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CARDIAC ARRHYTHMIAS IN THE INTENSIVE CARE UNIT

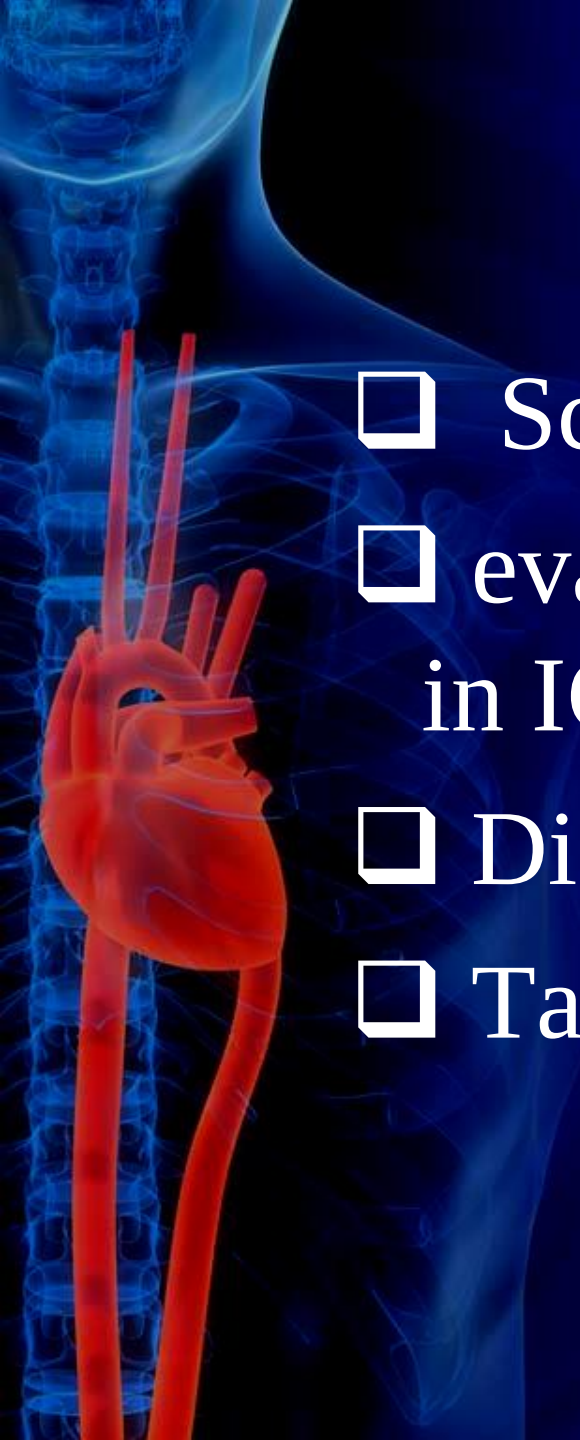
BY

DR: AHMED HOSNY EL-ADAWEY, MD

Lecturer of cardiology

Mansura university

S.R of intervention cardiology &CCU in DAFH



AGENDA

- Scope of the problem.
- evaluation of cardiac arrhythmia in ICU
- Diagnosis &management.
- Take a home massage.

That is what u feel when

Facing

arrhythmia

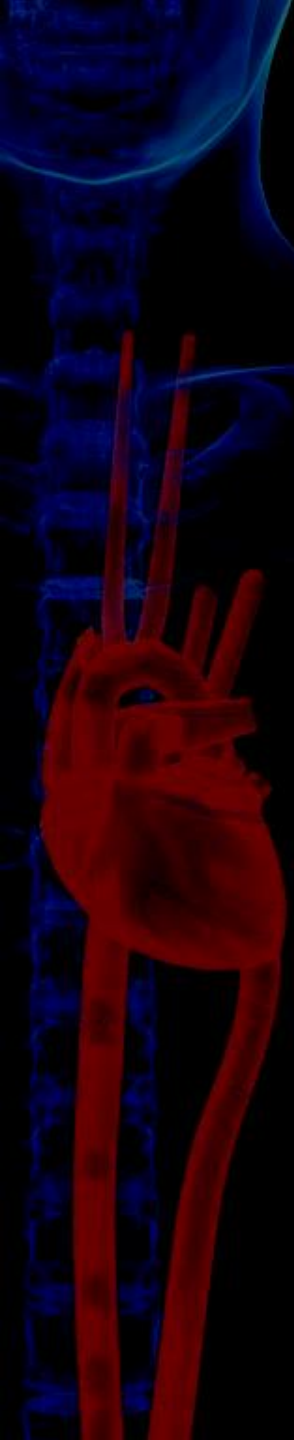
SCOPE OF THE PROBLEM

- Arrhythmias are a common dilemma confronting the intensivist.
- They represent a major source of morbidity, and they lengthen hospital stay.
- Arrhythmias are most likely to occur in patients with structural heart disease.
- The inciting factor for an arrhythmia may be an insult such as
  - hypoxia,
  - infection,
  - cardiac ischemia,
  - catecholamine excess (endogenous or exogenous),
  - or an electrolyte abnormality.

IMPACT OF ARRHYTHMIAS

- The physiological impact of arrhythmias depends on:
  - ventricular response rate
  - duration
  - the underlying cardiac function.
- **Bradyarrhythmias** : may decrease cardiac output due to heart rate alone in patients with relatively fixed stroke volumes, and loss of an atrial kick may cause a dramatic increase in pulmonary pressures in patients with diastolic dysfunction.
- **Tachyarrhythmias** : can decrease diastolic filling and reduce cardiac output, resulting in hypotension, in addition to producing myocardial ischemia.

TACHYARRYTMIAS



EVALUATION OF TACHYARRHYTHMIAS

- **The first step :** is to assess hemodynamic stability.
- If hemodynamics are compromised >> cardioversion should be performed unless pharmacological treatment is immediately successful.

  (However, before proceeding with cardioversion, one should consider whether the arrhythmia is in fact the basis for the deterioration in hemodynamics).
- **The next step :** is to determine whether the arrhythmia is supraventricular or ventricular in origin.

  (one examines QRS width >> A narrow QRS complex (<0.12 sec) indicates a SVT).

GOLDEN RULES

- One should try not to rely solely on a rhythm strip from one monitor lead for diagnosis; there can be variability in QRS width depending on which lead is examined.
- A 12-lead electrocardiogram (ECG) is more useful. Also, review of a previous ECG is often useful; for example,

  1 -to identify preexisting bundle branch block or QTc interval prolongation.

  2 -Marked left axis deviation (60 to 120 degrees) may indicate a ventricular origin of the arrhythmia.

- *It is noteworthy that ST segment depression during SVT lacks specificity in predicting ischemia*, (In one series of 100 patients with SVT, associated ST segment deviation was only 51% specific (with a positive predictive value of only 6%) for significant angiographic coronary artery disease or scintigraphic evidence of ischemia).

- **Carotid sinus massage** and other maneuvers that increase vagal tone slow AV conduction time and increase refractoriness, and this can aid in the diagnosis through demonstration of p waves or interruption of AVNRT or AV reentrant tachycardia (AVRT).

❑ **Adenosine** : can also be used for this purpose:

- Adenosine is given as a rapid intravenous (IV) bolus of 6 mg, and a second dose of 12 mg can be given 1 to 2 minutes later.

-The effects are more pronounced when given through a central venous line, in which case the dosage is then usually halved.

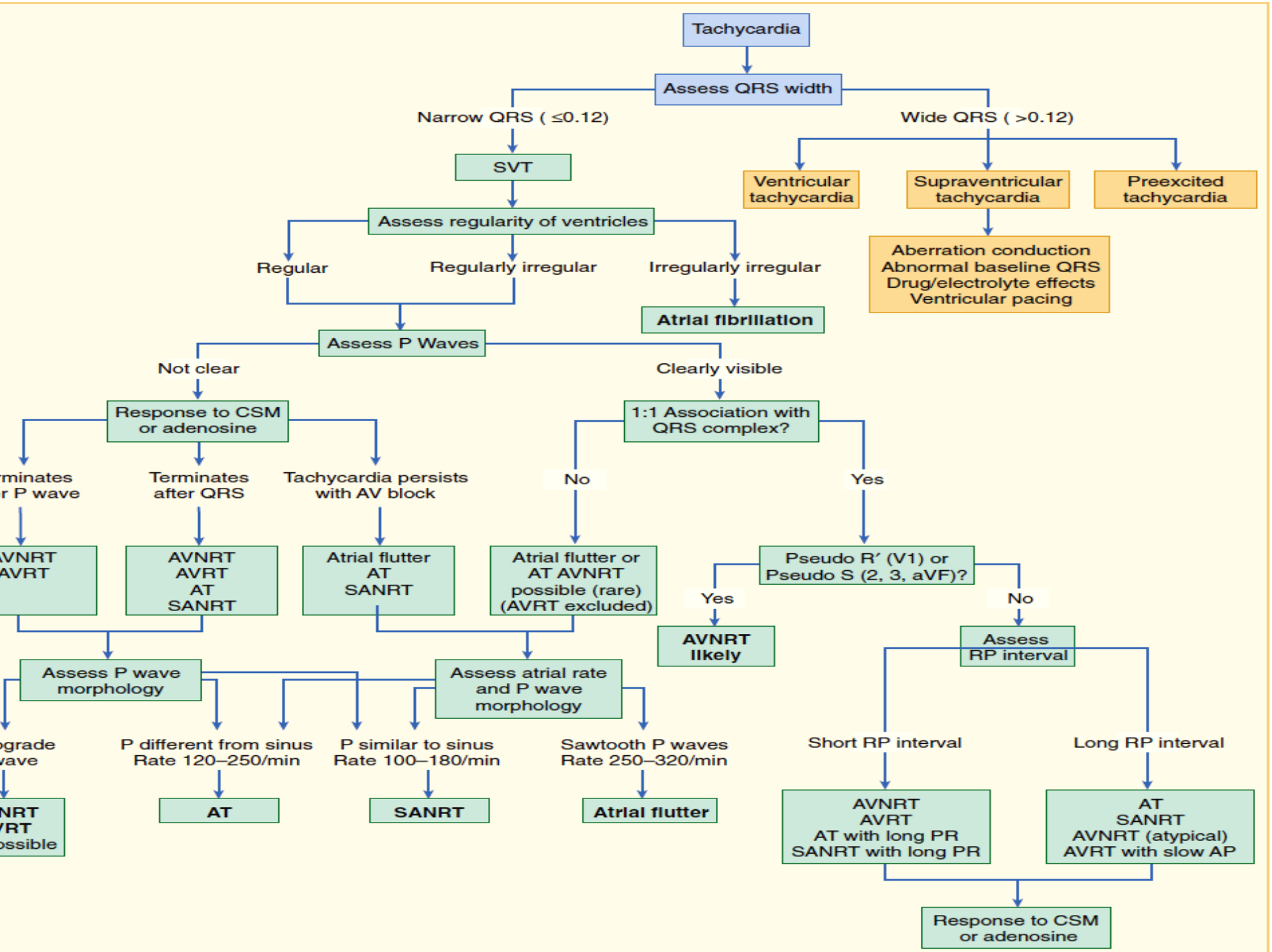
The half-life of adenosine is only 6 to 10 seconds.

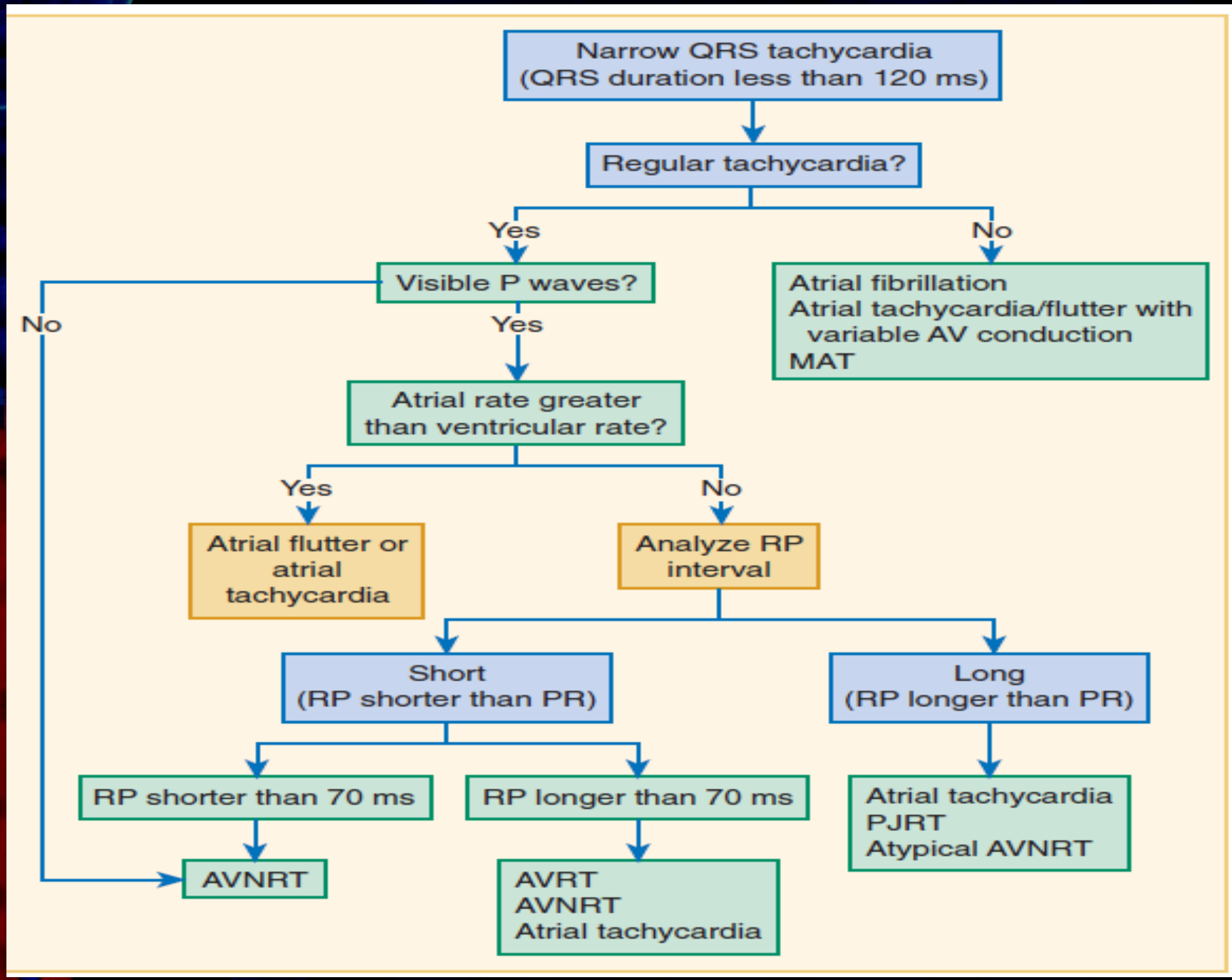
-Severe bronchospasm or wheezing can result from its use.

-Adenosine can be proarrhythmic, most commonly the induction of AF (2.7%),and there have been reports of asystole, VT, and ventricular fibrillation (VF) following its administration.

Table 1 Responses to Vagal Maneuvers or Adenosine

Arrhythmia	Response to Vagal Maneuvers/Adenosine
Sinus tachycardia	Gradual slowing with resumption of the tachycardia
Atrioventricular nodal reentrant tachycardia	Abrupt termination or only very transient slowing
Atrial fibrillation/flutter	Increased atrioventricular block briefly with slowed ventricular response rate
Multifocal atrial tachycardia	Increased atrioventricular block briefly with slowed ventricular response rate
Ventricular tachycardia	Usually no response


Tachycardia
Assess QRS width
Narrow QRS (≤0.12)
Wide QRS (>0.12)
SVT
Ventricular tachycardia
Supraventricular tachycardia
Preexcited tachycardia
Assess regularity of ventricles
Aberration conduction
Abnormal baseline QRS
Drug/electrolyte effects
Ventricular pacing
Regular
Regularly irregular
Irregularly irregular
Atrial fibrillation
Assess P Waves
Not clear
Clearly visible
Response to CSM or adenosine
1:1 Association with QRS complex?
rminates r P wave
Terminates after QRS
Tachycardia persists with AV block
No
Yes
VNRT AVRT
AVNRT AVRT AT SANRT
Atrial flutter AT SANRT
Atrial flutter or AT AVNRT possible (rare) (AVRT excluded)
Pseudo R′ (V1) or Pseudo S (2, 3, aVF)?
Yes
No
AVNRT likely
Assess P wave morphology
Assess atrial rate and P wave morphology
Assess RP interval
ograde wave
P different from sinus Rate 120–250/min
P similar to sinus Rate 100–180/min
Sawtooth P waves Rate 250–320/min
Short RP interval
Long RP interval
NRT VRT ossible
AT
SANRT
Atrial flutter
AVNRT AVRT AT with long PR SANRT with long PR
AT SANRT AVNRT (atypical) AVRT with slow AP
Response to CSM or adenosine


Narrow QRS tachycardia
(QRS duration less than 120 ms)
Regular tachycardia?
Yes
No
Visible P waves?
Atrial fibrillation
Atrial tachycardia/flutter with variable AV conduction
MAT
No
Yes
Atrial rate greater than ventricular rate?
Yes
No
Atrial flutter or atrial tachycardia
Analyze RP interval
Short
(RP shorter than PR)
Long
(RP longer than PR)
RP shorter than 70 ms
RP longer than 70 ms
Atrial tachycardia
PJRT
Atypical AVNRT
AVNRT
AVRT
AVNRT
Atrial tachycardia

- **Narrow complex tachycardias :**
  - AF,
  - Sinus tachycardia
  - AVNRT
  - AVRT & WPW
  - Atrial flutter
  - Atrial tachycardia.
- **Wide QRS tachycardias :**
  - VT
  - SVT with preexisting bundle branch block
  - aberrant ventricular conduction
  - SVT from AV reentry using an antegrade accessory pathway (WPW) = ACMT

NARROW COMPLEX TACHYCARDIA

- **Regular narrow complex SVTs** :
  - ✓ sinus tachycardia
  - ✓ AVNRT
  - ✓ AVRT
  - ✓ Ectopic
  - ✓ atrial tachycardia
  - ✓ atrial flutter.
- **Irregular narrow complex SVTs** :
  - ✓ AF
  - ✓ multifocal atrial tachycardia (MAT)
  - ✓ atrial flutter with variable block,
  - ✓ sinus tachycardia with frequent premature atrial complexes.

➢**The p wave morphology >>** can suggest the origin of the atrial impulse.

- ❑ The p wave should be upright in lead II with a normal sinus mechanism.
- ❑ If inverted, this is suggestive of AVRT, AVNRT, or ectopic atrial tachycardia.
- ❑ P waves may be absent or difficult to discern in the setting of tachycardia.

