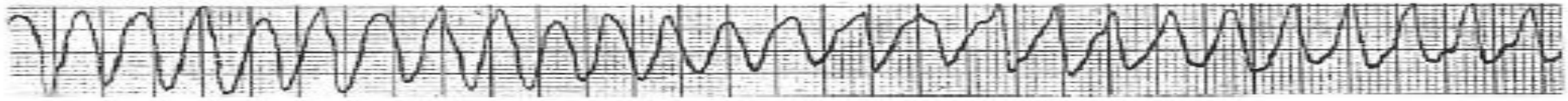
c. Ventricular tachycardia.



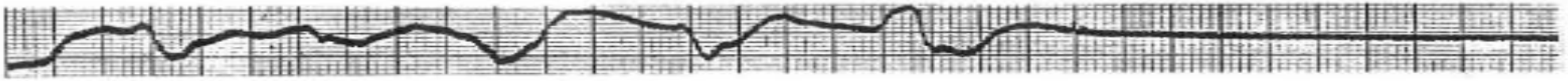
d. Torsade de pointes.



e. Ventricular flutter.



f and g. Ventricular fibrillation followed by asystole.



Supraventricular Arrhythmias with Life-Threatening Potential

Supraventricular Tachycardia Leading to Compromise

01

SVT Onset

Abrupt increase in heart rate (150–250 bpm) with narrow QRS complexes.

02

Hemodynamic Effects

Reduced ventricular filling leads to hypotension, syncope, or ischemia.

03

Recognition

Prompt ECG identification of the SVT pattern.

04

Initial Response

Attempt vagal maneuvers to terminate the arrhythmia.

05

Escalation

Adenosine if vagal fails; immediate cardioversion if unstable.

Atrial Fibrillation with Rapid Ventricular Response

ECG Characteristics

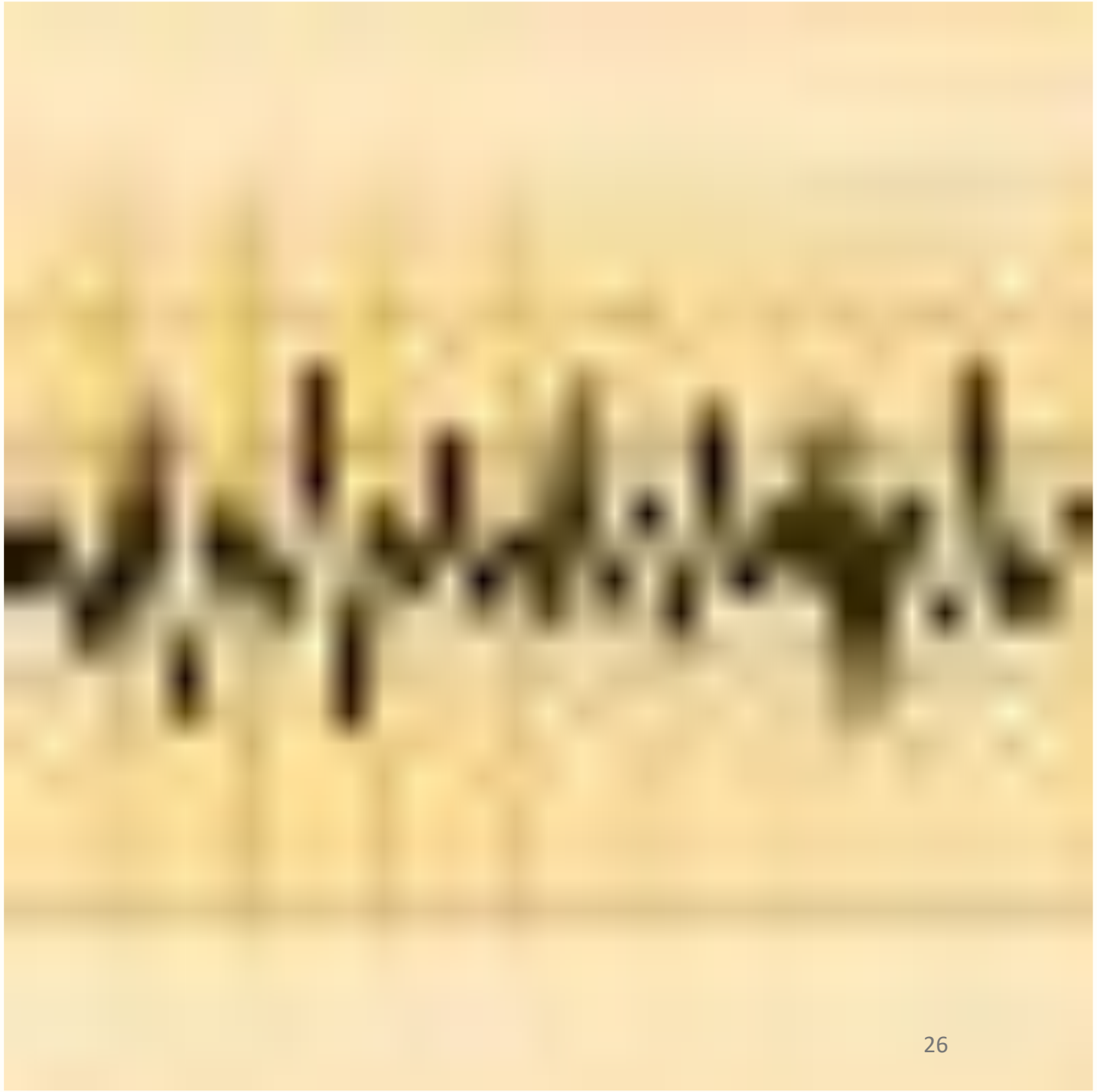
Irregularly irregular rhythm, absent discrete P waves, ventricular rate >100 bpm.