➢**The RP interval>> should be assessed on the 12-lead ECG:**

- ❑ short RP interval (RP shorter than PR, and less than 70 msec) suggesting AVNRT,
- ❑a long RP interval most likely indicating AVRT via a slowly conducting accessory pathway.

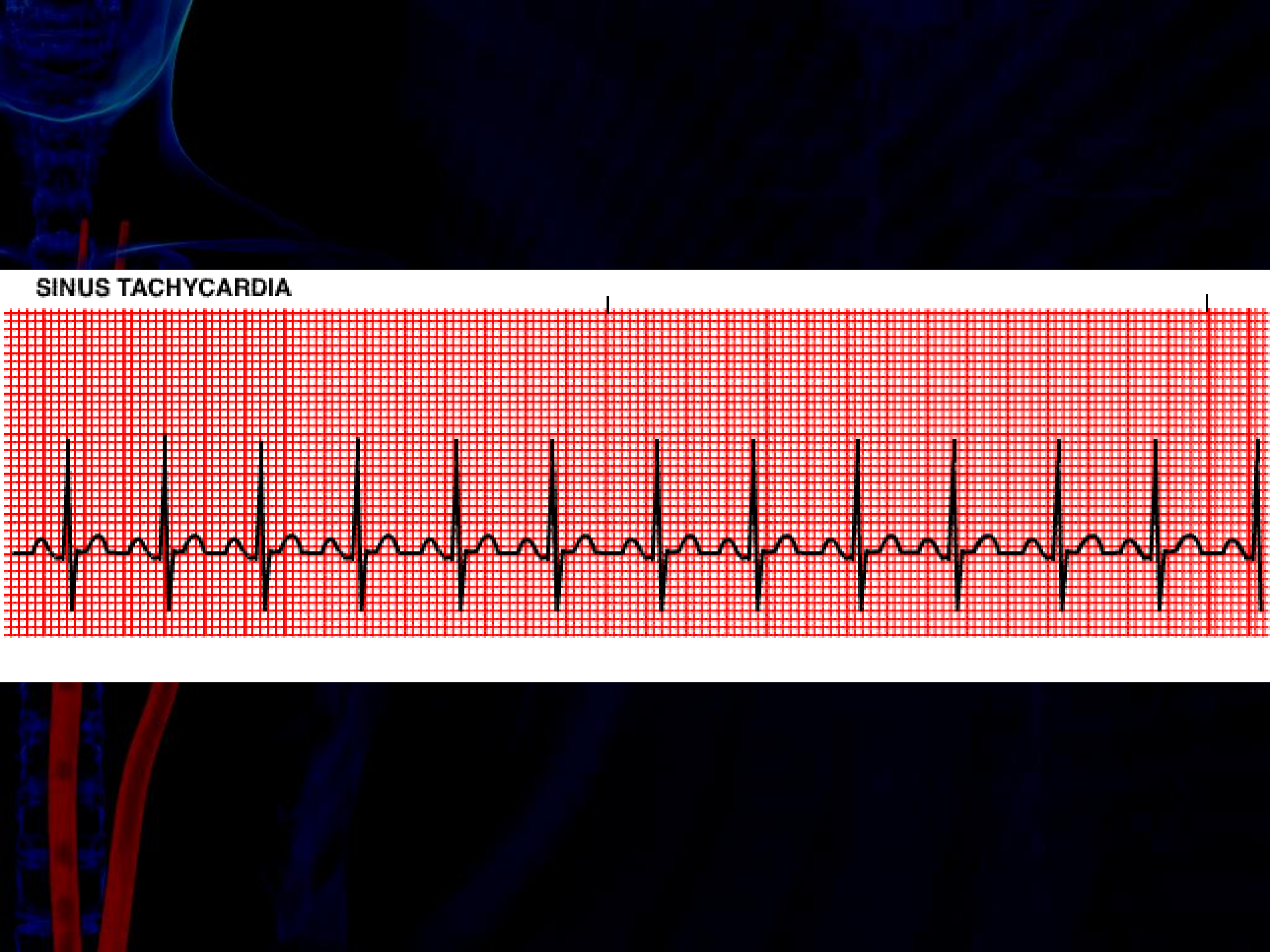
(A heart rate of 150 bpm should raise the suspicion of atrial flutter with 2:1 conduction)

Regular Rhythms

SINUS TACHYCARDIA

- **Incidence:** Sinus tachycardia often occurs as a response to a sympathetic stimulus *(hypoxia, vasopressors, inotropes, pain, dehydration, hyperthyroidism, etc.).*
- Evaluation: The first step is to review patient medications, including infusions, to exclude an iatrogenic etiology of the tachyarrhythmia.
- **Treatment :** correct the underlying cause.

  If ischemia is the cause and treatment is warranted, b-blockers are the first treatment option.
- However, it is worth considering that the sinus tachycardia may be an appropriate hemodynamic response to hypotension, hypovolemia, or low cardiac output; if this is the case, overzealous use of b-blockers can reduce cardiac output, with potentially disastrous consequences.


SINUS TACHYCARDIA

AVNRT

- **Incidence :** AVNRT typically occurs at a heart rate of 140 to 180 bpm.
- It is more prevalent in females and is not usually associated with structural heart disease.
- **Evaluation:** AVNRT involves dual AV nodal pathways, usually with slow conduction antegrade and retrograde conduction via a transiently refractory second pathway .
- **treatment :** is to block AV conduction.
- Acute treatment includes vagal maneuvers and IV adenosine.
- Long-term preventative therapy includes medications that suppress the initiating premature atrial contractions (b-blockers) or slow AV conduction (nondihydropyridine calcium-channel blockers, b-blockers, and digoxin), or
- catheter ablation of one of the pathways.

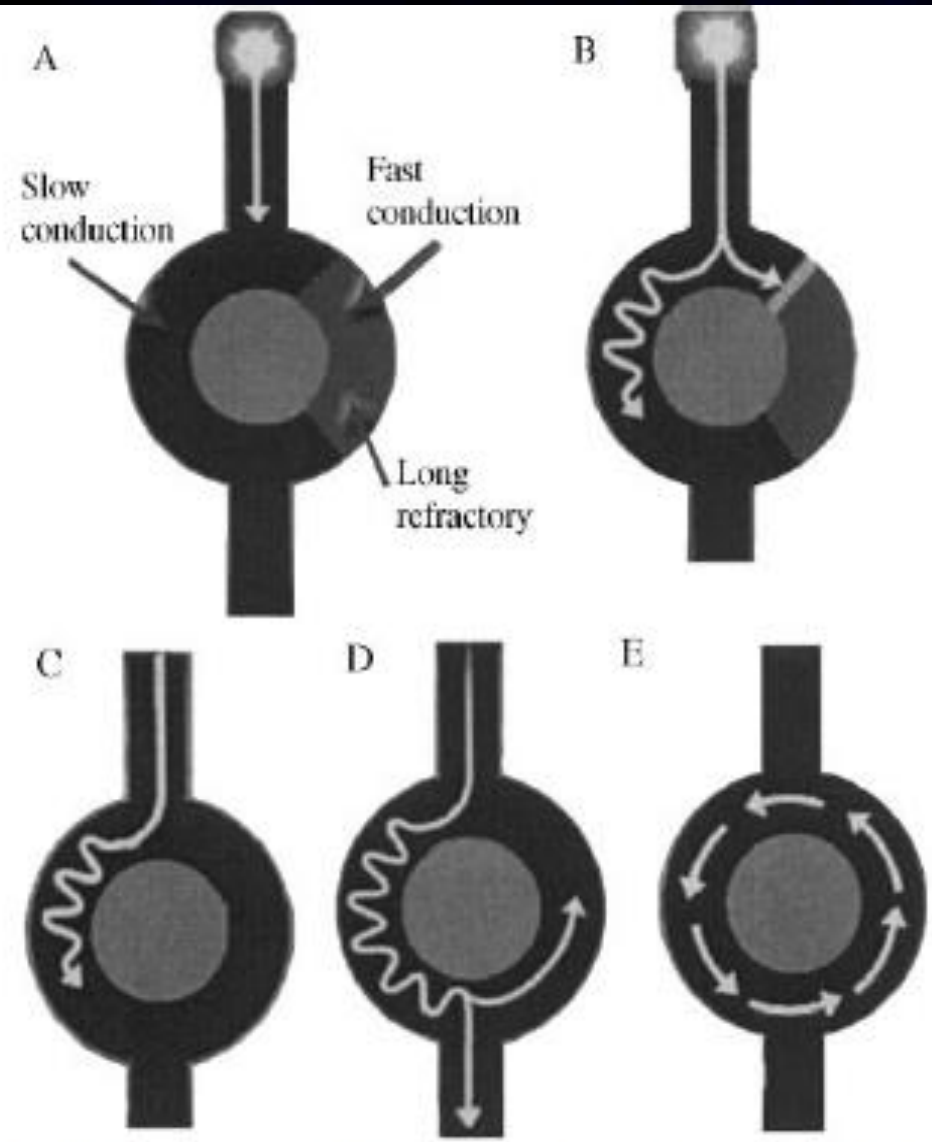
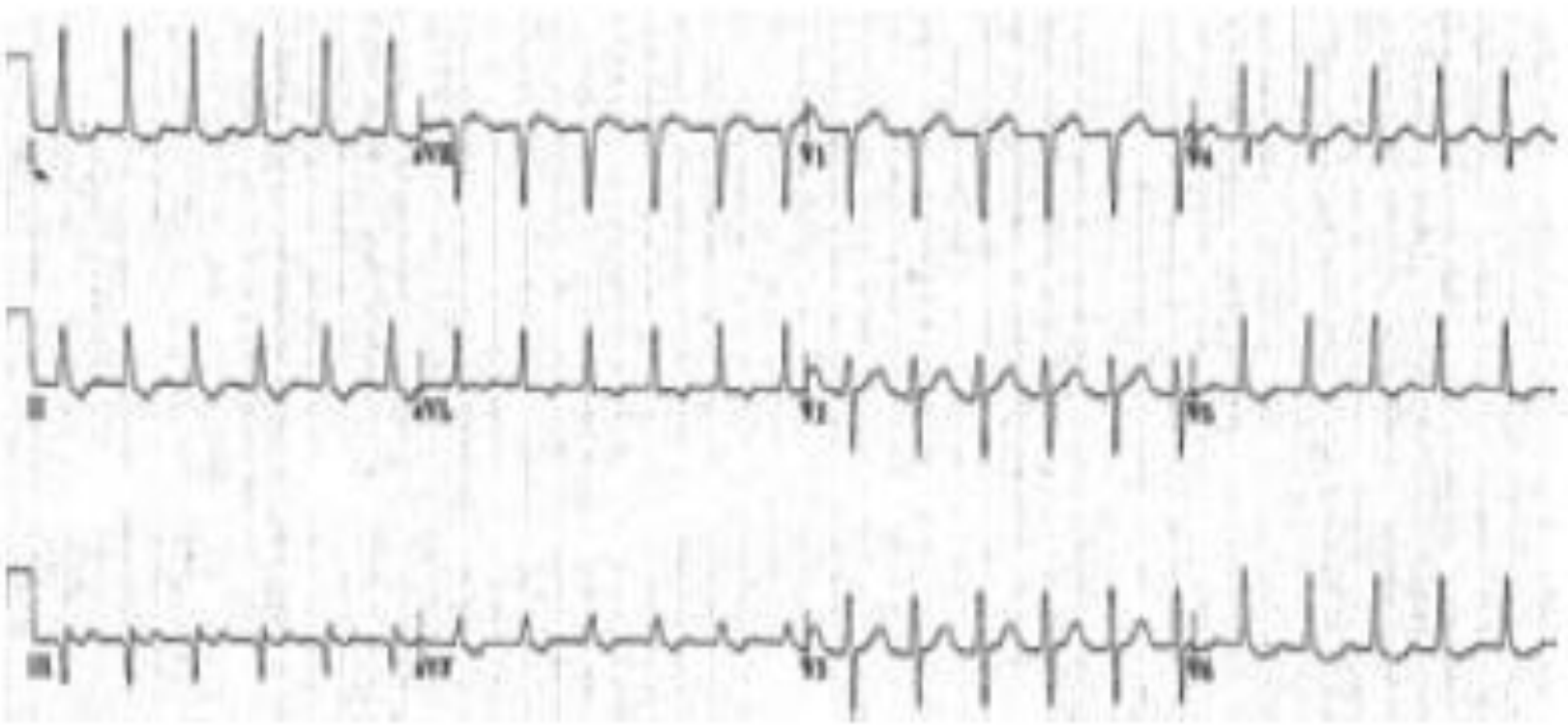
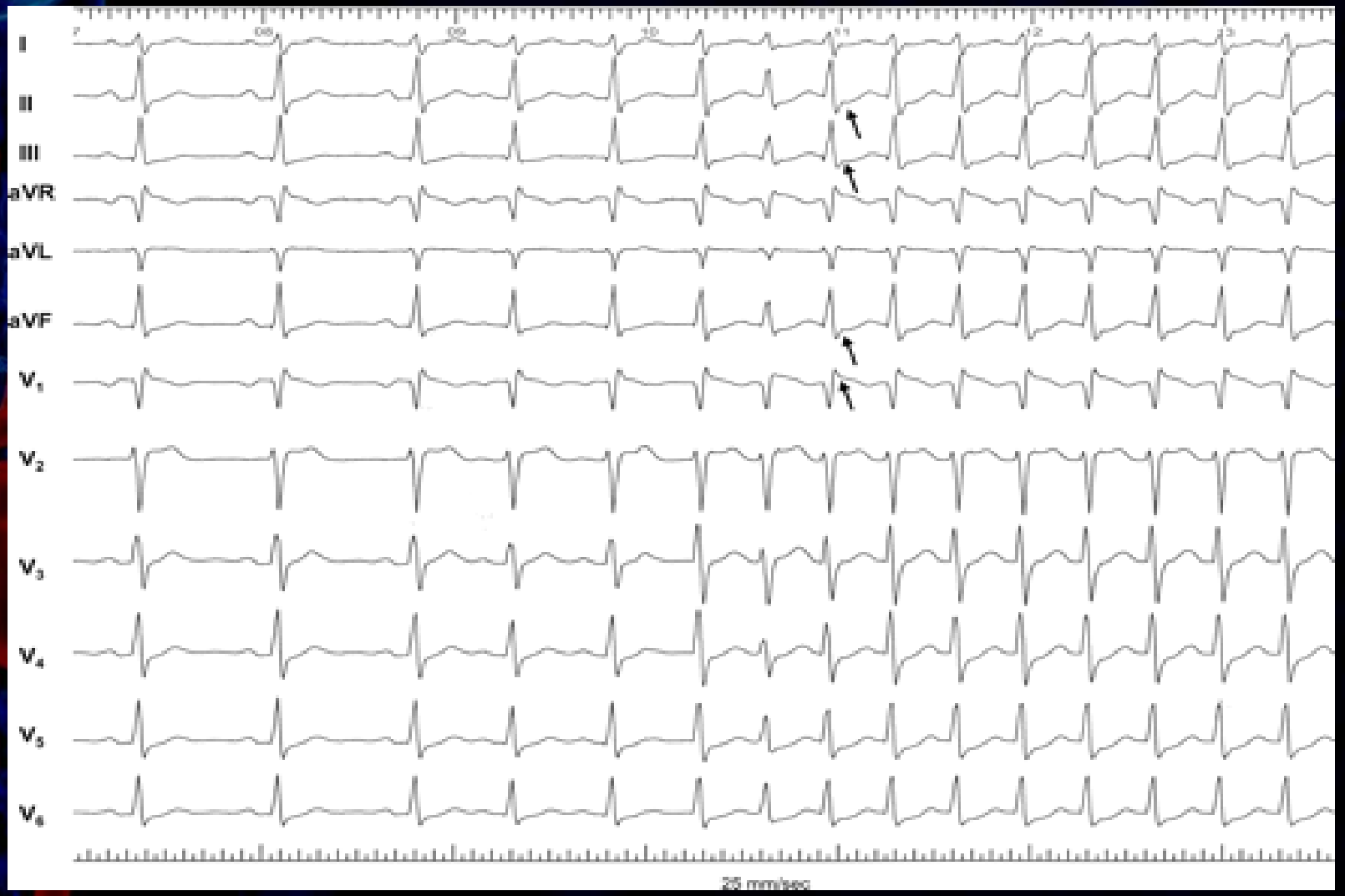


Figure 1 (A) Atrioventricular (AV) node demonstrating dual pathways: slow (α) pathway with short refractory period and fast (β) pathway with long refractory period. (B) Premature impulse conducts down slow pathway while fast pathway is still refractory to conduction. (C) As impulse conducts down slow pathway, the fast pathway recovers. (D) Impulse goes up fast pathway as it conducts to the ventricle. (E) Impulse reenters cycle in AV node completing reentrant circuit.

Figure 4. AVNRT




I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
25 mm/sec

ATRIAL FLUTTER

- Atrial flutter is a macroreentrant arrhythmia identified by flutter waves, often best seen in the inferior leads, at 250 to 350 bpm.
- Patients often present with 2:1 AV conduction with a ventricular rate of 150 bpm, although the AV conduction ratio can change abruptly.
- **Treatment :**

❑ Acute treatment consists of AV-nodal-blocking drugs for rate control.

- If the patient becomes clinically unstable>>DC-synchronized.
- cardioversion with 50 J is usually sufficient, with success rate of 95 to 100%.

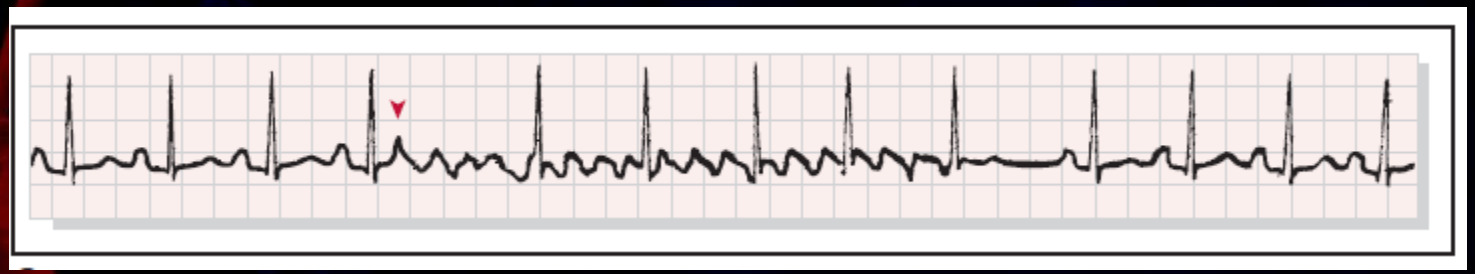
❑ IV ibutilide has an efficacy rate of 76% for conversion to sinus rhythm in clinical trials , but prolongs the QT interval and can provoke sustained polymorphic VT in 1 to 2% of cases.

- Ibutilide should not be used in patients with a prolonged QTc interval (greater than 420 msec), or in those with underlying sinus node disease.

- Other antiarrhythmics such as sotalol, procainamide, and flecainide have demonstrated less efficacy for acute conversion.
- If a temporary or permanent pacemaker is in place, atrial overdrive (burst) pacing can sometimes restore sinus rhythm via overdrive suppression.

- Long-term treatment of the ventricular rate in atrial flutter usually consists of diltiazem, verapamil, b blockers, or digoxin.

- Class IC drugs (flecainide) are very effective in preventing atrial flutter, but by slowing the atrial rate, they have the potential to cause 1:1 AV conduction, and should always be combined with AV nodal- blocking agents.



Irregular Rhythms

ATRIAL FIBRILLATION

- **Incidence:** AF is the most common narrow complex tachyarrhythmia in the ICU (second to VT overall).
- **prevalence :** of AF in the general population increases exponentially with age, from 0.9% at age 40 to 5.9% in those over age 65.
- **The most important risk factors** for development of AF in the general population are structural heart disease (70%), hypertension (50%), valvular heart disease (34%), and left ventricular hypertrophy.
- **AF should be approached in the following manner:**

- find the cause, fix the cause, control the rate, consider rhythm control, and consider anticoagulation.
- Pharmacological agents for acute rate control include b-blockers, nondihydropyridine calcium channel blockers, and digoxin.

❑**Beta-blockers** provide more effective rate control than calcium channel blockers at rest and during exercise. (Both oral and IV formulations are available) .

• The most often used IV medication is metoprolol given at 2.5 to 5.0 mg IV over 1 to 2 minutes every 5 to 10 minutes for a total of 15 mg as blood pressure tolerates.

• Esmolol, 0.5 mg/kg bolus, then 0.05 mg/kg/min infusion, is an alternative with a more rapid onset and offset, which can be useful in unstable patients..

❑**Nondihydropyridine CCB:** (diltiazem and verapamil) are also effective AV nodal blockers.

• Verapamil may have more negative inotropic properties than diltiazem and thus may induce hypotension in patients with left ventricular dysfunction and borderline blood pressure.

• Diltiazem is available in IV form and is commonly used as a continuous infusion at a rate of 5 to 15 mg per hour. Up to 93% of patients will maintain a ventricular response rate <100 bpm during a 24-hour infusion.

❑**Digoxin :** controls ventricular response through a centrally mediated vagal mechanism and by direct action on the AV node.

- It controls resting heart rates in patients who do not have increased catecholamine levels but is less effective in the ICU.
- IV digoxin begins to slow the heart rate in 30 minutes.

❑**Cardioversion** of a patient with AF carries a stroke risk from 1.1% if anticoagulated for 3 weeks to 7% if not anticoagulated, even if AF duration is less than 1 week.

Due to delay between resumption of organized atrial electrical activity and of organized mechanical contraction, there can be delay between cardioversion and embolic events ranging from 6 hours to 7 days.

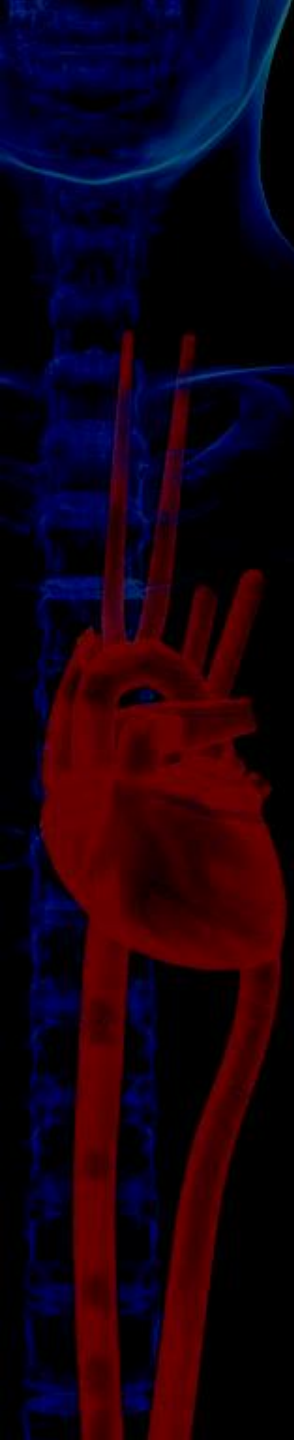
Post-cardiac surgery AF

- occurs in 25 to 40% of patients, with peak incidence on day 2.
- Use of b blockers, amiodarone, sotalol and biatrial overdrive pacing to prevent postoperative AF has been studied in clinical trials.
- Preoperative administration of sotalol and amiodarone is equally effective, but side effects of sotalol limit its use in comparison to amiodarone or b blockers.
- Standard treatment for postoperative AF is to establish rate control, initially with IV and then with oral AV nodal blocking medications.

- There are numerous risk factors for postoperative AF, with *advanced age* being the most important.

- AF often runs a self-correcting course in this setting, with resumption of sinus rhythm in more than 90% of patients by 6 to 8 weeks after surgery, and so cardioversion is not always necessary.

- Immediate cardioversion should be performed in patients with recent onset AF accompanied by symptoms or signs of hemodynamic instability resulting in angina, myocardial ischemia, shock, or pulmonary edema without waiting for prior anticoagulation.



ANTICOAGULATION

- Anticoagulation with IV heparin should be considered if AF persists for greater than 48 hours.
- The stroke risk in unanticoagulated patients taken as a whole is 2% per year (0.05% per day), but individual factors modulate that risk.
- The risk factors for stroke (CHA2D VASc) are heart failure, hypertension, age >75 years, diabetes, prior history of transient ischemic attack (TIA) or stroke, and female gender.

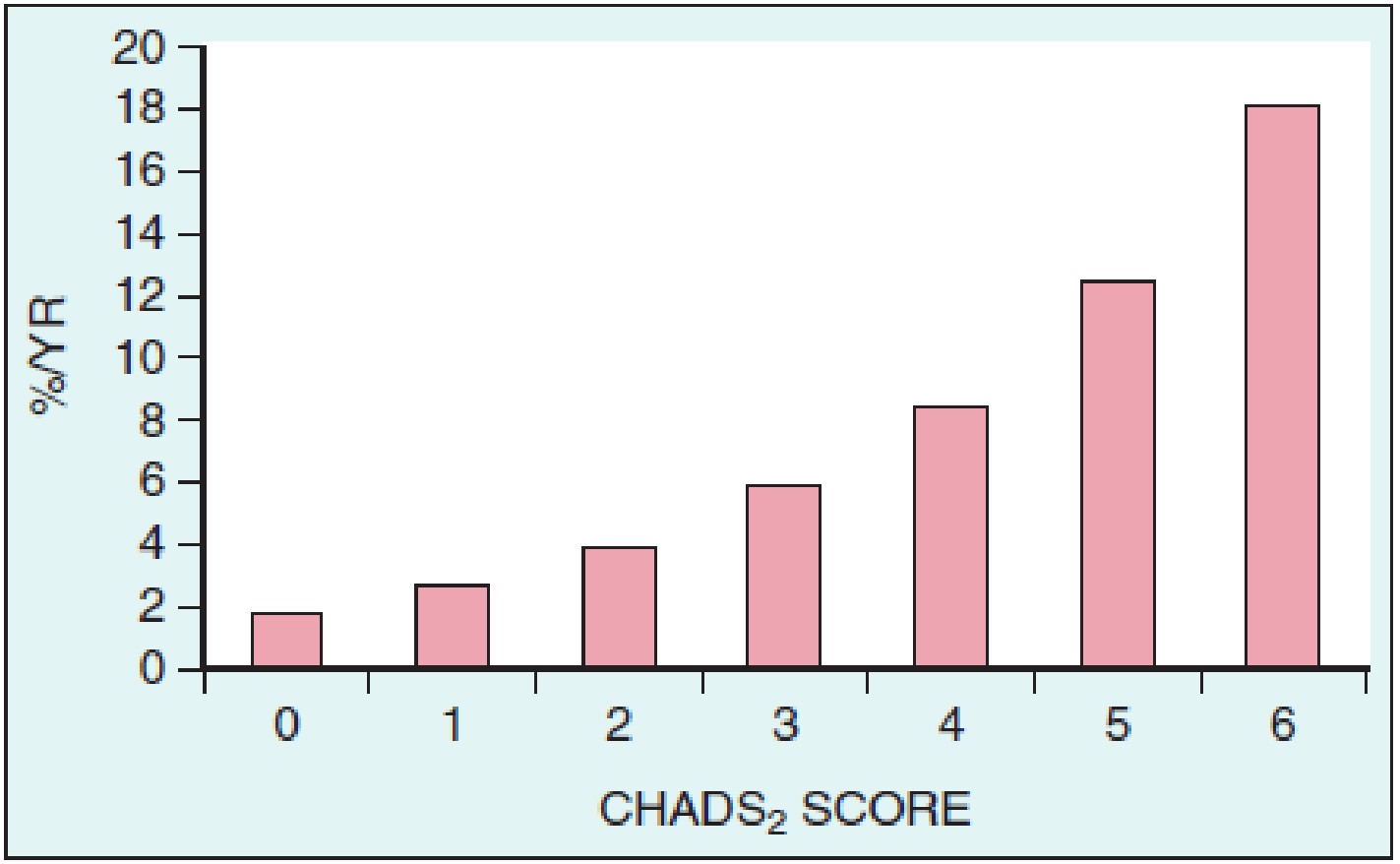
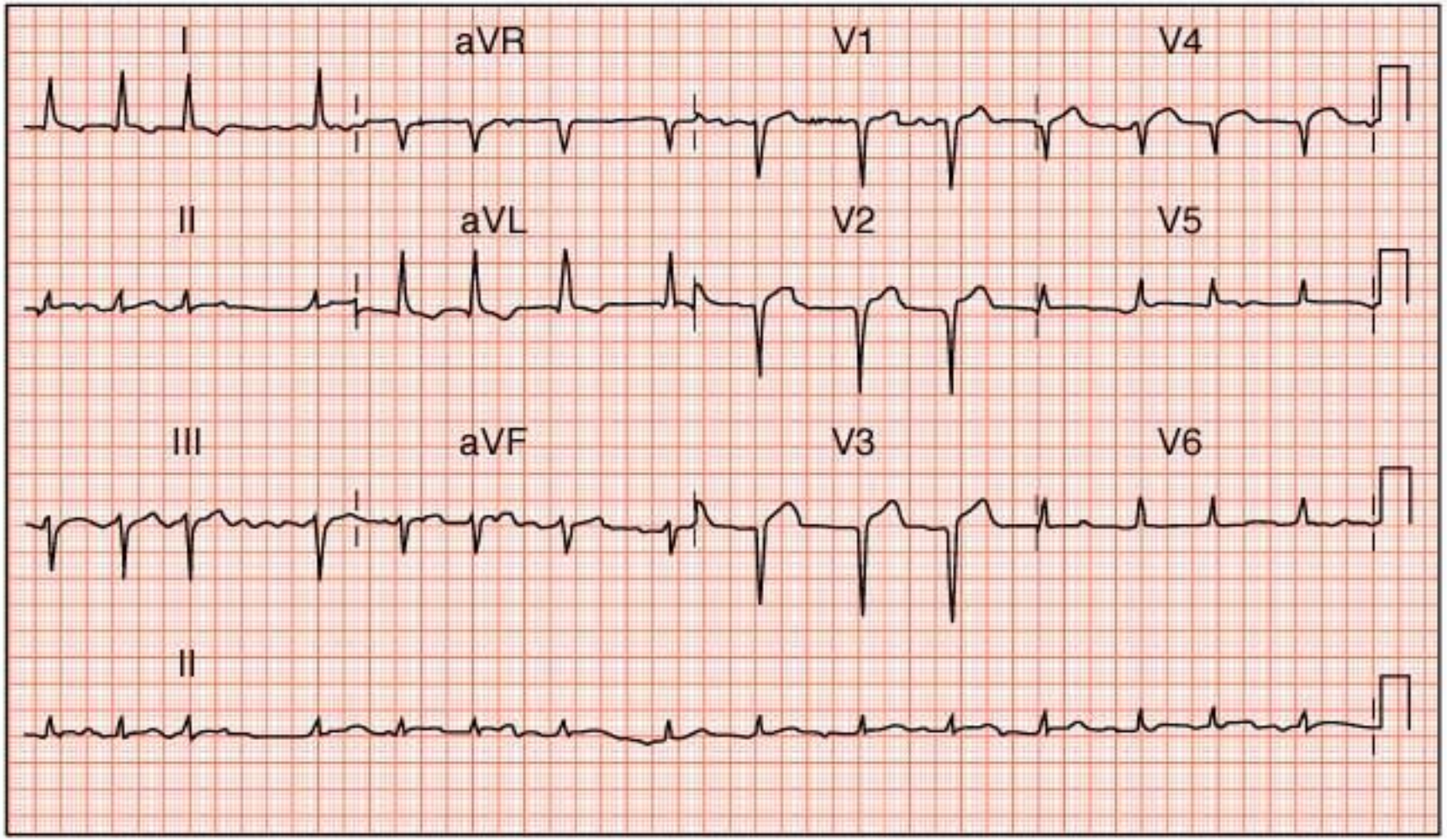
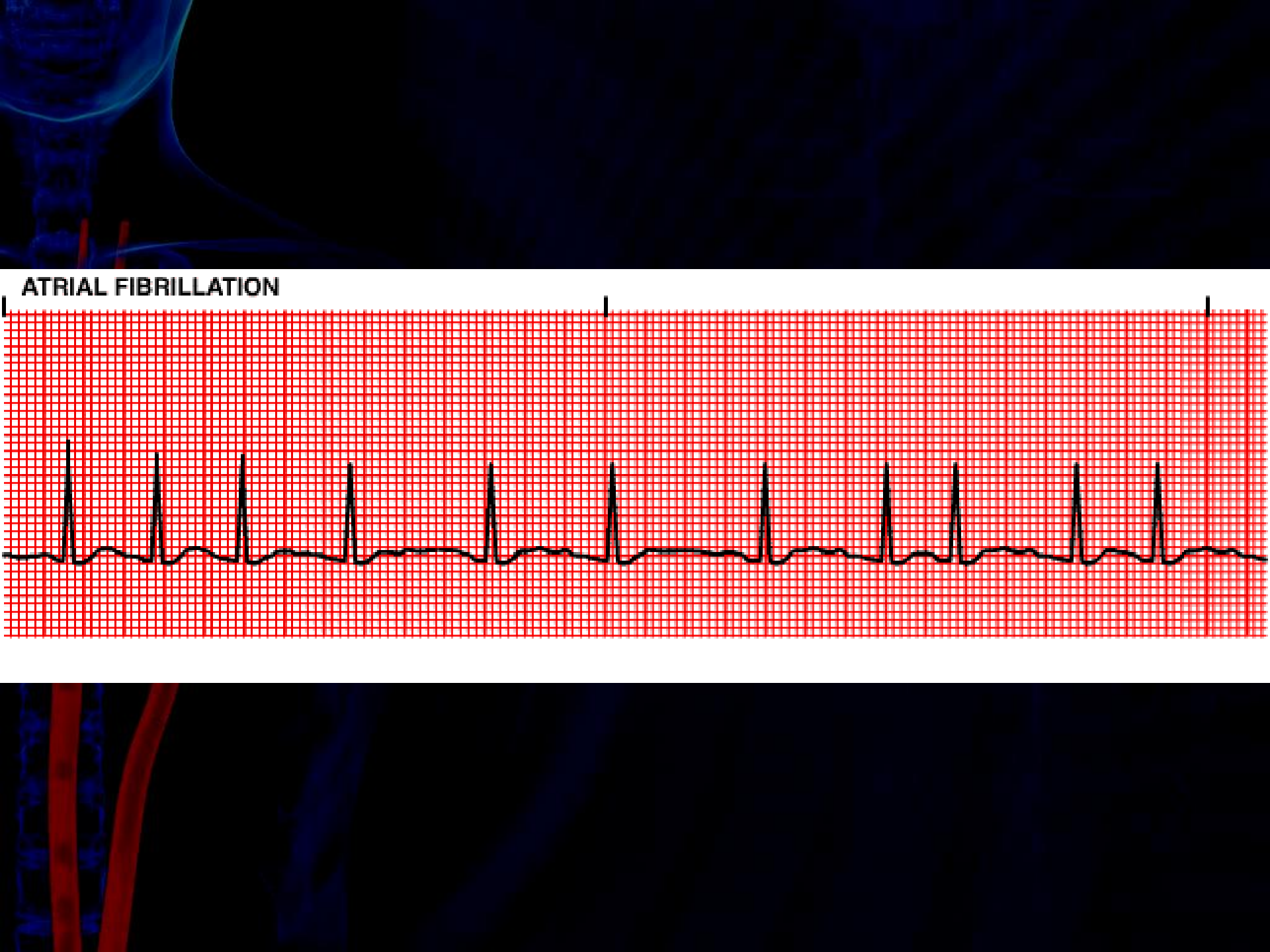


FIGURE 40-5 The annual risk of stroke based on the $CHADS_2$ score. *(Based on data in Table 9 of reference 7.)*

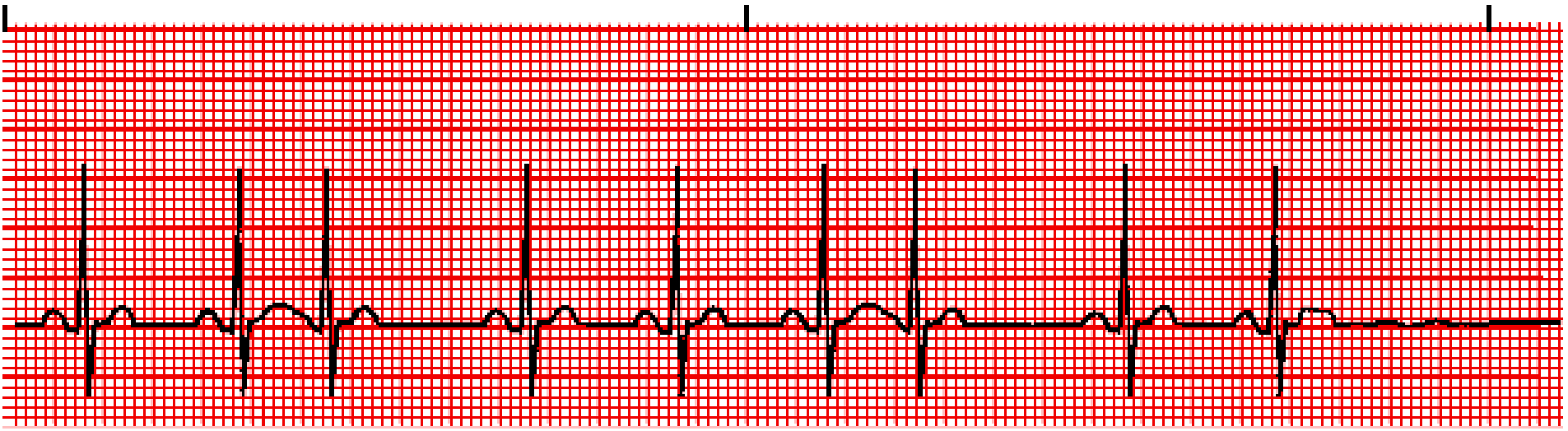
TABLE 40G-2 ACC/AHA Recommendations for Prevention of Thromboembolism in Atrial Fibrillation