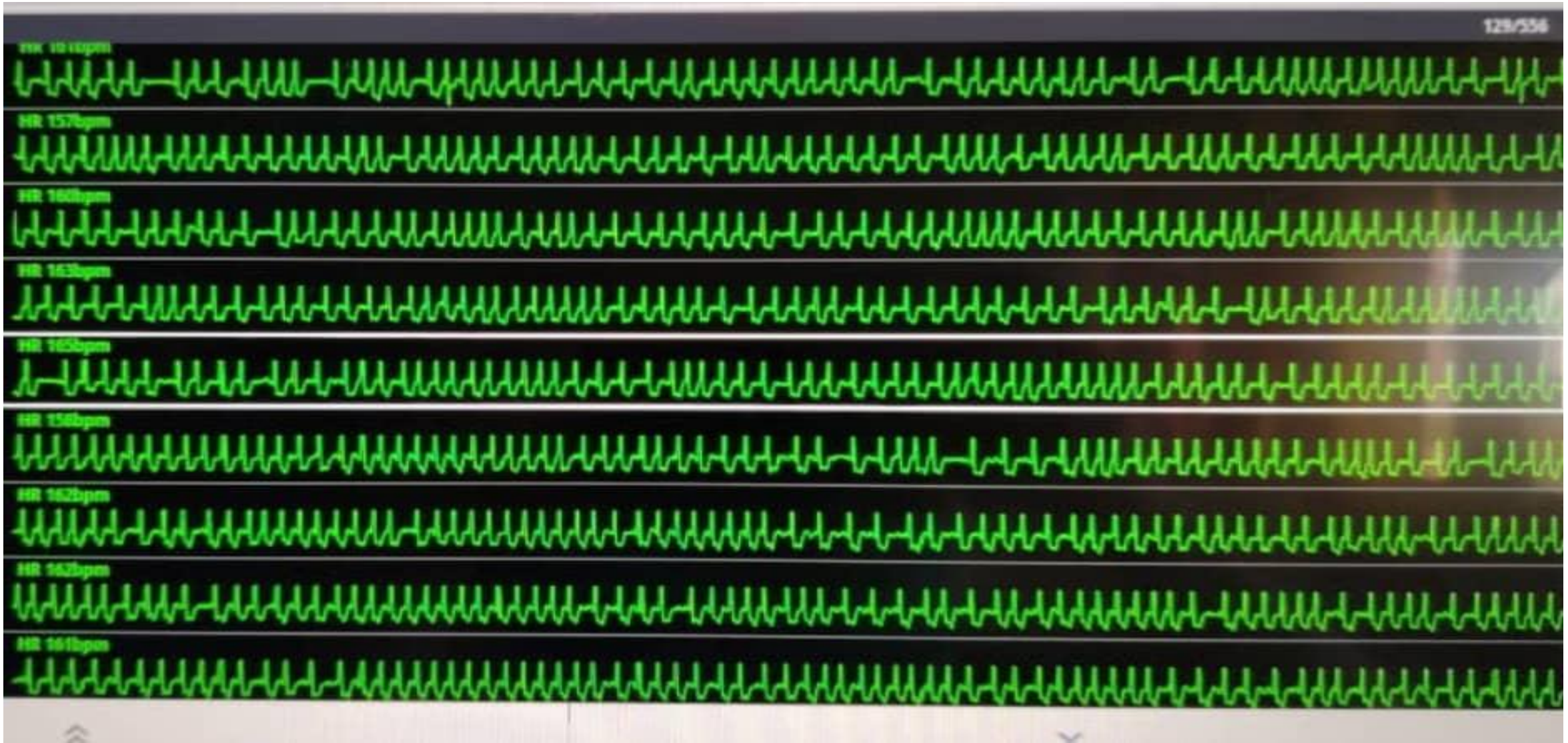
Clinical Impact

Rapid rates reduce cardiac output; may trigger ischemia or heart failure.

Management Priorities

Rate control, rhythm control, and anticoagulation to prevent morbidity/mortality.




129/556

Atrial Fibrillation

- Rate control: β-blocker ± digoxin.
- Rhythm control: chemical: amiodarone. Electric: CV
- Anticoagulation: CHA_2DS_2-VASc guided.

📚 Guidelines: ESC 2024 AF; AHA 2019 AF Focused Update

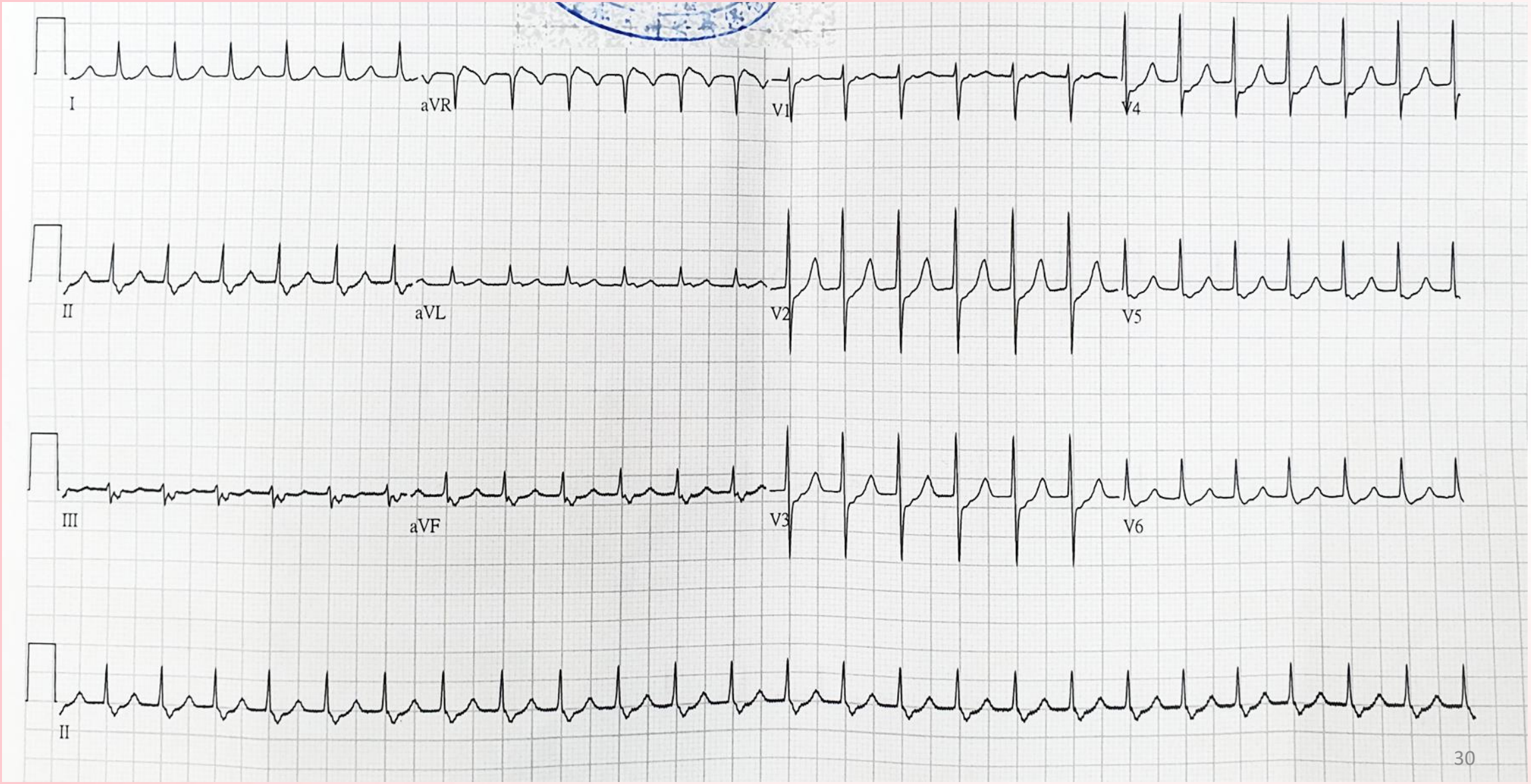
SVT- AVNRT/AVRT/AT/AFLUTTER

- Less common but possible due to
  - sympathetic surge,
  - atrial ischemia, or scarring.

- Typically transient and not prognostically significant unless causing hemodynamic compromise.

- Management:
  - Treat ischemia first (often SVT resolves).
  - If persistent and symptomatic: vagal maneuvers → adenosine → β-blockers.
  - Avoid verapamil/diltiazem if LV dysfunction is present.