CLASS	INDICATION	LEVEL OF EVIDENCE
Class I (indicated)	Antithrombotic therapy to prevent thromboembolism is recommended for all patients with AF, except those with lone AF or contraindications	A
	The selection of the antithrombotic agent should be based on the absolute risks of stroke and bleeding and the relative risk and benefit for a given patient	A
	For patients without mechanical heart valves at high risk of stroke, chronic oral anticoagulant therapy with a vitamin K antagonist is recommended in a dose adjusted to achieve the target intensity international normalized ratio (INR) of 2.0 to 3.0 unless contraindicated. Factors associated with highest risk for stroke in patients with AF are prior thromboembolism (stroke, transient ischemic attack, or systemic embolism) and rheumatic mitral stenosis.	A
	Anticoagulation with a vitamin K antagonist is recommended for patients with more than one moderate risk factor. Such factors include age ≥75 years, hypertension, heart failure, impaired left ventricular systolic function (ejection fraction ≤35% or fractional shortening <25%), and diabetes mellitus.	A
	INR should be determined at least weekly during initiation of therapy and monthly when anticoagulation is stable	A
	Aspirin, 81 to 325 mg daily, is recommended as an alternative to vitamin K antagonists in low-risk patients or in those with contraindications to oral anticoagulation	A
	For patients with AF who have mechanical heart valves, the target intensity of anticoagulation should be based on the type of prosthesis, maintaining an INR of at least 2.5	B
	Antithrombotic therapy is recommended for patients with atrial flutter as for those with AF	C


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


ATRIAL FIBRILLATION

PREMATURE ATRIAL CONTRACTION



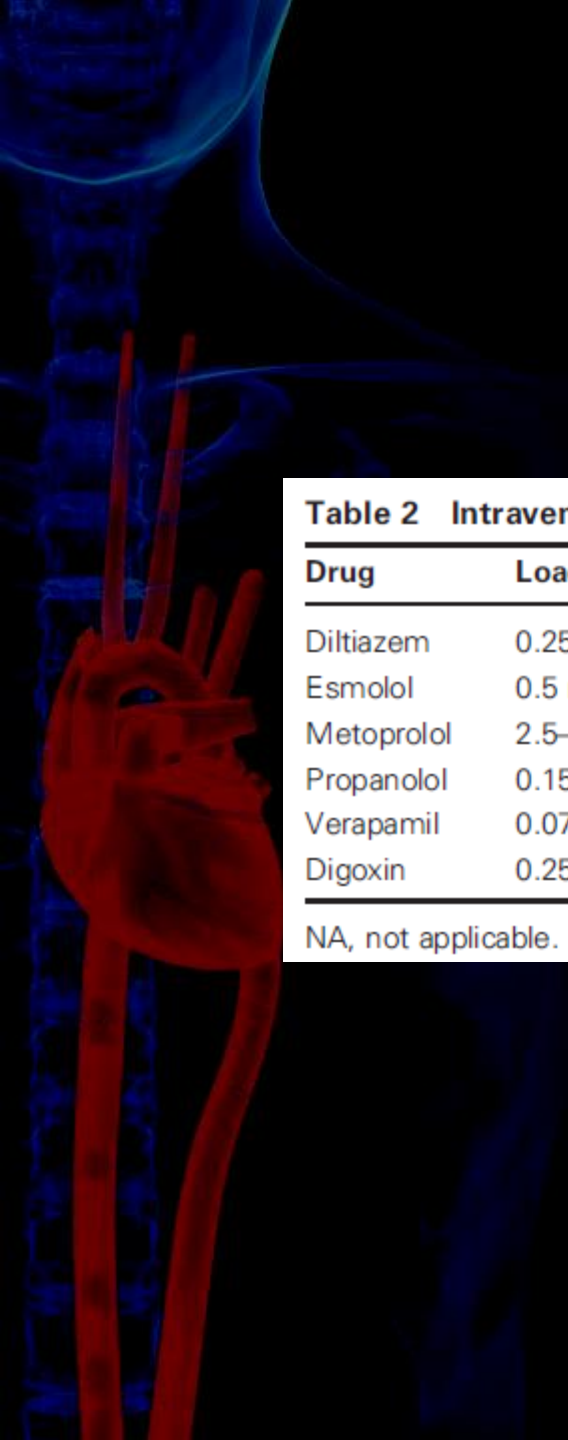


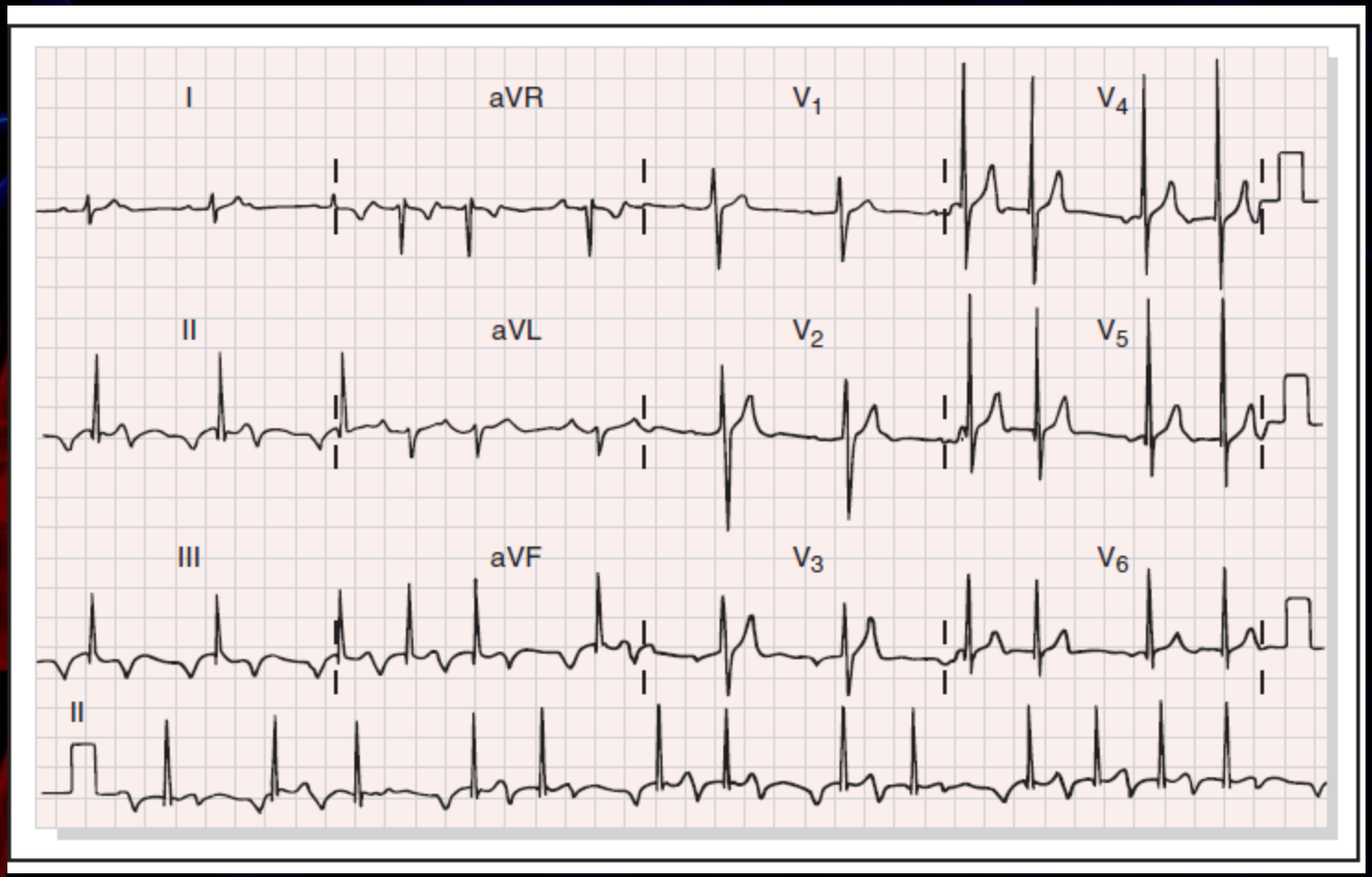
Table 2 Intravenous Medications for Heart Rate Control in Atrial Fibrillation

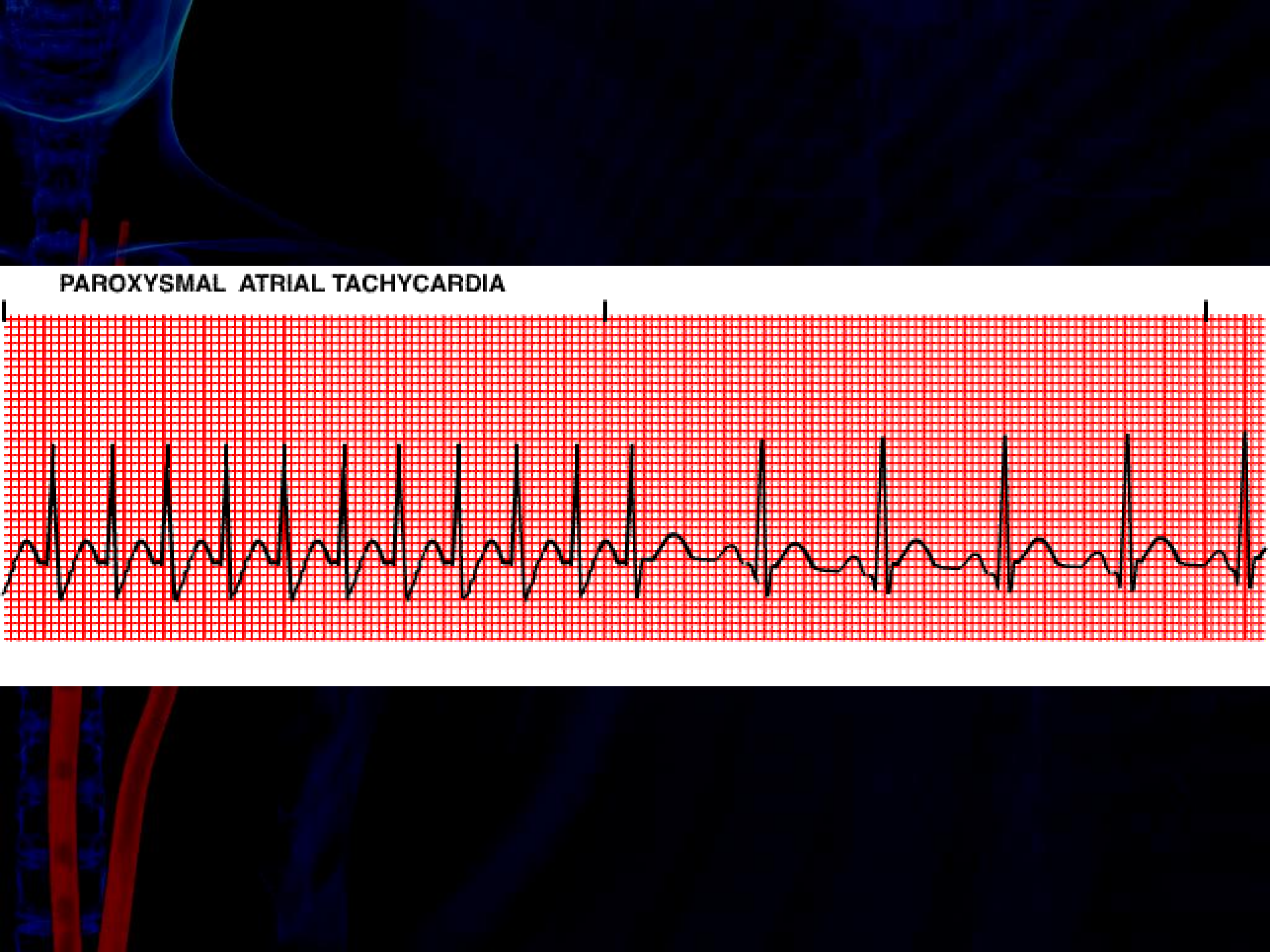
Drug	Loading Dose	Onset	Maintenance Dose
Diltiazem	0.25 mg/kg over 2 min	2–7 min	5–15 mg/h infusion
Esmolol	0.5 mg/kg over 1 min	5 min	0.05–0.2 mg/kg/min
Metoprolol	2.5–5.0 mg over 2 min up to three doses	5 min	NA
Propanolol	0.15 mg/kg	5 min	NA
Verapamil	0.075–0.15 mg/kg over 2 min	3–5 min	NA
Digoxin	0.25 mg each 2 h up to 1.5 mg	2 h	0.125–0.25 mg daily

NA, not applicable.

MULTIFOCAL ATRIAL TACHYCARDIA

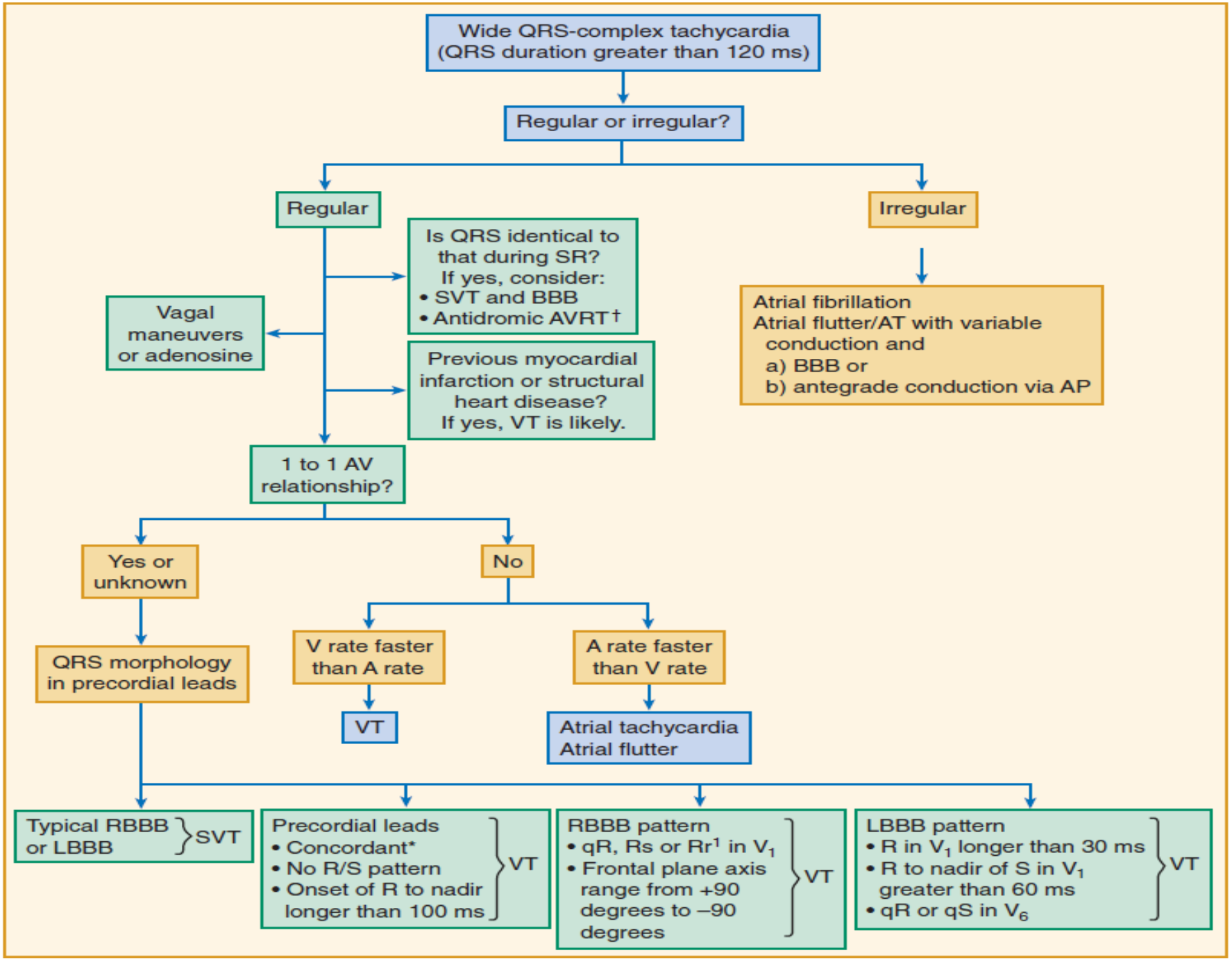
- MAT is an irregular atrial tachycardia diagnosed by identification of three or more p wave morphologies and PR intervals.
- MAT is most often associated with hypoxia in the setting of pulmonary disease but may occasionally be due to use of theophylline, metabolic derangements, and end-stage cardiomyopathy.
- Treatment consists of correcting hypoxia by either or both treating underlying pulmonary disease and correcting electrolyte abnormalities.
- AV nodal blockers are sometimes useful to control the ventricular response in the interim.


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


PAROXYSMAL ATRIAL TACHYCARDIA

WIDE COMPLEX TACHYCARDIA

- The most frequently reported tachyarrhythmia in the ICU setting is a wide complex tachycardia.
- The first step in treatment is establishing the diagnosis, because VT is more ominous than SVT with aberrancy.
- VT is defined by three or more consecutive ventricular beats.
- Sustained VT is defined as more than 30 seconds of ventricular beats at a rate of more than 100 bpm.
- Initial evaluation should include obtaining a 12-lead ECG, and measurement of serum potassium, calcium, and magnesium.
- The ECG should be examined and compared with prior ECGs with attention to QRS width in sinus rhythm, prior Q waves that may indicate prior myocardial infarction (MI), the presence of delta waves, as well as the QT interval.
- A careful review of medications is paramount in excluding iatrogenic causes of VT.


Wide QRS-complex tachycardia (QRS duration greater than 120 ms)
Regular or irregular?
Regular
Irregular
Is QRS identical to that during SR? If yes, consider:
• SVT and BBB
• Antidromic AVRT†
Vagal maneuvers or adenosine
Previous myocardial infarction or structural heart disease? If yes, VT is likely.
Atrial fibrillation
Atrial flutter/AT with variable conduction and
a) BBB or
b) antegrade conduction via AP
1 to 1 AV relationship?
Yes or unknown
No
QRS morphology in precordial leads
V rate faster than A rate
A rate faster than V rate
VT
Atrial tachycardia
Atrial flutter
Typical RBBB or LBBB
SVT
Precordial leads
• Concordant*
• No R/S pattern
• Onset of R to nadir longer than 100 ms
VT
RBBB pattern
• qR, Rs or Rr¹ in V1
• Frontal plane axis range from +90 degrees to –90 degrees
VT
LBBB pattern
• R in V1 longer than 30 ms
• R to nadir of S in V1 greater than 60 ms
• qR or qS in V6
VT

VT can be diagnosed using some clinical and electrocardiographic clues, as outlined following here:

1. Play the odds :

VT is approximately four times more common than SVT with aberrancy. In one study of 200 consecutive patients with a wide QRS tachycardia, 164 were ventricular, 30 were SVT with aberrancy, and six were SVT with antegrade conduction.

2. Ask the right questions.

VT is much more common in patients who have a history of MI or heart failure.

3. Do not rely on hemodynamic alone.

Circulatory collapse is more common with VT than with SVT, but patients with VT may maintain a normal blood pressure.

4. Do not count on AV dissociation.

This is present in less than 50% of cases of VT and is difficult to identify at faster heart rates.

5. Do not count on irregularity.

Regularity was identified in 90% of patients with SVT versus 78% with VT.

Other clues are useful in distinguishing VT from SVT:

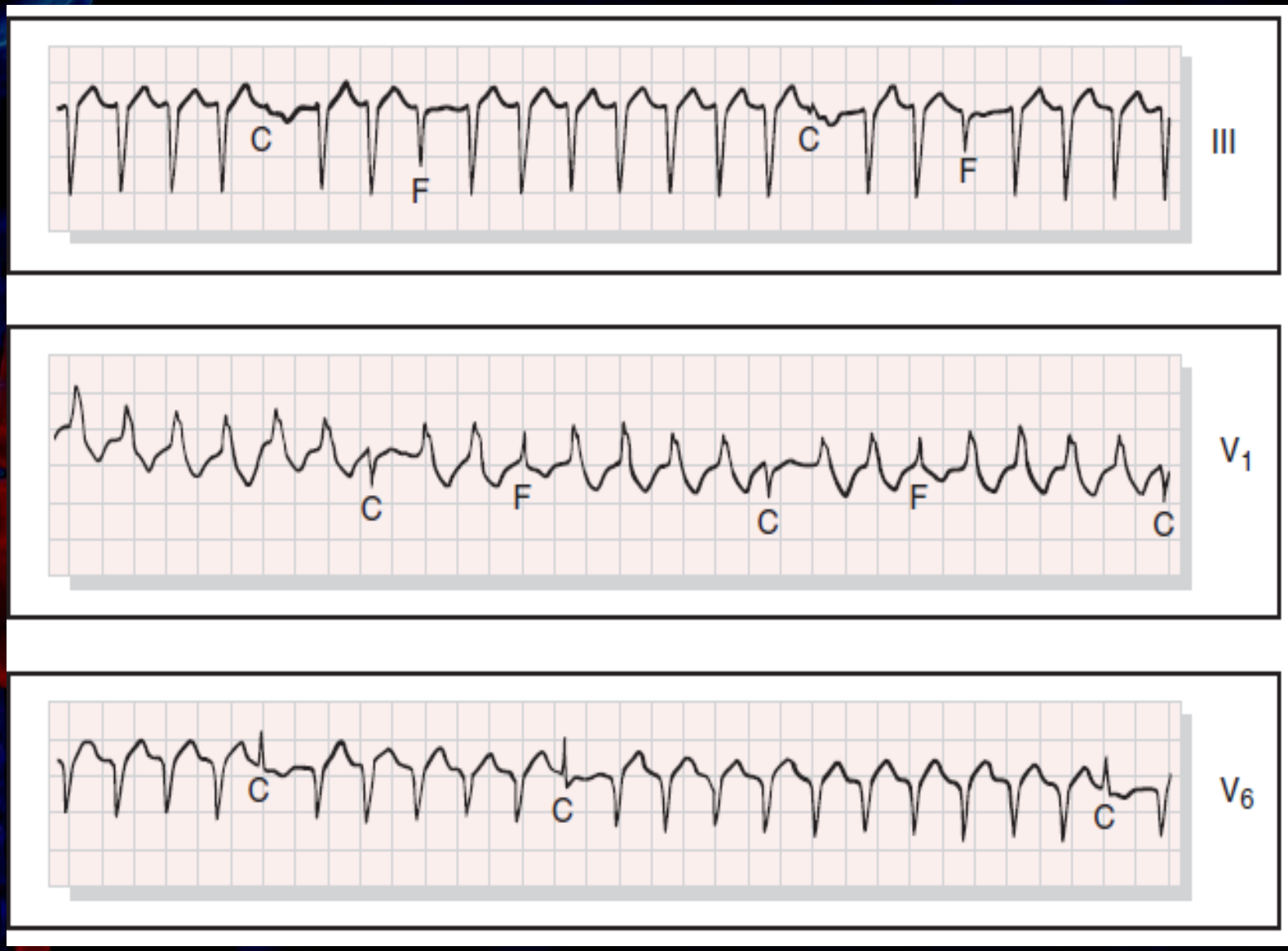
- **A QRS width :**
  - > 0.14 seconds with right bundle branch block
  - or > 0.16 seconds during left bundle branch block

favors VT

- Comparison of QRS morphology during the tachycardia with the morphology of ventricular premature beats in sinus rhythm can be helpful.
- Other diagnostic clues suggestive of VT are ***fusion and capture*** **beats**, but these are seen in only 20 to 30% of cases of VT.

✓**Fusion beats**, a hybrid of the supraventricular and ventricular complexes, occur when two impulses, one supraventricular and one ventricular, simultaneously activate the same territory of ventricular myocardium.

✓**Capture beats** are occasional beats conducted with a narrow complex, and such beats rule out fixed bundle branch block.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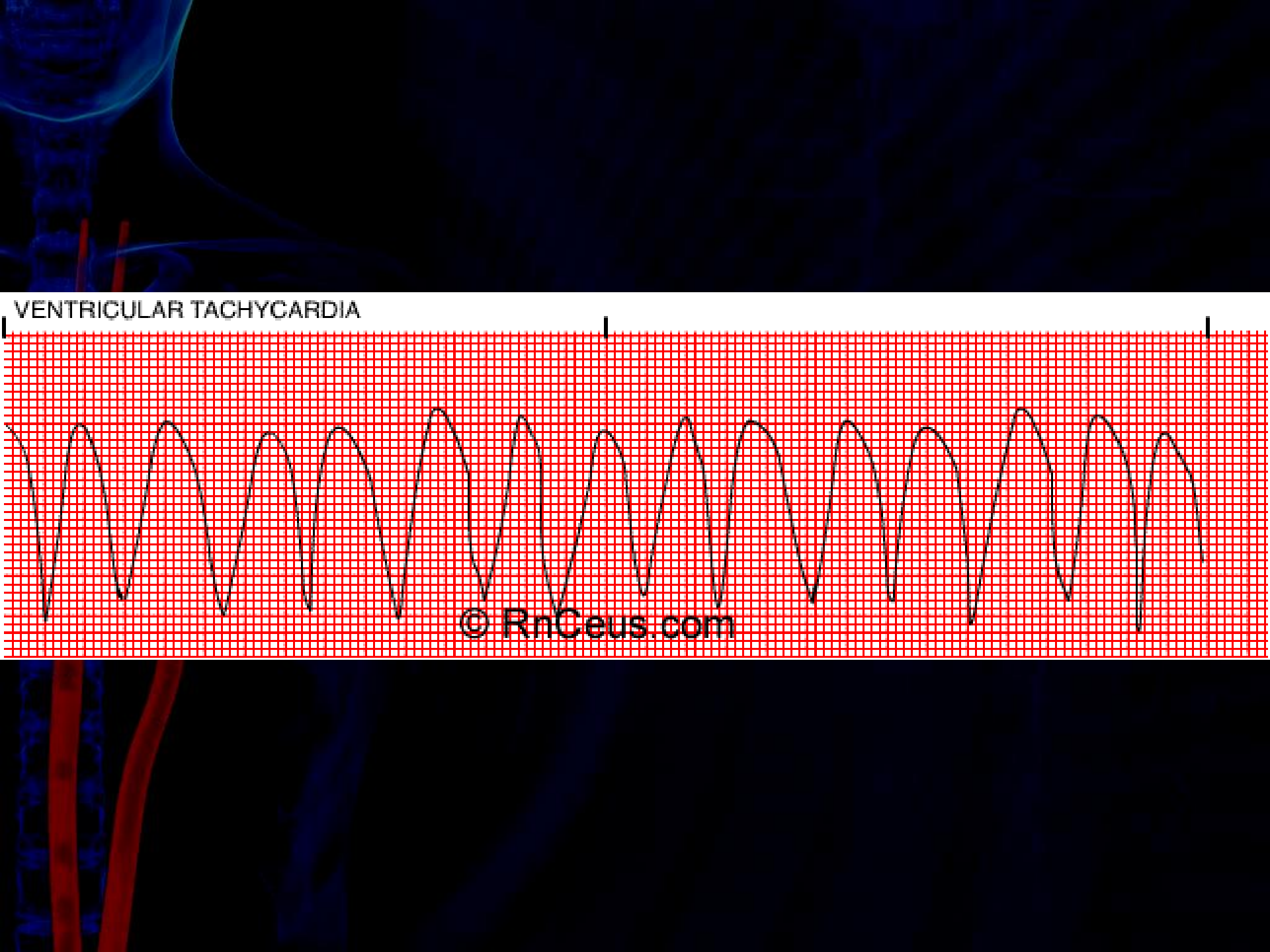
- ***It is better to err on the side of over diagnosis of VT.***

  (The potential consequences of misdiagnosis were demonstrated in a study analyzing adverse events incurred by patients with VT misdiagnosed as SVT and given calcium channel blockers. Many of the patients promptly decompensate and some required resuscitation).

- **Interestingly, all of these patients were hemodynamically stable when first seen in VT.**


VENTRICULAR TACHYCARDIA
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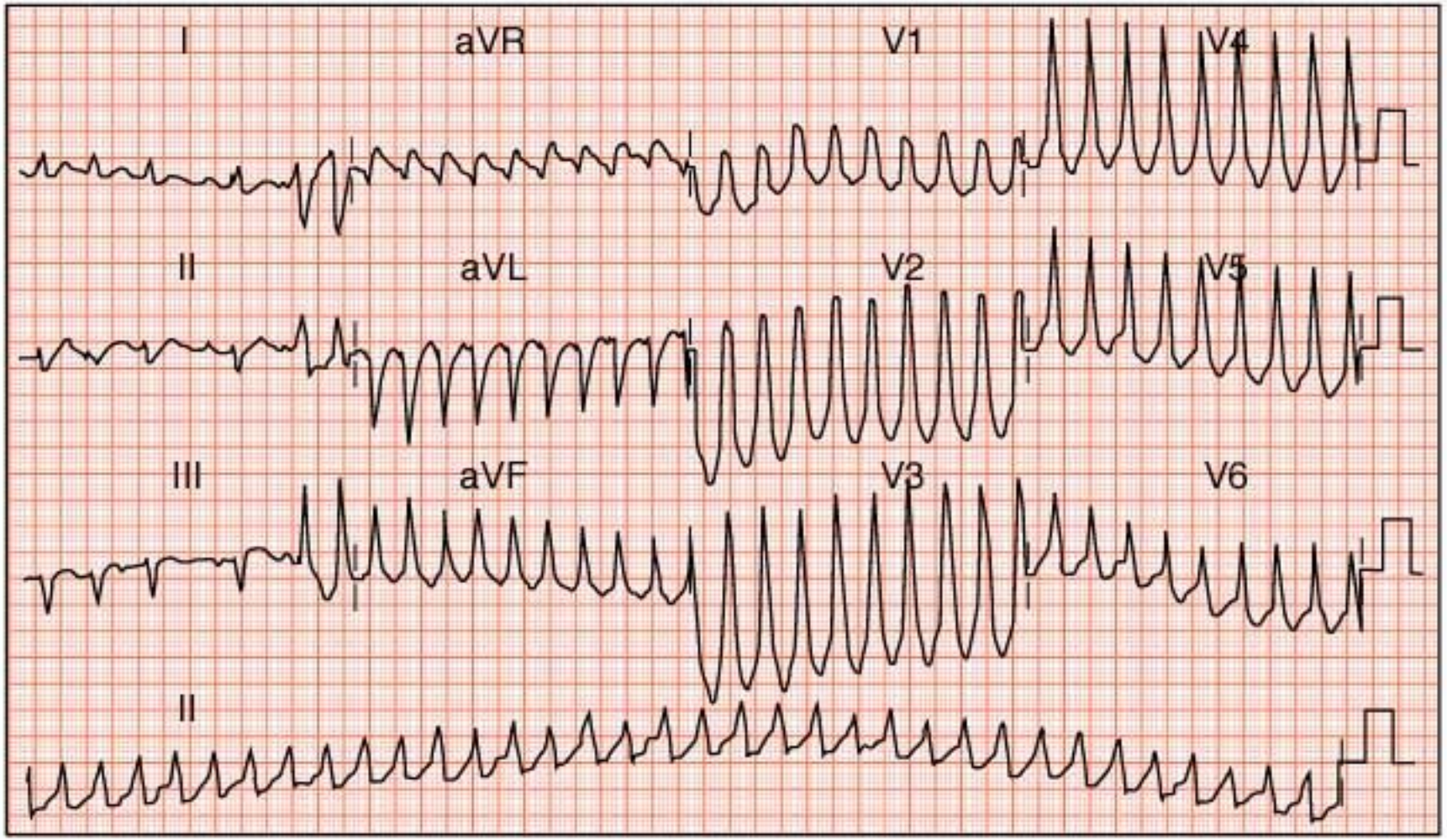
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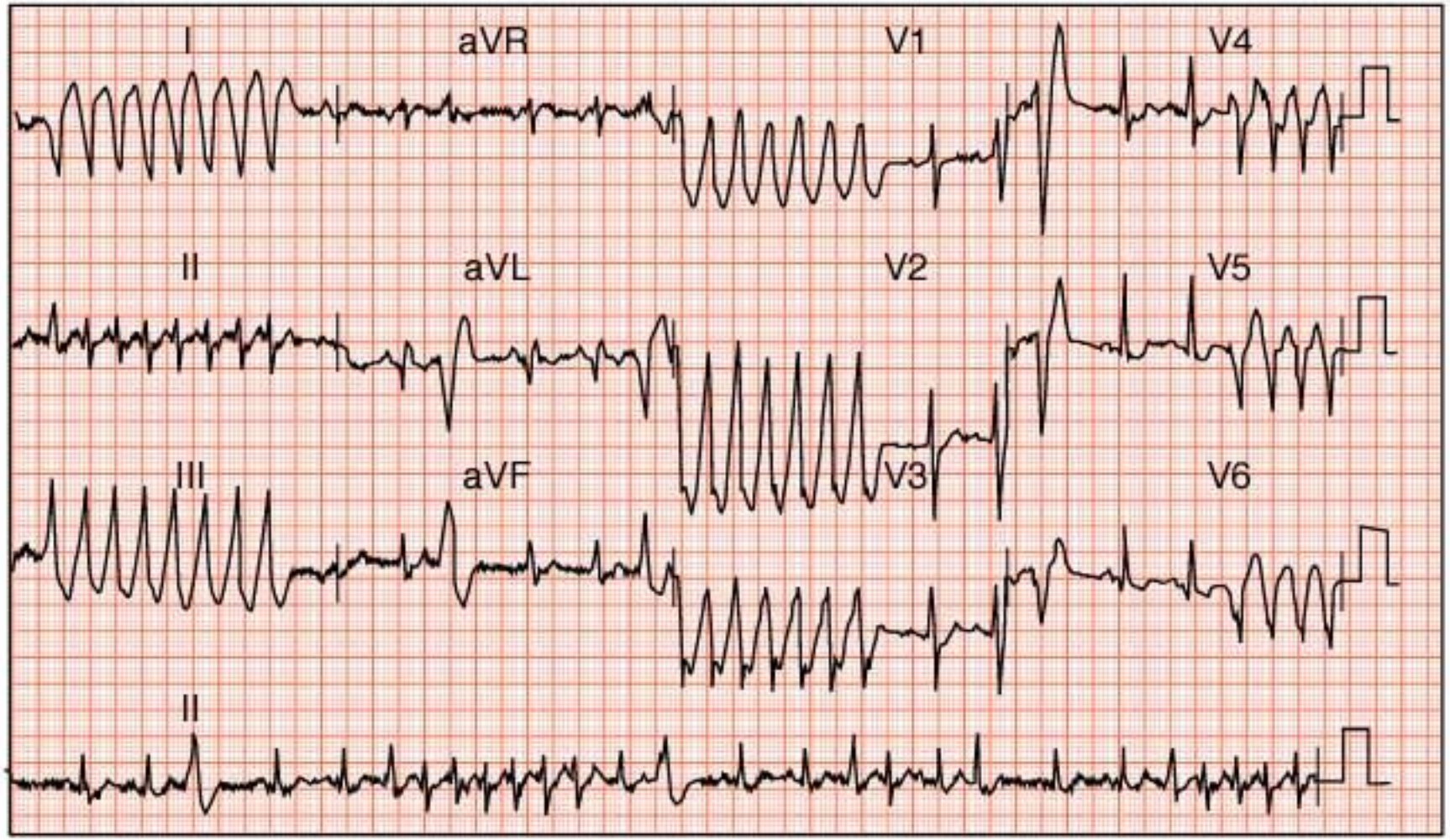
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NONSUSTAINED VT

- This common clinical problem, occurring equally in women and men, is usually asymptomatic, with an incidence of 0 to 4% in the general population.
- A major determinant of prognosis is the presence or absence of underlying structural heart disease.
- The Baltimore Longitudinal Study of Aging screened patients aged 60 to 85 years old for cardiovascular disease and followed them for 10 years; NSVT did not predict coronary events in this population.
- Therefore, in asymptomatic patients with NSVT, a thorough history and physical examination, echocardiography, and stress testing are usually sufficient to exclude prognostically significant structural heart disease.

- Patients with symptoms of palpitations, syncope, or presyncope should undergo further evaluation to exclude episodes of sustained VT or other arrhythmias.

- Patients who have NSVT with structural heart disease (coronary heart disease, dilated cardiomyopathy, or valvular heart disease) require more comprehensive evaluation and management.

- the prognosis of NSVT following a myocardial MI is dependent upon the **timing of onset of VT** in relation to the incident MI :
  - NSVT occurring in the **first 48 hours** of an MI is most likely related to reperfusion or ischemia and has no prognostic significance.
  - NSVT occurring **more than 1 week** after MI doubles the risk of sudden cardiac death (SCD) in patients with preserved left ventricular function.

(The risk of SCD is increased more than fivefold in patients with left ventricular dysfunction (EF less than 40%).
The risk of SCD is greatest in the first 6 months post-MI and persists for up to 2 years).

- NSVT is present in up to 80% of patients with an idiopathic dilated cardiomyopathy (EF<40%).

- The current ACC/AHA guidelines recommend implantation of ICD for NSVT in patients with coronary disease, prior MI, LV dysfunction, and inducible VF or sustained VT at EPS that is not suppressible by a class I antiarrhythmic drug.