🗐 Guidelines: ESC 2023: Management of ACS


I
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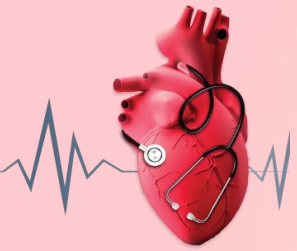
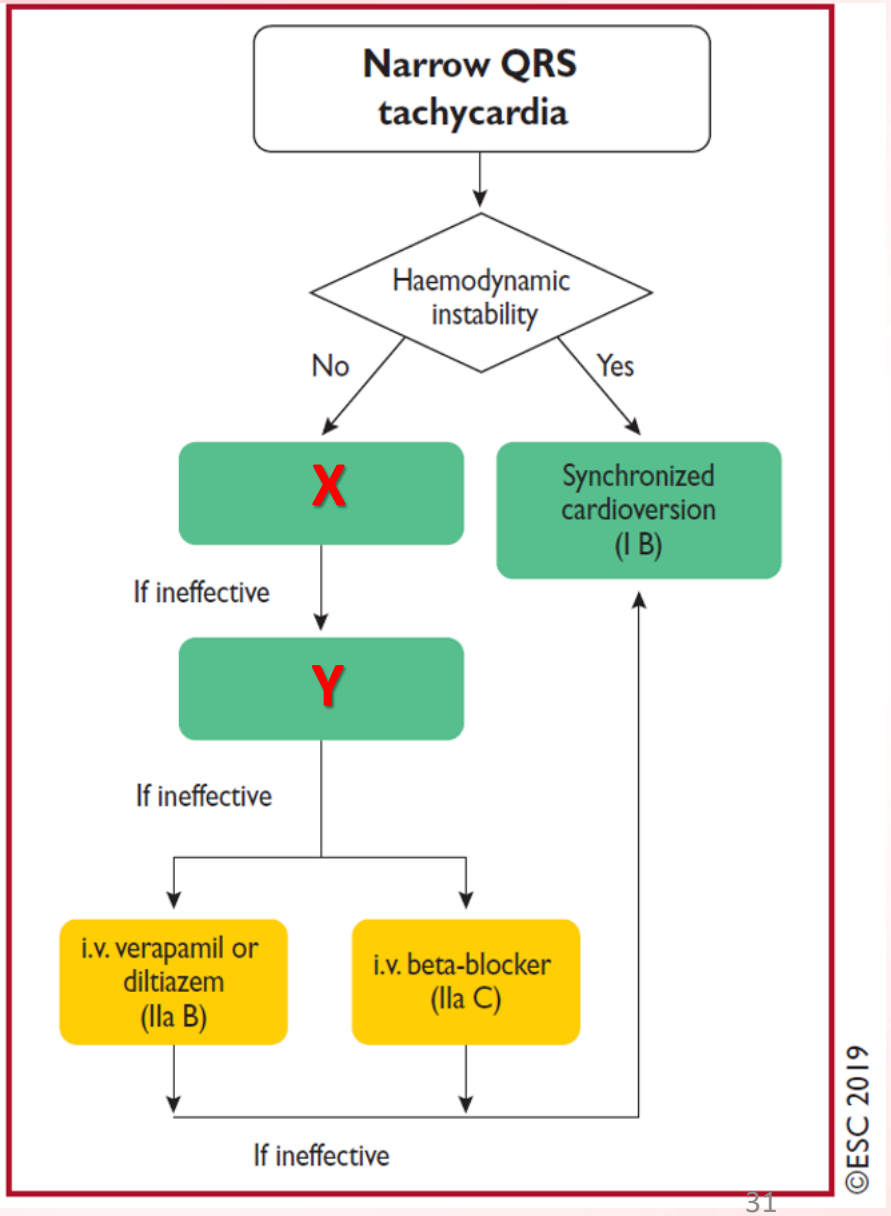
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II



What is X?

A: Cardioversion
B: IV Adenosine
C: Carotid massage
D: IV Amiodarone



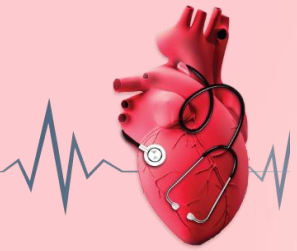


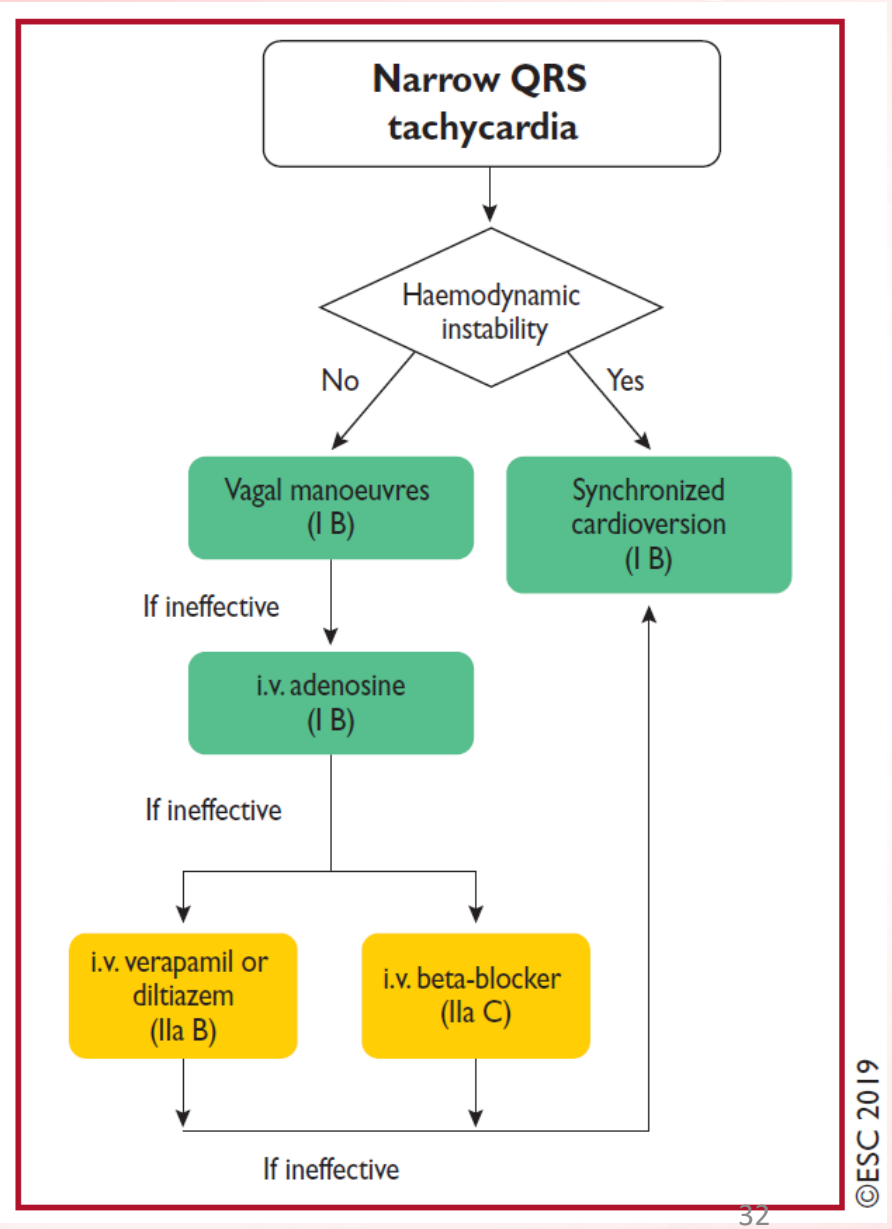
What is X?