- Initial treatment of NSVT in the setting of DCM should include correction of electrolyte abnormalities, removal of exacerbating factors (hypoxia, dehydration, medications, vasopressors, etc.), and up titration of b-blockers.

- **Mitral and aortic valve disease** is associated with NSVT, occurring in up to 20% of patients with MVP and 5% of patients with AS >>>> does not appear to be associated with increased risk of SCD.

- **In patients at high risk** , further evaluation is warranted. This may include cardiac catheterization, EPS , and/or signal averaged ECG.

MONOMORPHIC VT

- Monomorphic VT in the setting of a normal QT interval usually occurs from a fixed substrate (i.e., scar) rather than acute ischemia.
- The importance of monomorphic VT depends on the clinical milieu in which it occurs & on the presence of underlying structural heart disease.
- Sustained monomorphic VT, either with or without acute ischemia, portends a worse prognosis even after hospital discharge.

❑ **Treatment of sustained monomorphic VT :**

-hemodynamic instability>> synchronized DCC

-Stable or recurrent monomorphic VT>>> lidocaine, procainamide, or amiodarone.

❑ The next step in evaluation and management of the patient is dependent on left ventricular function :

- LV function is normal : procainamide, amiodarone, lidocaine, or sotalol.
- impaired LV function (EF <40%) : The choices are limited to amiodarone or lidocaine .

- **Amiodarone:** can be given as a 150 mg IV bolus over 10 minutes followed by an infusion of 360 mg (1 mg/min) over 6 hours, and then 540 mg (0.5 mg/min) over the remaining 18 hours. **(The maximum total dose is 2.2 g over 24 hours).**
  - Bradycardia and hypotension can result from IV amiodarone, in which case the rate of the infusion should be decreased.

- **Lidocaine:** is administered by IV bolus of 0.5 to 0.75 mg/kg, followed by continuous infusion at 1 to 4 mg/min.

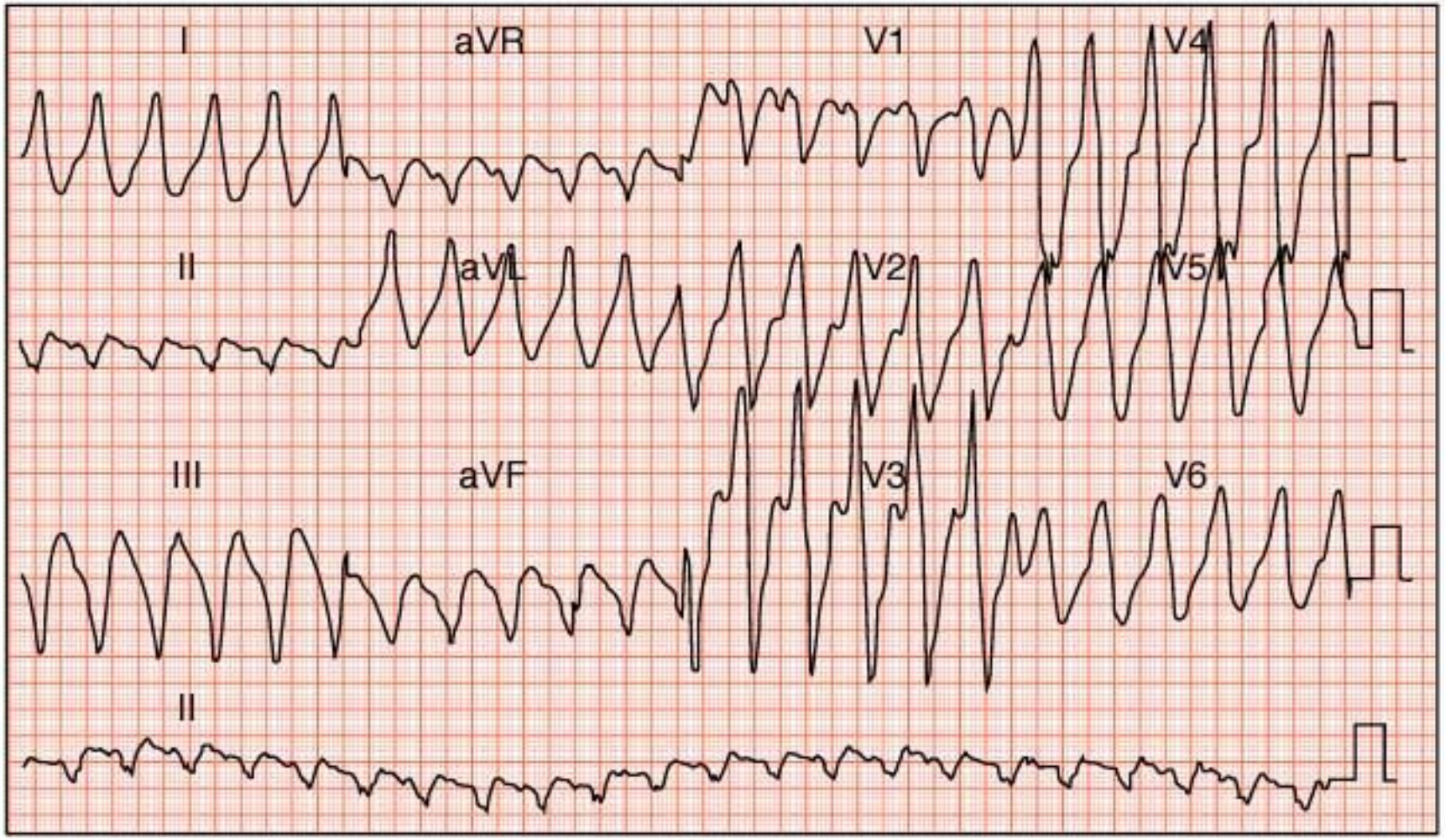
- **Procainamide:** is administered at 20 mg/ min IV for a loading dose of 17 mg/kg, then continued as an infusion at 1 to 4 mg/min.
  - The infusion should be stopped if the patient becomes hypotensive or the QRS widens by 50% above baseline.

The most serious side effects of procainamide are hypotension and proarrhythmia (most commonly torsades de - pointes), both of which increase in frequency in patients with renal insufficiency because of decreased excretion.

- **If the QTc is longer than 500 msec** the drug should be stopped immediately and the QTc followed closely.
- Cimetidine and amiodarone can increase levels of procainamide and its metabolite Nacetyl procainamide.
- **Measurement of serum levels** may be useful, especially in patients with renal insufficiency.

- In patients with transvenous or epicardial pacemakers, overdrive antitachycardia pacing is an option. **(**The ventricular pacing rate should be 10 to 20 bpm faster than the VT**).**

- Absent a reversible cause >>> ICD should be considered in patients with recurrent monomorphic VT and an EF less than 40% or a history of syncope.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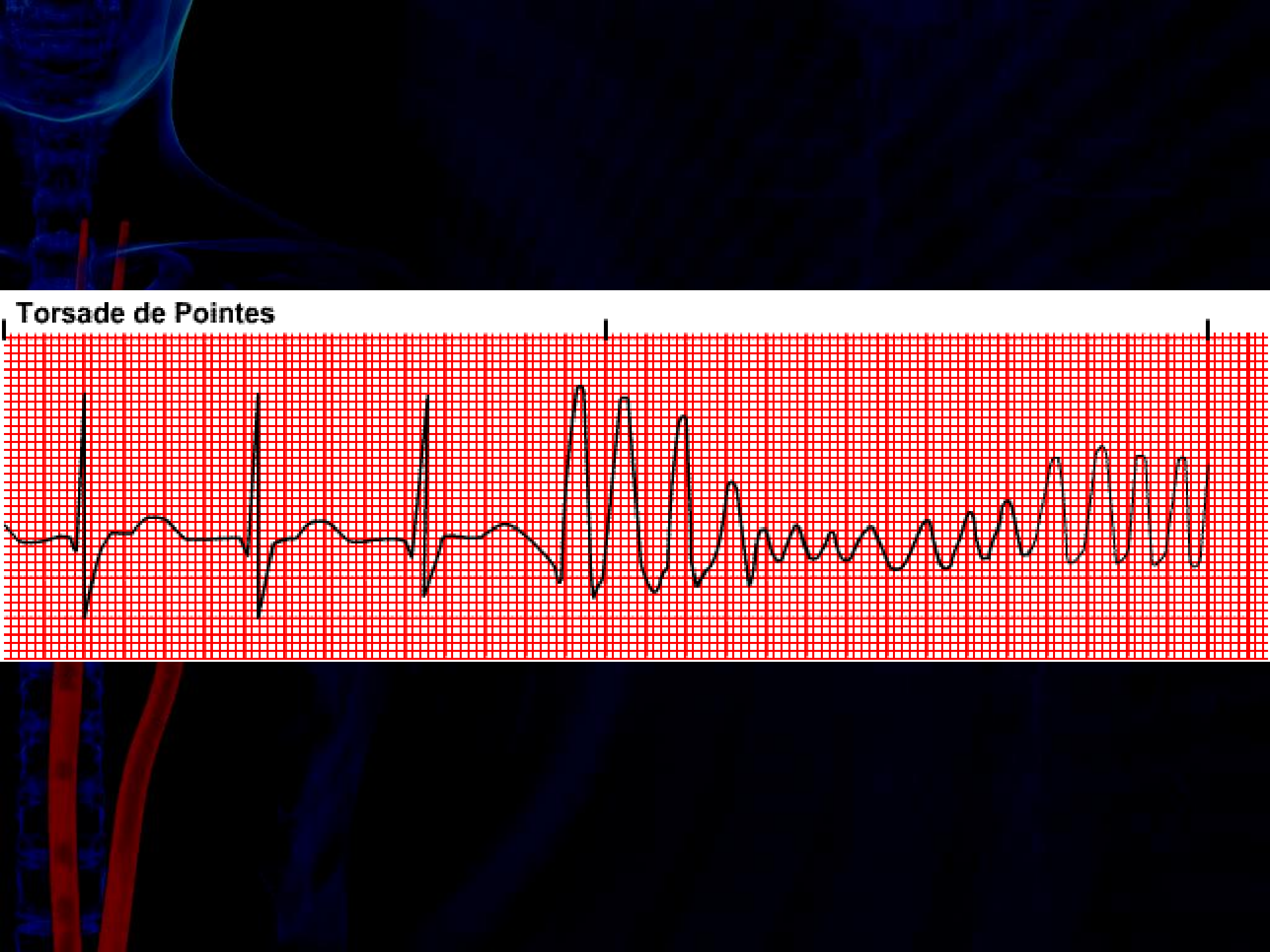
POLYMORPHIC VT

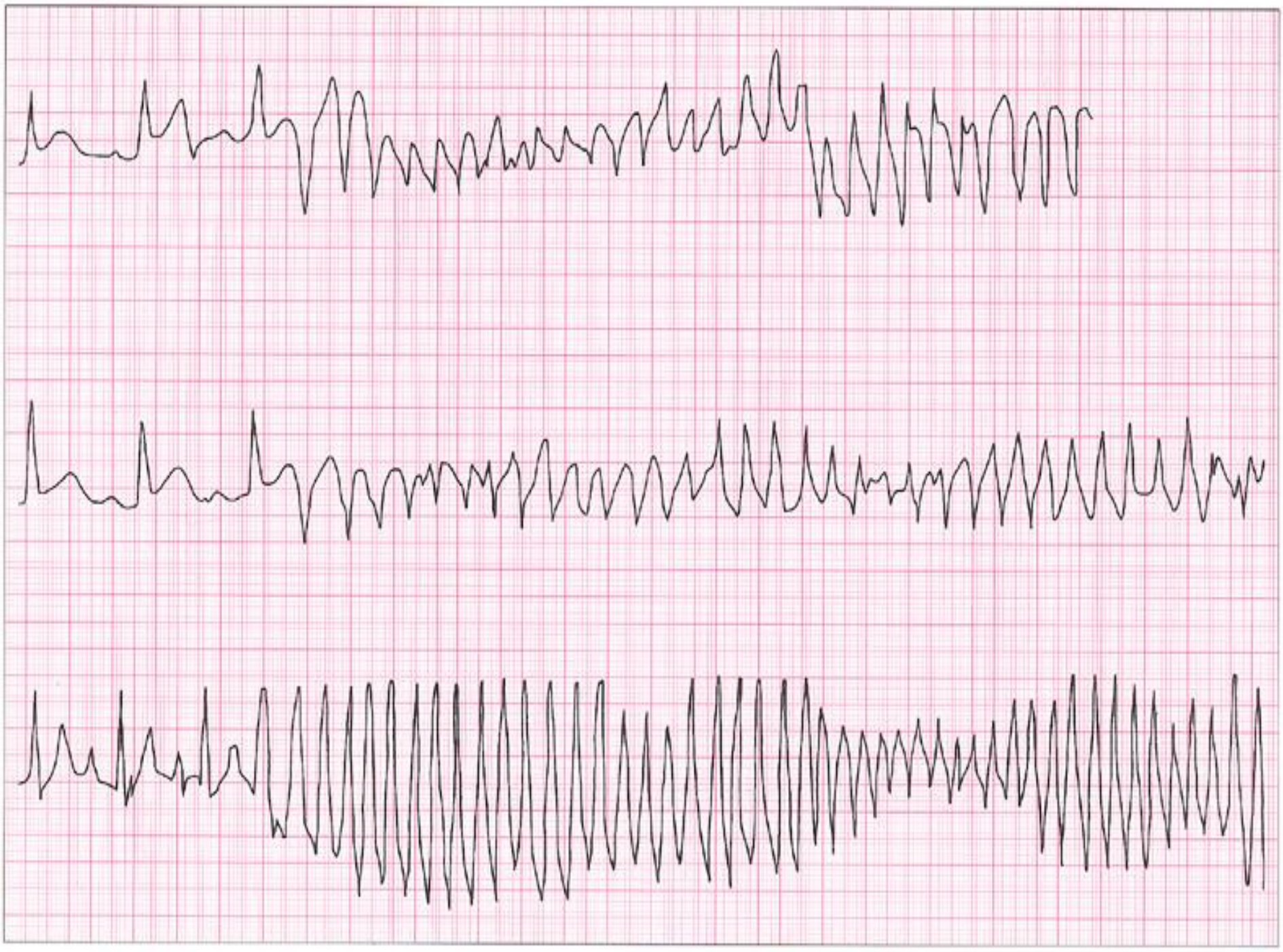
- Polymorphic VT with a normal QT interval is considered to be an ischemic rhythm that typically degenerates into VF.
- It is almost never asymptomatic and thus DC synchronized cardioversion is the initial recommended treatment.
- Polymorphic VT with a normal QTc is a more ominous sign than monomorphic VT in patients with myocardial ischemia.
- Medications that might predispose to ischemia, such as inotropes or vasopressors, should be stopped or tapered, (if possible) , and b-blockers started(if blood pressure permits).
- Intraaortic balloon pumping may be useful as a supportive measure, but revascularization is usually required.
- If withdrawal of vasopressors is contraindicated on a clinical basis, IV infusion of lidocaine or amiodarone should be initiated.

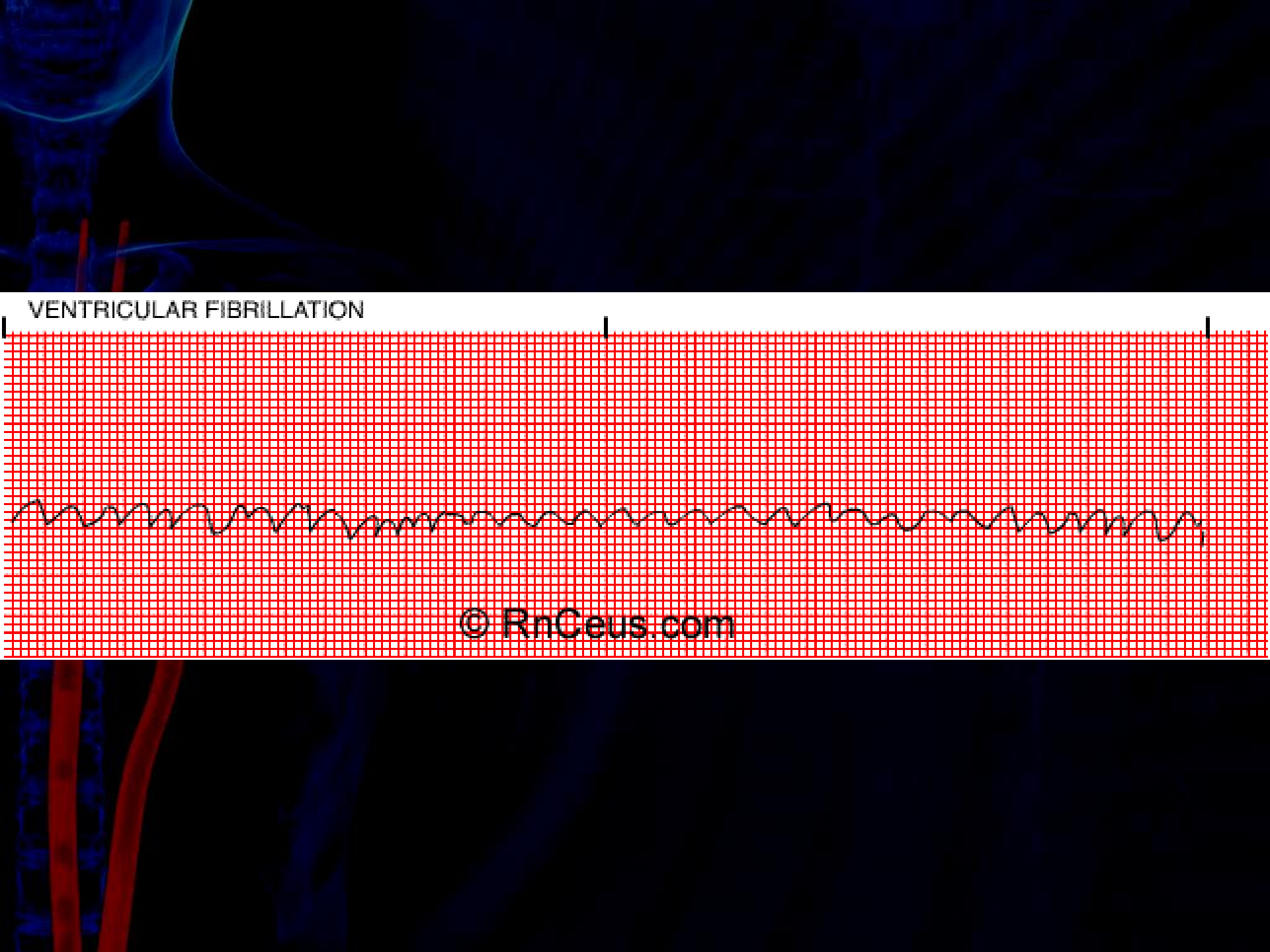
TORSADES DE POINTES

- Torsades de pointes is a French term translated as ‘‘twisting of the points.’’ It is a syndrome composed of polymorphic VT and a prolonged QTc interval (by definition 460 millisecondsec).
- This may be due to various medications, including procainamide, disopyramide, sotalol, phenothiazines, quinidine, some antibiotics (erythromycin, pentamidine, ketoconazole), some antihistamines (terfenadine, astemizole), and tricyclic antidepressants.
- Other etiologies include hypokalemia, hypocalcemia, subarachnoid hemorrhage, congenital prolongation of the QTc interval, and insecticide poisoning.

- **A key to treatment is** correction of any exacerbating factors and normalization of electrolyte disturbances, particularly hypomagnesemia, hypocalcemia, and hypokalemia.
- **Magnesium** should be given 1 to 2 g IV push over 30 to 60 minutes.
- **Other potential treatments** may include overdrive pacing or isoproterenol to increase heart rate and thus shorten QTc.
- Administration of **sodium bicarbonate IV** can be useful to antagonize the proarrhythmic effects of class I antiarrhythmics.


Torsade de Pointes




VENTRICULAR FIBRILLATION
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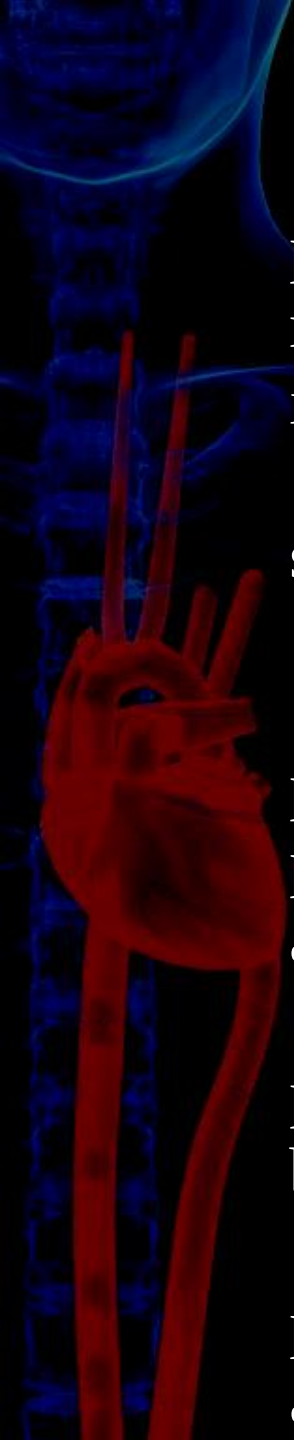
WPW SYNDROME (VENTRICULAR PREEXCITATION)

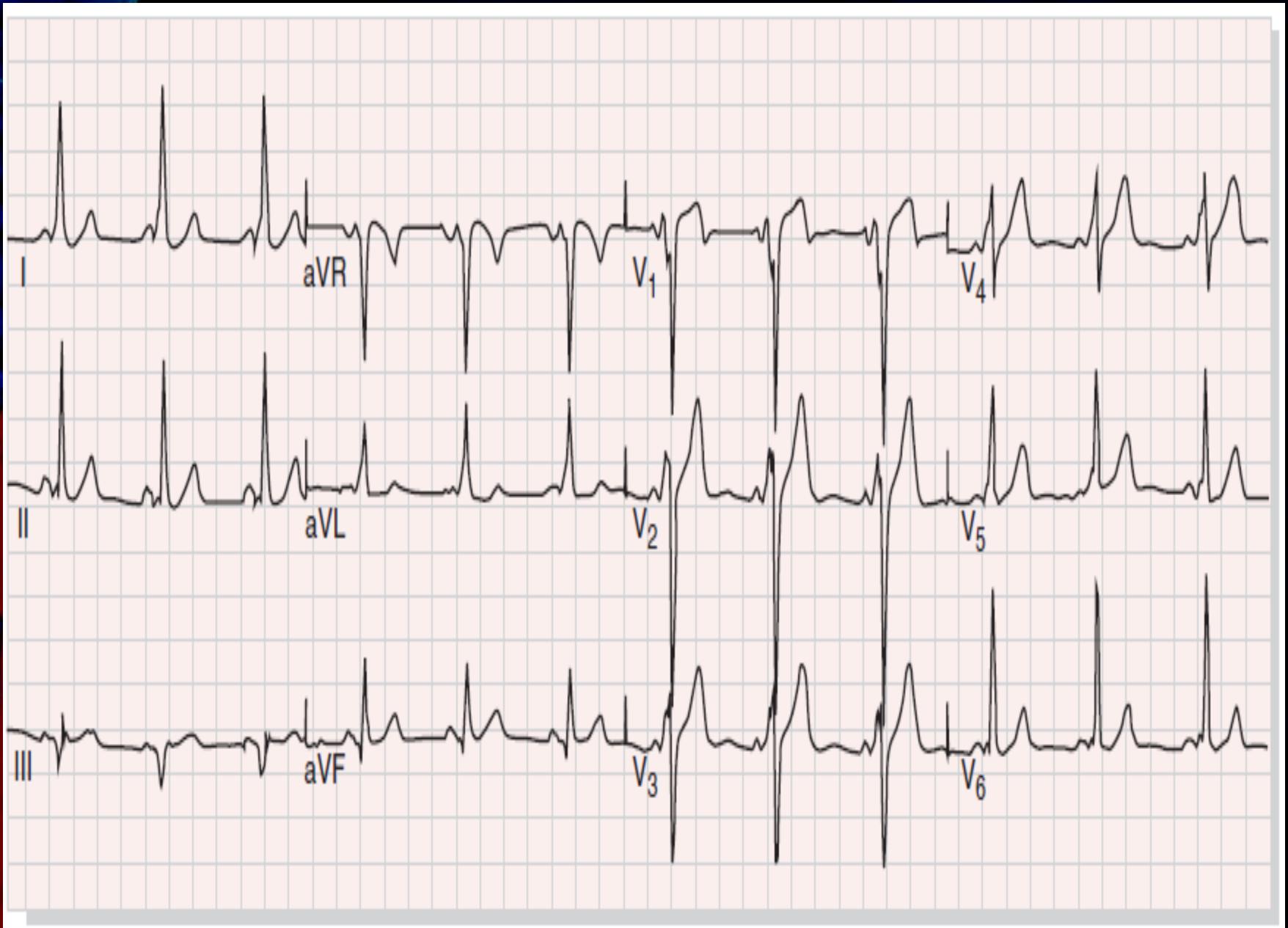
- AVRT using an accessory bypass tract, WPW, occurs in 0.1 to 0.3% of the general population.
- An accessory pathway bypass tract (bundle of Kent), bypasses the AV node and can activate the ventricles prematurely in sinus rhythm, producing the characteristic delta wave.
- The diagnosis of WPW is reserved for patients with both preexcitation and tachyarrhythmias.
- In AVRT conduction :
  - **antidromic:** down the bypass tract and back up the AV node, producing a wide QRS complex
  - **orthodromic:** down the AV node and back up the bypass tract, producing a narrow QRS complex

(AVRT should be suspected in any patient whose heart rate exceeds 200 bpm).



- AF is a potentially life-threatening arrhythmia in patients with WPW syndrome because it can generate a rapid ventricular response with subsequent degeneration into VF.
- This is important because 1/3 of patients with WPW syndrome have AF.

- Adenosine should be used with caution in any young patient suspected of having WPW because it may precipitate AF with a rapid ventricular response rate down an antegrade accessory pathway.
- Procainamide, ibutilide, and flecainide are preferred agents because they slow conduction through the bypass tract.
- The long-term treatment of choice for symptomatic patients is radiofrequency catheter ablation of the accessory pathway.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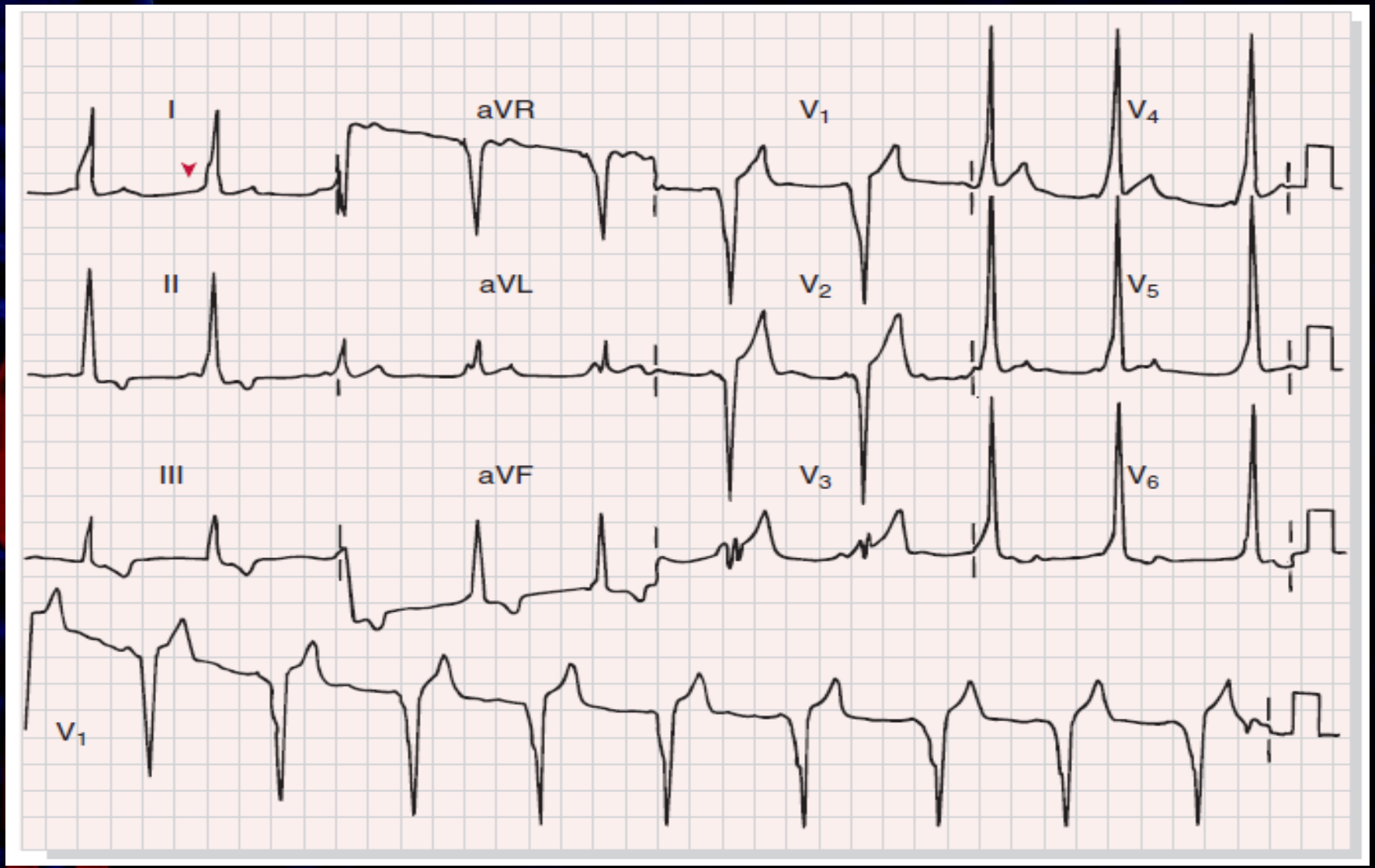
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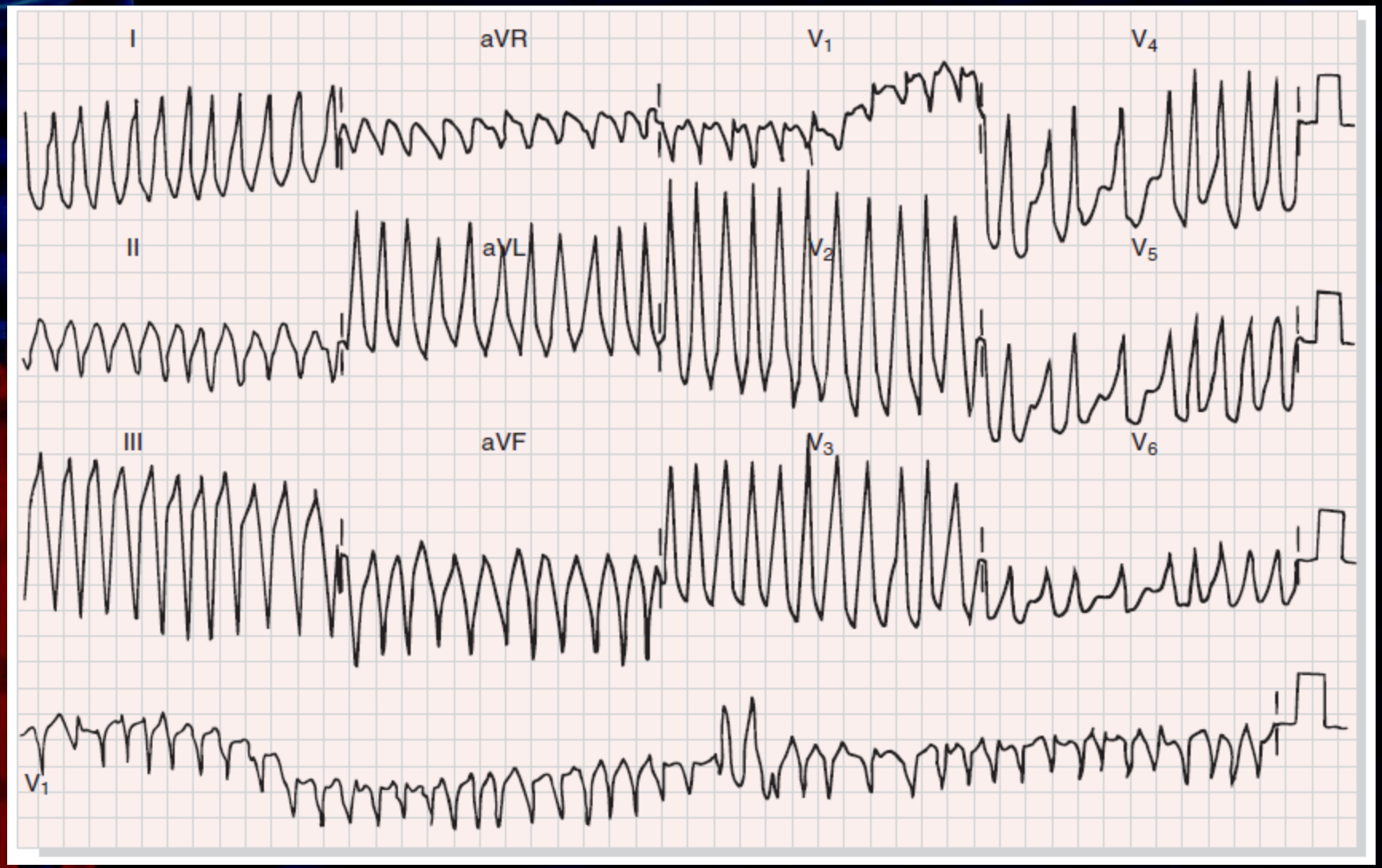
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ELECTRICAL STORM

- **The definition** of an electrical storm is **> 3** distinct episodes of VT/VF within a 24-hour period.
- In patients with ventricular arrhythmias requiring ICD implantation, the incidence of ventricular storm ranges from **10 to 30%**
- According to one study, the event occurred at an average of 13 days after ICD implantation.
- **Precipitating factors** (hypokalemia, myocardial ischemia, and heart failure) were identified in only 26% of the patients in one study.
- Evaluation should include measurement of serum electrolytes, obtaining an ECG, and further evaluation for ischemic heart disease, which may include coronary angiography.
- Proarrhythmia secondary to antiarrhythmic drugs that prominently slow conduction velocity, such as flecainide, propafenone, and moricizine, should be excluded.

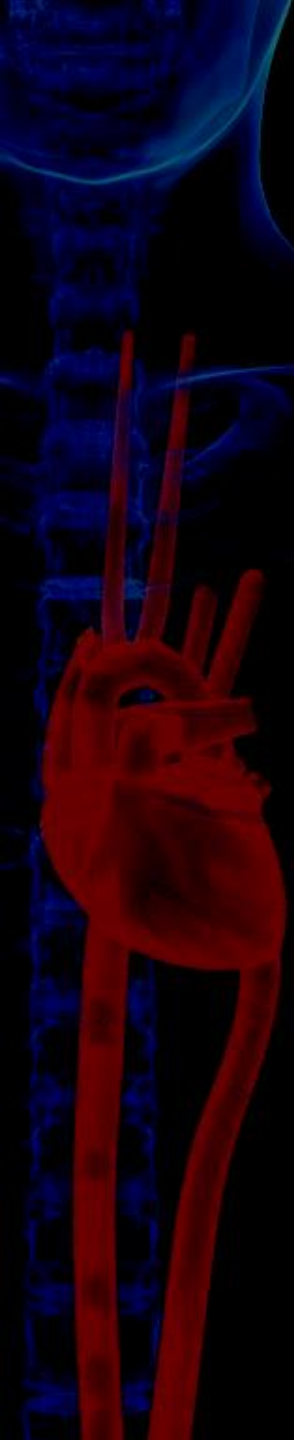
- **Treatment for proarrhythmia** is hemodynamic support until the drug is excreted.
- If exacerbating factors (acute heart failure, electrolyte abnormalities, proarrhythmia, myocardial ischemia, and hypoxia) are corrected, repeated doses of IV amiodarone should be given (even if the patient is already on oral amiodarone).
- Deep sedation can help reduce sympathetic activation.
- Mechanical ventilatory support and IV b-blockers can be used in conjunction, but IV amiodarone is the pharmacological treatment of choice for this condition.

- If pharmacological therapy and antitachycardia pacing are unsuccessful, electrophysiology mapping-guided catheter ablation can be considered, although this is often difficult in unstable patients.

- The prognosis of patients with electrical storm after ICD implantation is poor, with a 2.4-fold increase in the risk of subsequent death, independent of ejection fraction.

The risk of SCD is greatest 3 months after an electrical storm.