A: Cardioversion
B: IV Adenosine
C: Carotid massage
D: IV Amiodarone

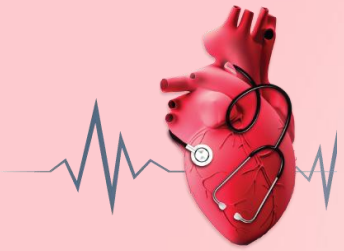
Carotid massage:

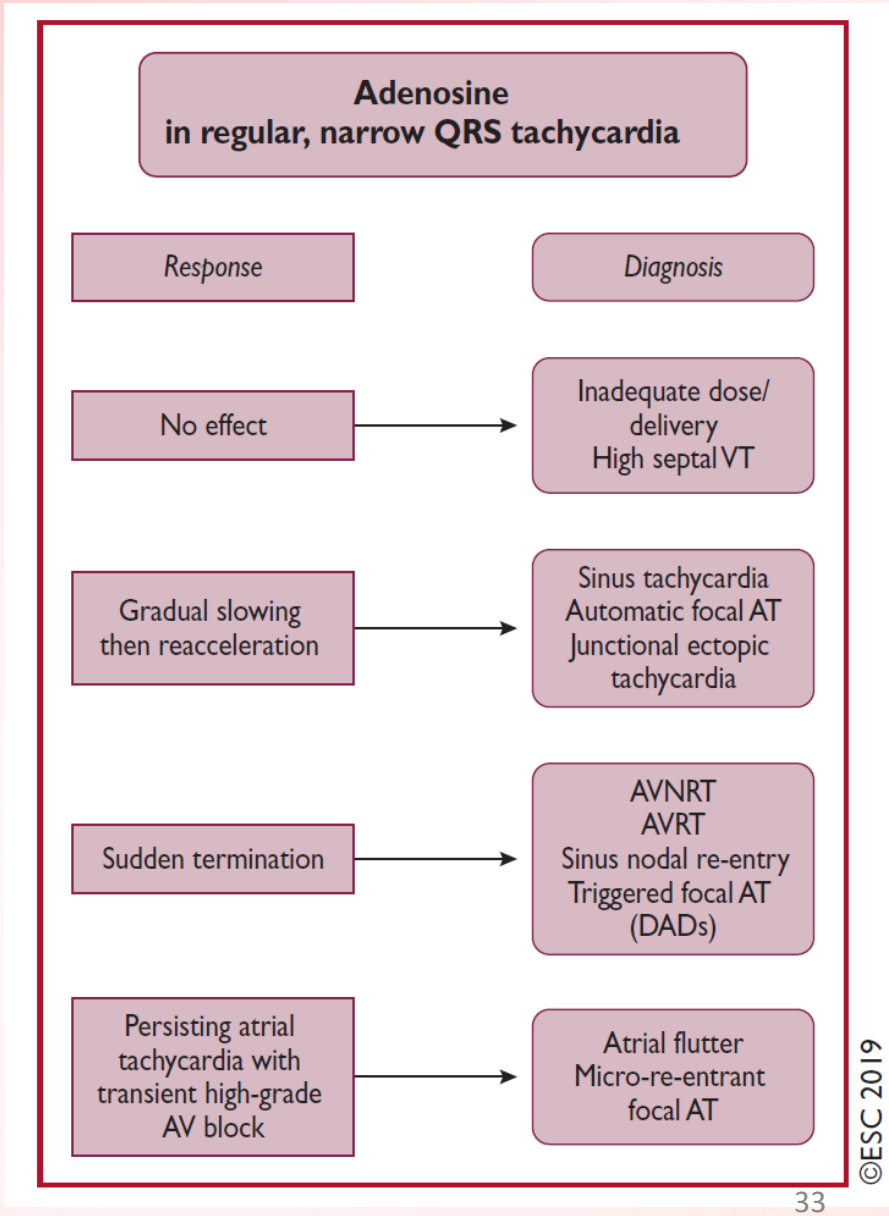
- Not massage both side at the same time
- Not for carotid bruit patient





Adenosine




Adenosine
in regular, narrow QRS tachycardia
Response
Diagnosis
No effect
Inadequate dose/ delivery
High septal VT
Gradual slowing then reacceleration
Sinus tachycardia
Automatic focal AT
Junctional ectopic tachycardia
Sudden termination
AVNRT
AVRT
Sinus nodal re-entry
Triggered focal AT (DADs)
Persisting atrial tachycardia with transient high-grade AV block
Atrial flutter
Micro-re-entrant focal AT
©ESC 2019

Adenosine given



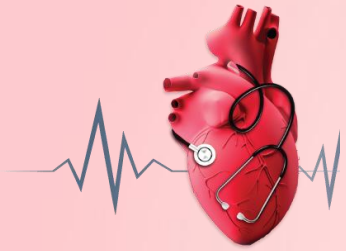
What is your diagnosis?

ECG taken from : Youtube of Dr. Joshua Cooper - Arrhythmia Education

Adenosine given

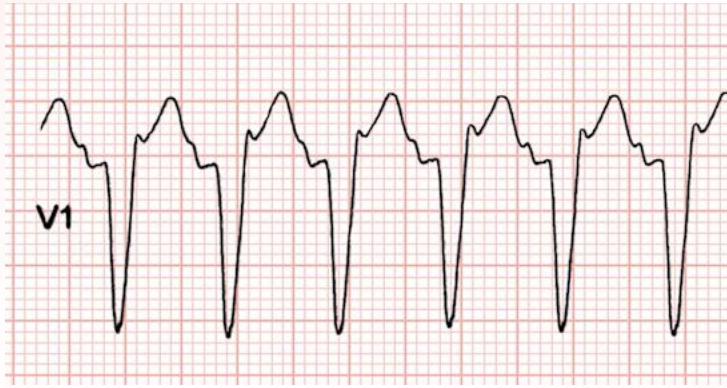
AV node recovers





Sudden termination	→	AVNRT AVRT Sinus nodal re-entry Triggered focal AT (DADs)

Adenosine given



What is your diagnosis?

ECG taken from : Youtube of Dr. Joshua Cooper - Arrhythmia Education

Adenosine given

Atrial beats

Ventricular escape beats

Tachycardia resumes

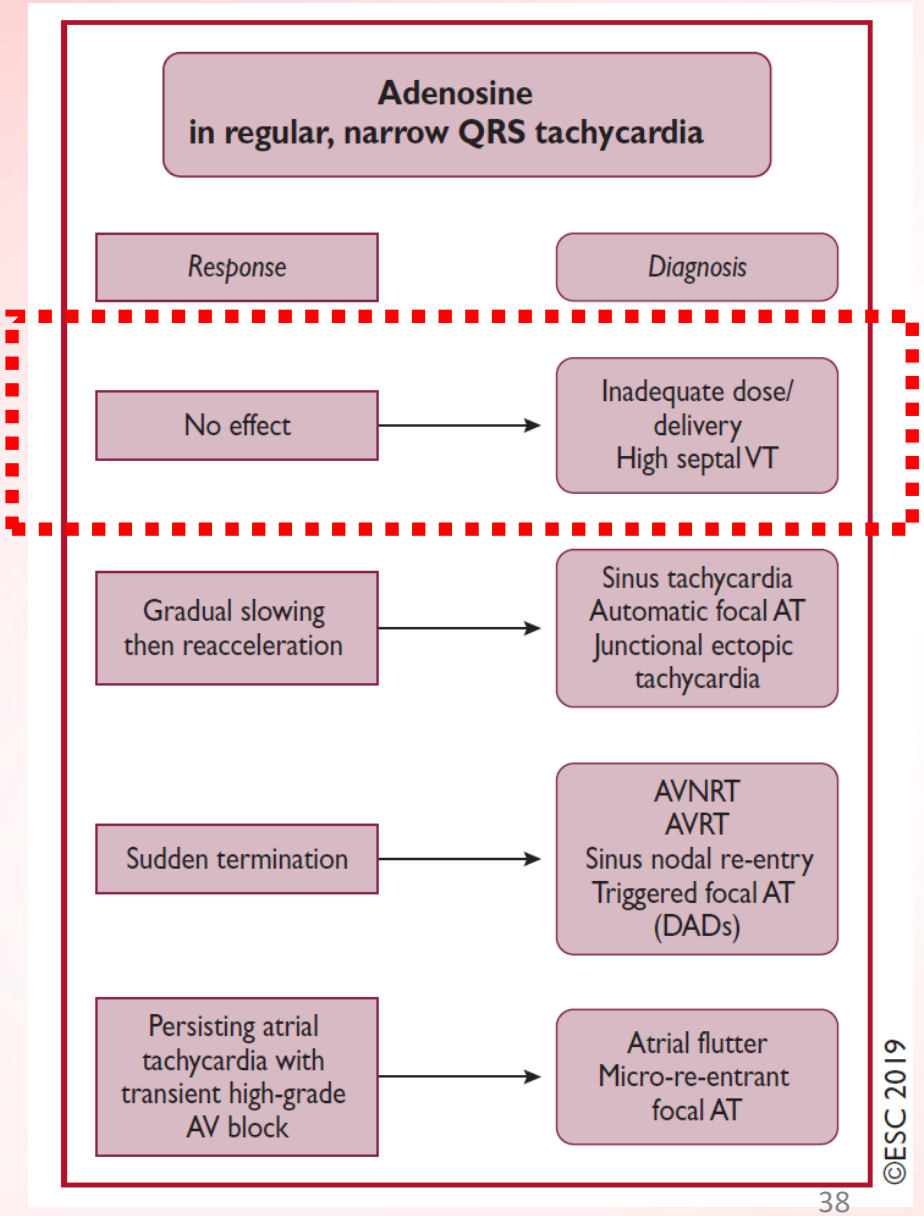


Persisting atrial tachycardia with transient high-grade AV block → Atrial flutter
Micro-re-entrant focal AT

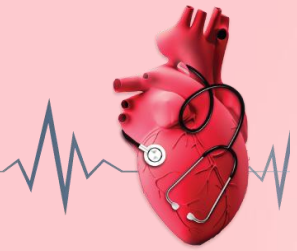
Adenosine

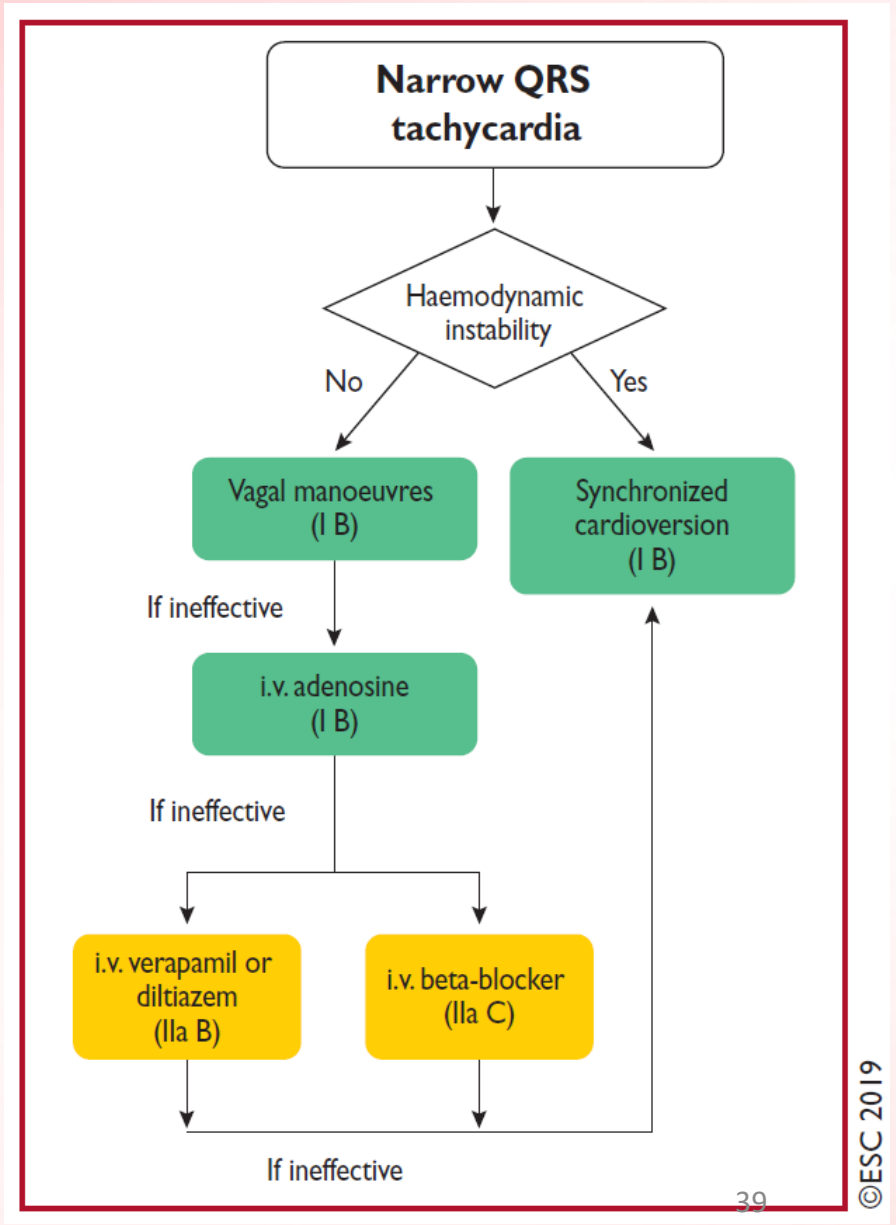
- 6mg → 12mg → 18mg
- Large bore cannula (antecubital / neck)



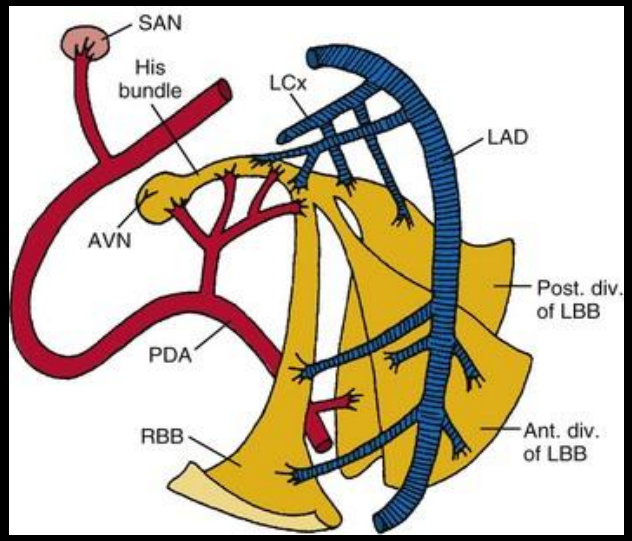


If adenosine fails, what's next?





Bradyarrhythmias & AV Block



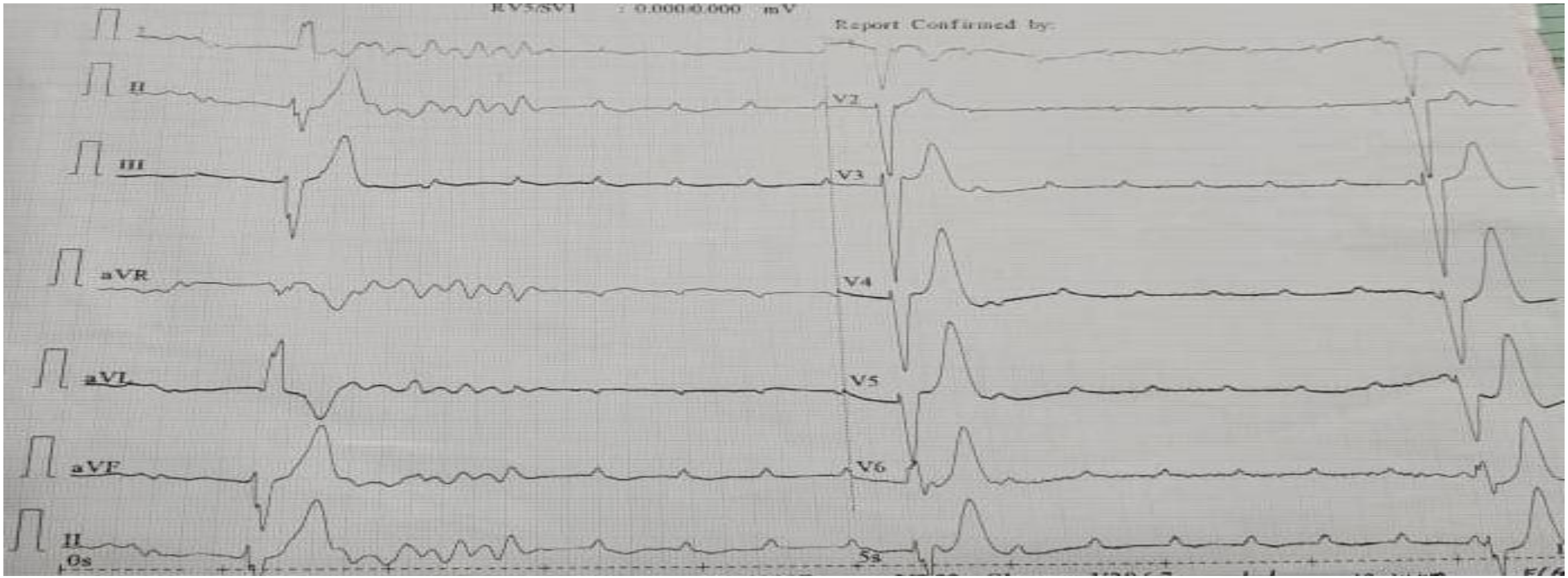
- Inferior MI: often transient.
- Anterior MI: high-grade block → poor prognosis.
- Atropine, temporary pacing → permanent pacemaker if persistent.

PPM

3-5 DAYS ANTERIOR

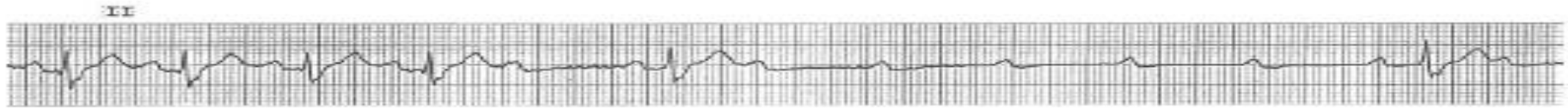
5-7 DAYS INFERIOR

Guidelines: ESC 2023 ACS; AHA 2018 Bradycardia & Conduction Delay


RV5/SV1 : 0.000/0.000 mV
Report Confirmed by:
I
II
III
aVR
aVL
aVF
II
V2
V3
V4
V5
V6
0s
5s

High Grade AV Block

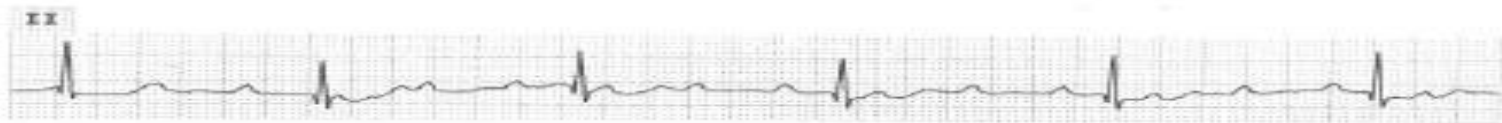
(Initially every atrial impulse is conducted to the ventricles while many consecutive atrial impulses are blocked later.)



3° (Complete) AV Block

- No atrial impulses are conducted to the ventricles
- Escape rhythm is very regular, especially if it is AV junctional rhythm

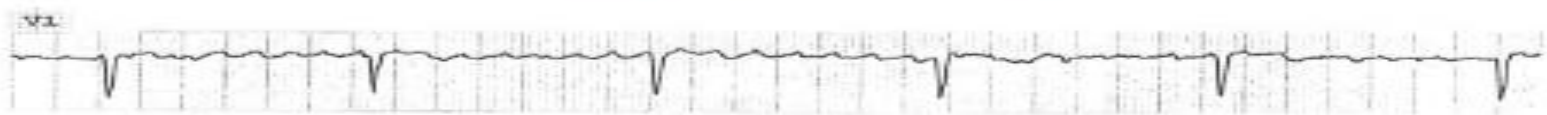
Complete (3°) AV block with AV junctional escape rhythm



Complete (3°) AV block with ventricular escape rhythm

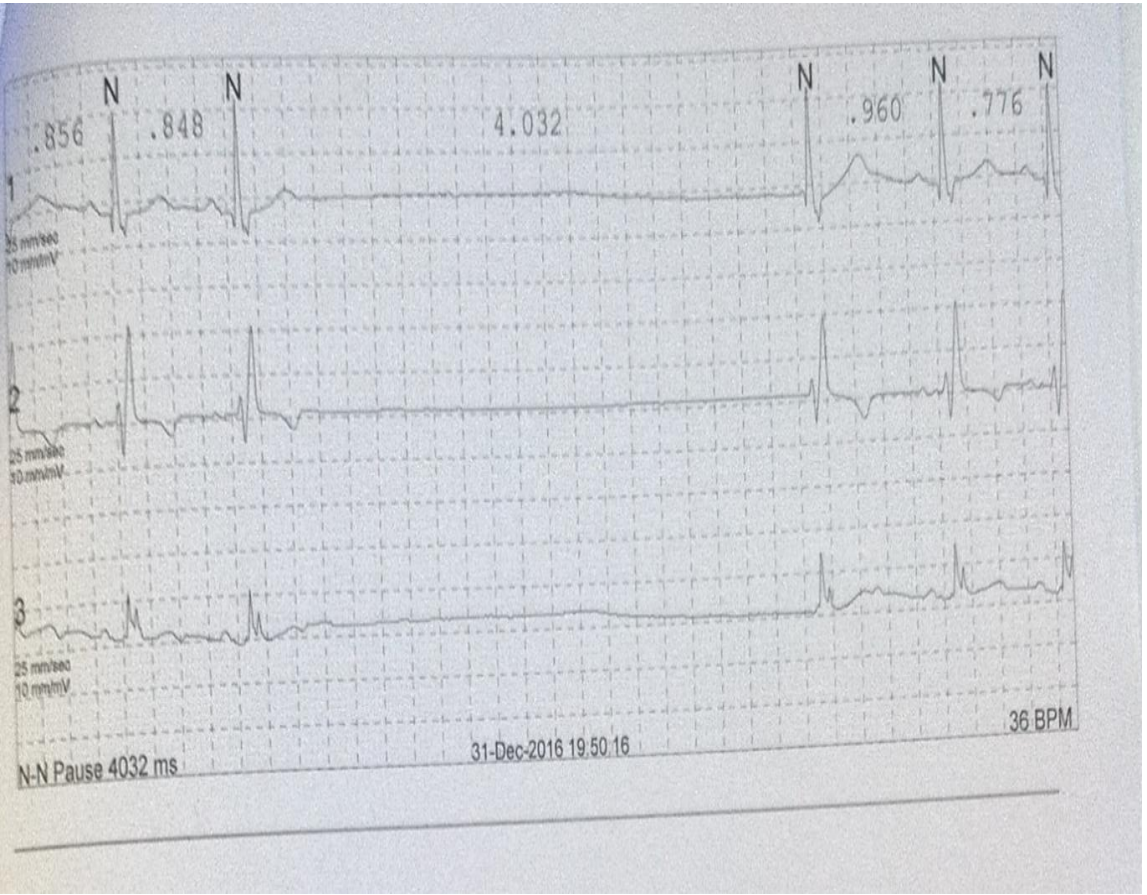


Atrial fibrillation with complete (3°) AV block and AV junctional escape rhythm.
Note the QRSs are narrow and occur regularly.

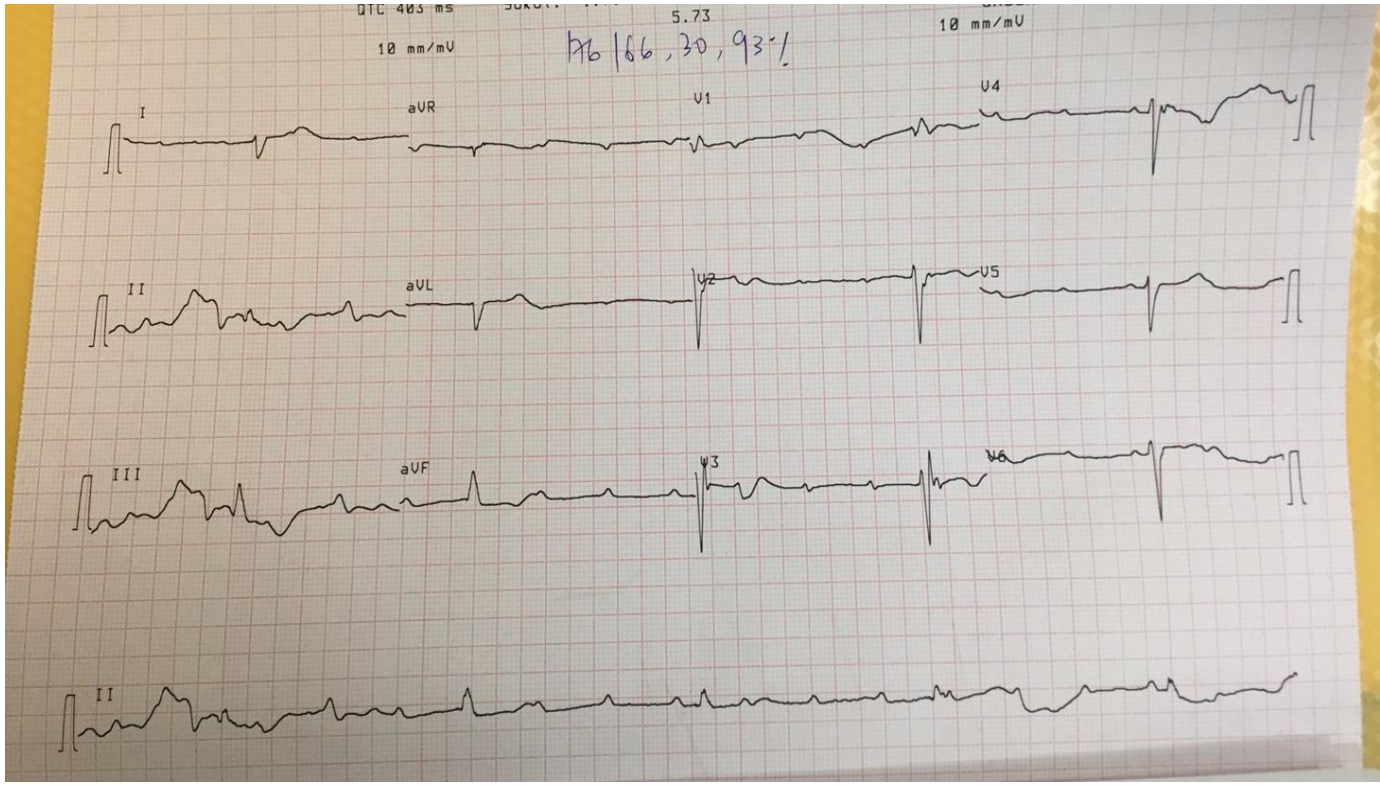


BRADYCARDIA- MASALAH SINUS NODE & AV NODE

SINUS NODE SICK

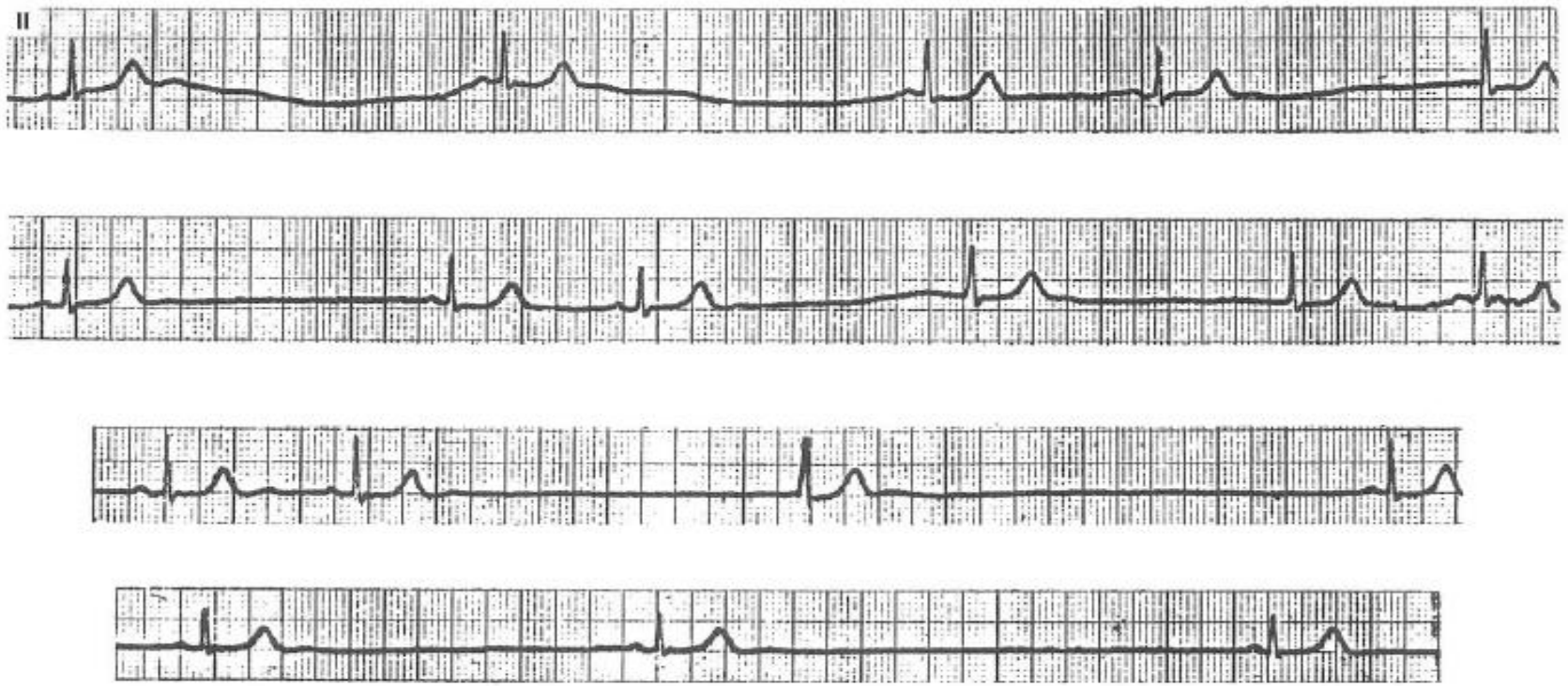


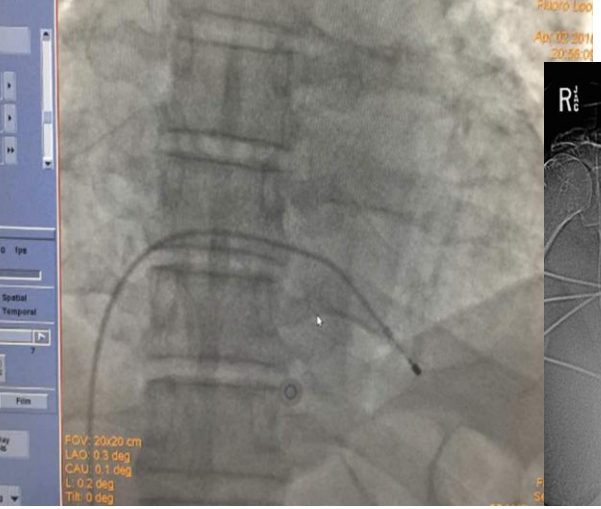
AVN BLOCKED

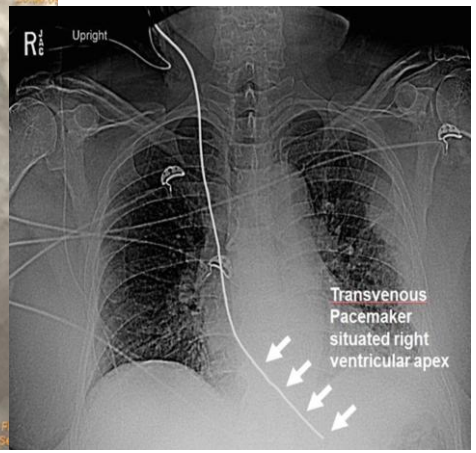


Sick Sinus Syndrome

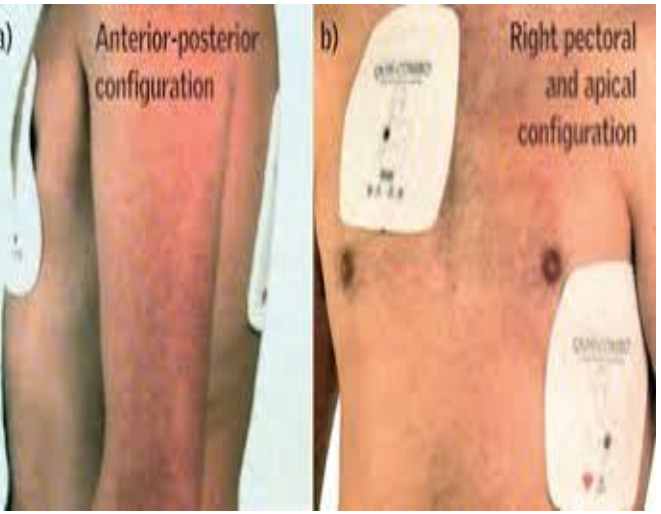
An example of sick sinus syndrome with a marked degree of sinus bradycardia. The occasional AV junctional escape beats also show marked slowing.













Adult Bradycardia Algorithm

Assess appropriateness for clinical condition.
Heart rate typically <50/min if bradyarrhythmia.

Identify and treat underlying cause
- Maintain patent airway; assist breathing as necessary
- Oxygen (if hypoxemic)
- Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
- IV access
- 12-Lead ECG if available; don't delay therapy
- Consider possible hypoxic and toxicologic causes

Persistent bradyarrhythmia causing:
- Hypotension?
- Acutely altered mental status?
- Signs of shock?
- Ischemic chest discomfort?
- Acute heart failure?

No → **Monitor and observe**

Yes →

Atropine
If atropine ineffective:
- Transcutaneous pacing
and/or
- **Dopamine** infusion
or
- **Epinephrine** infusion

Consider:
- Expert consultation
- Transvenous pacing

© 2020 American Heart Association

Doses/Details

Atropine IV dose:
First dose: 1 mg bolus.
Repeat every 3-5 minutes.
Maximum: 3 mg.

Dopamine IV infusion:
Usual infusion rate is 5-20 mcg/kg per minute.
Titrate to patient response; taper slowly.

Epinephrine IV infusion:
2-10 mcg per minute infusion.
Titrate to patient response.

Causes:
- Myocardial ischemia/ infarction
- Drugs/toxicologic (eg, calcium-channel blockers, beta blockers, digoxin)
- Hypoxia
- Electrolyte abnormality (eg, hyperkalemia)

		ACUTE	PERSISTENT/ CHRONIC
VERY FAST	**ASYSTOLE** **Pulseless VT** **VT/VF**	CPR+ IV Adrenaline ACLS- CPR/ DEFIB/ CARDIOVERSION	ICD (2˚48 HOURS, 1˚CONSIDER AFTER 40 DAYS) VT ABLATION FOR SCAR VT
	Af **SVTs**	AMIODARONE LIGNOCAINE/INOTROPES RATE VS RHYTHM ADENOSINE	RATE VS RHYTHM+ OAC STRATEGY EPS+/-RFA
VERY SLOW	**CHB** **SND**	ATROPINE/ INOTROPES TEMPORARY PACING	PPM If due to MI (5-7 DAYS) INFERIOR 3-5 DAYS ANTERIOR

THANK YOU