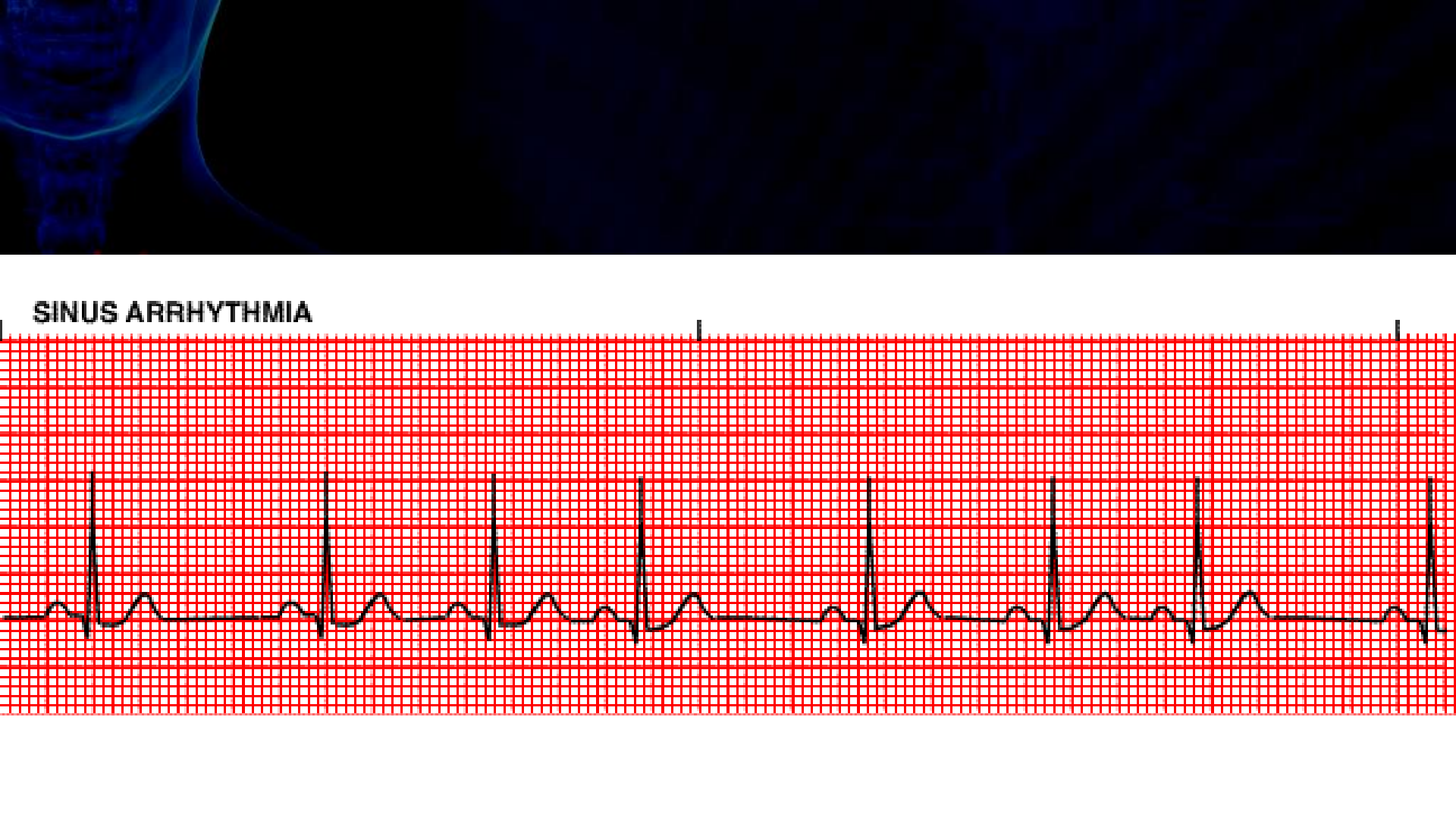
BRADYARRHYTHMIAS & PACING

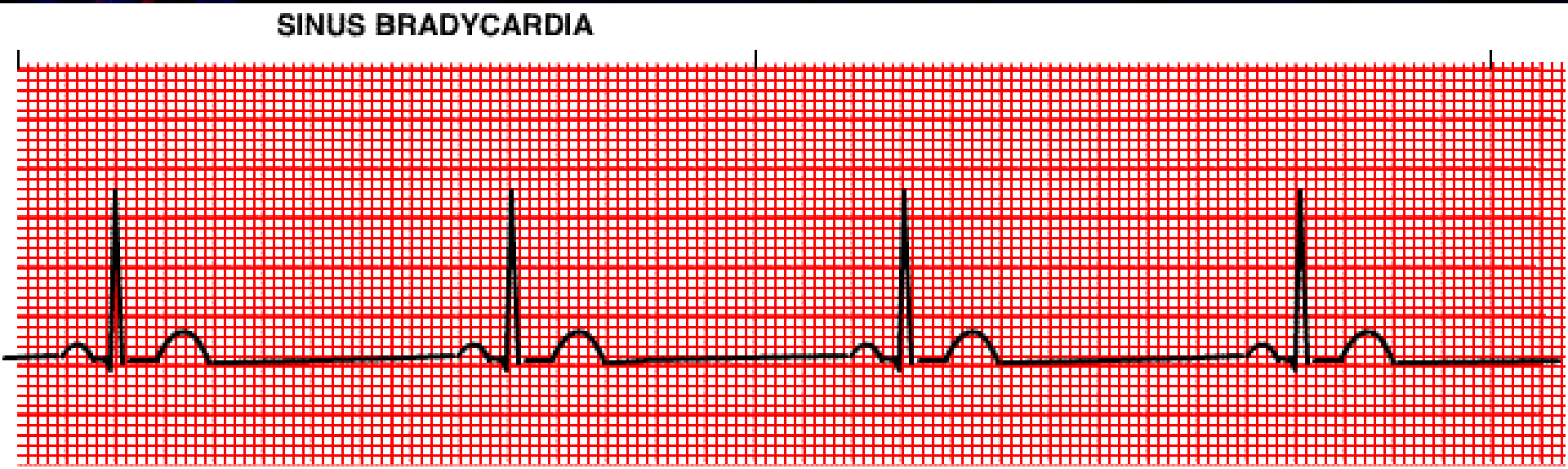


- Asymptomatic bradyarrhythmias do not carry a poor prognosis and in general no therapy is indicated.
- Recommended initial therapy for bradycardia inducing

end organ perfusion problems is atropine IV 1.0 mg.

- The presence of syncope, heart failure, or other symptoms accompanying bradycardias is an indication for pacemaker implantation.
- Third degree or advanced heart block with either symptomatic bradycardia, pauses 3 sec, or heart rate <40 bpm is also an indication for pacemaker insertion.

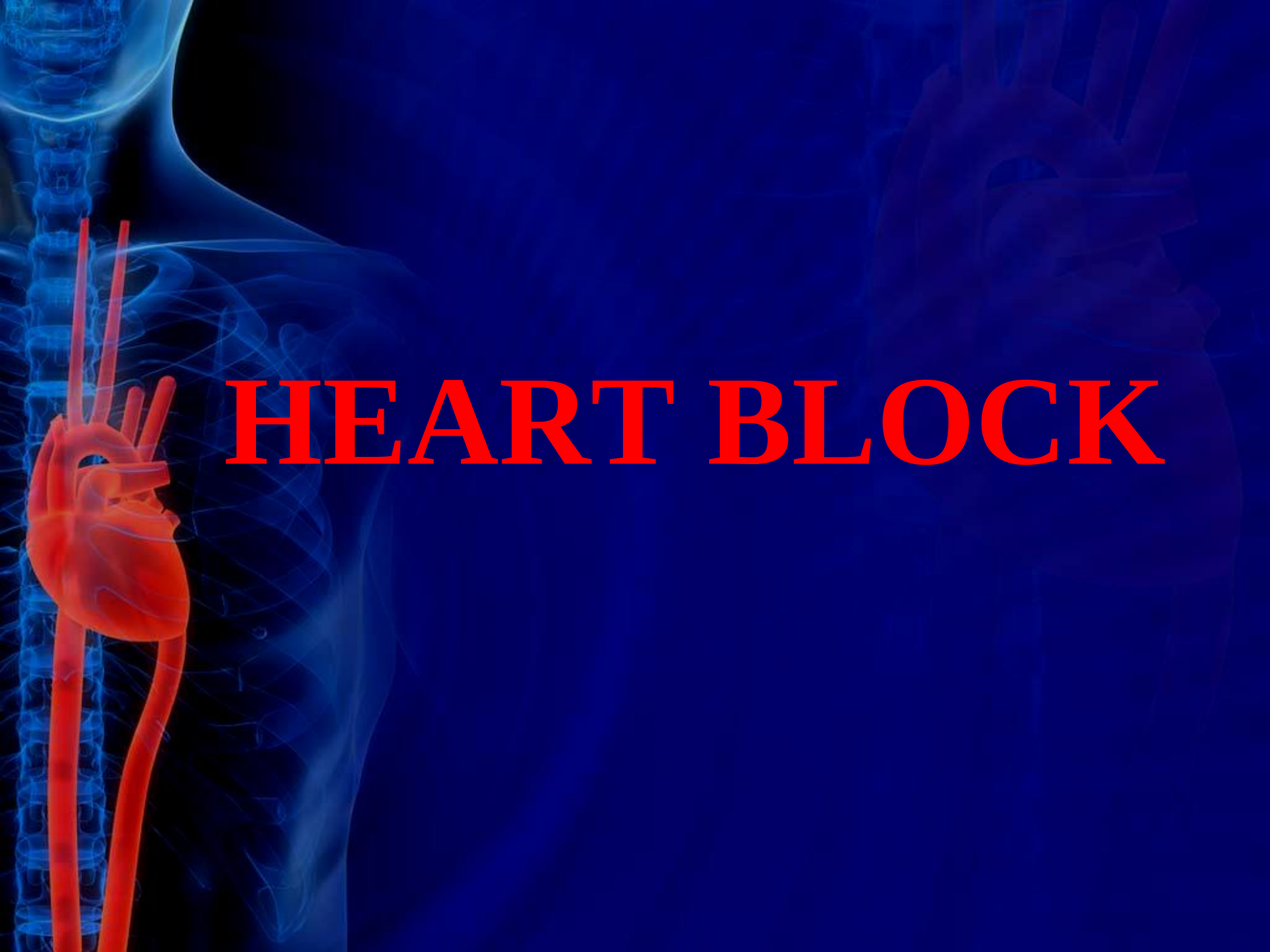

SINUS ARRHYTHMIA

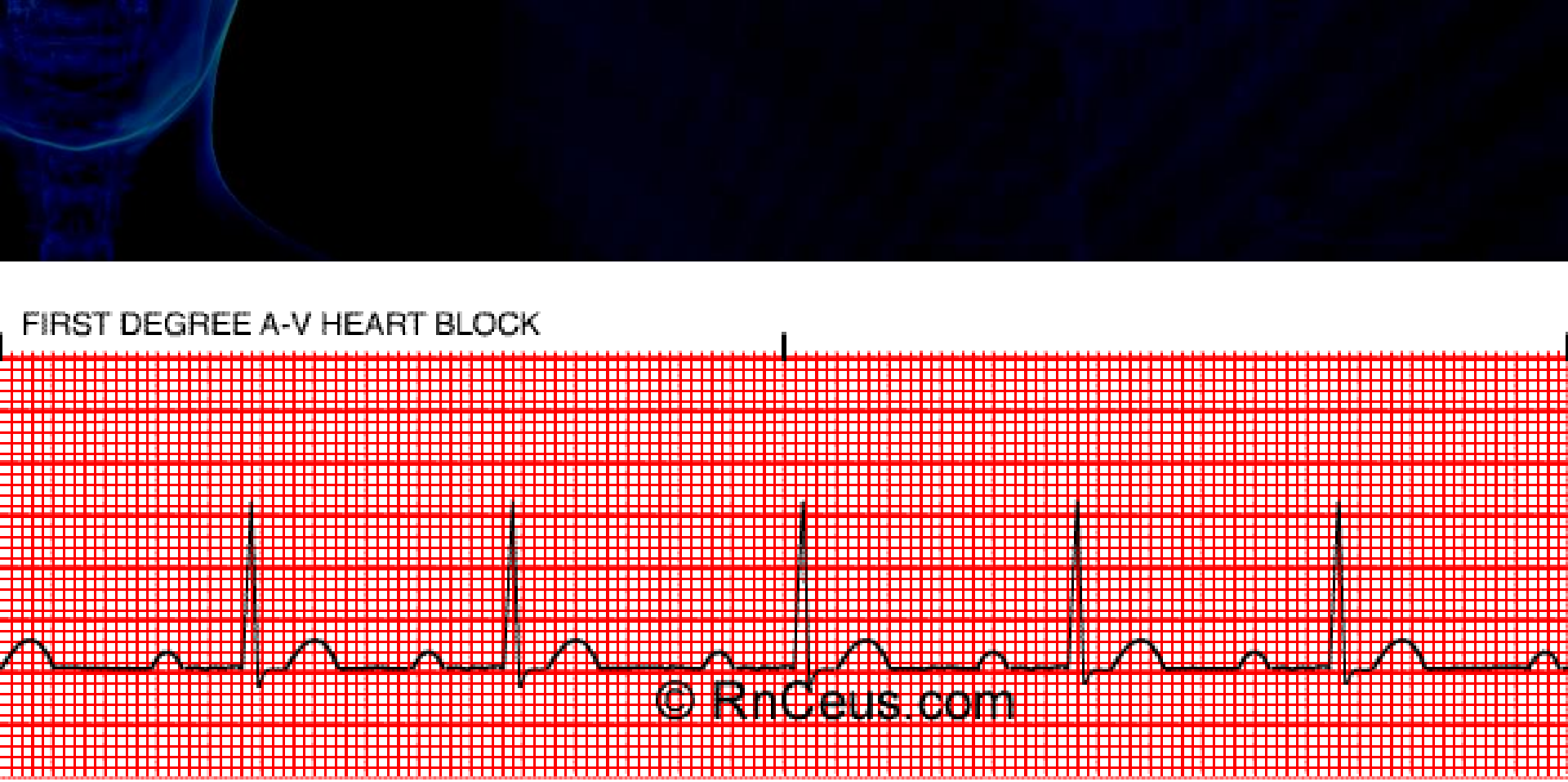

SINUS BRADYCARDIA

SINUS PAUSE, ARREST

- **Rate:** normal
- **P wave:** those that are present are normal
- **QRS:** normal
- **Conduction:** normal
- **Rhythm:** The basic rhythm is regular. The length of the pause is not a multiple of the sinus interval.
- This may occur in individuals with healthy hearts. It may also occur with increased vagal tone, myocarditis, MI, and digitalis toxicity.
- If the pause is prolonged, escape beats may occur.
- The treatment of this dysrhythmia depends on the underlying cause.
  - If the cause is due to increased vagal tone and the patient is symptomatic, atropine may be indicated.


SINUS PAUSE, ARREST


HEART BLOCK


FIRST DEGREE A-V HEART BLOCK
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SECOND DEGREE AV BLOCK - MOBITZ TYPE I (WENCKEBACH)
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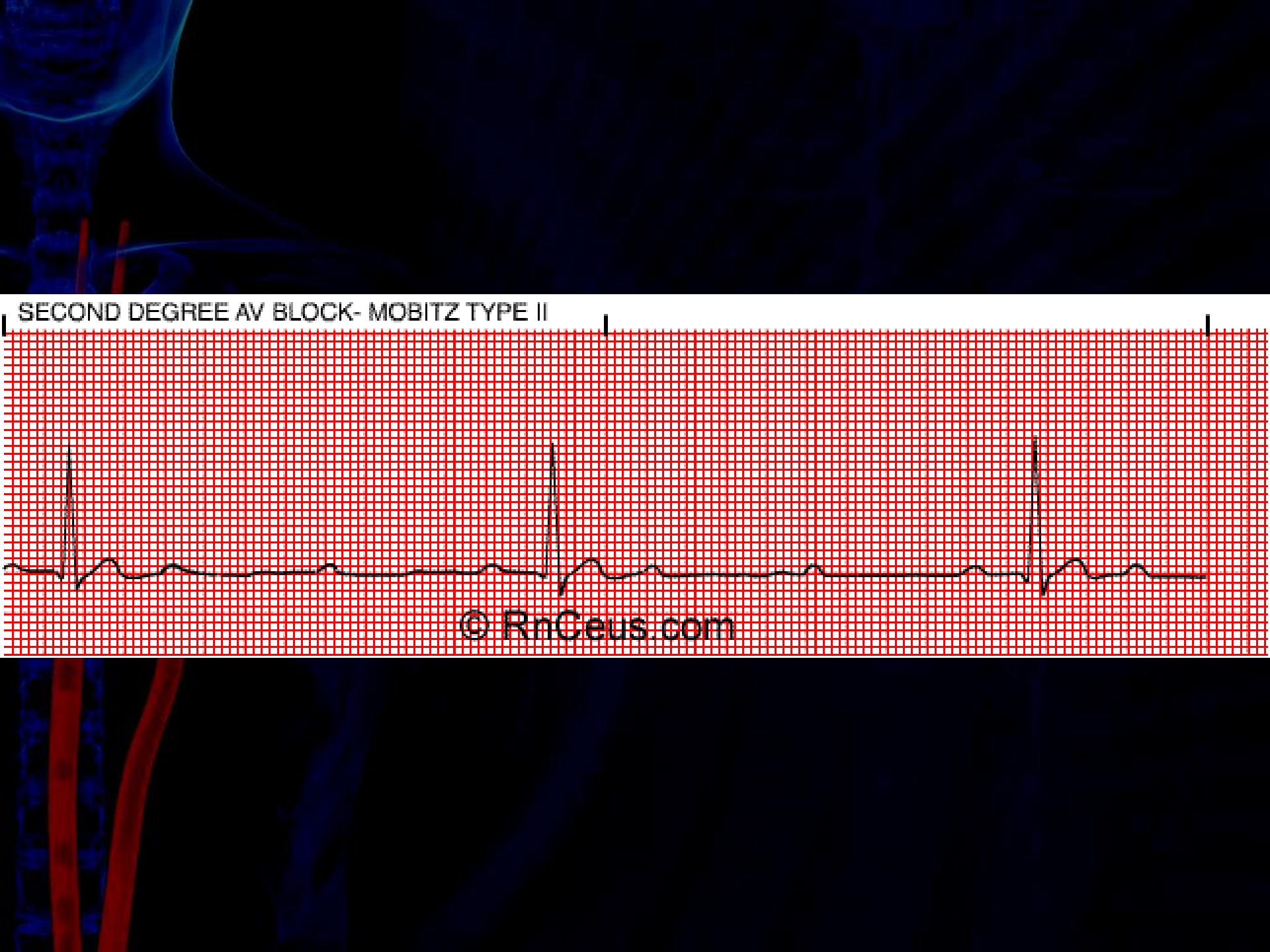
SECOND DEGREE AV BLOCK- MOBITZ TYPE II
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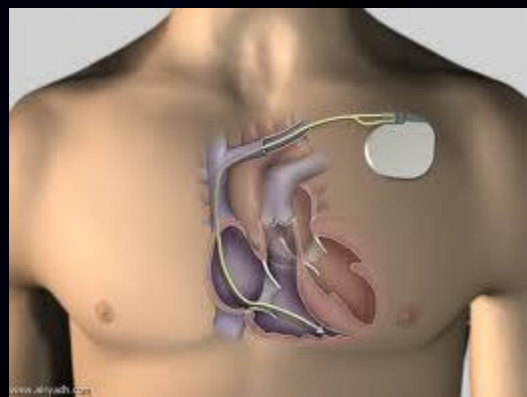
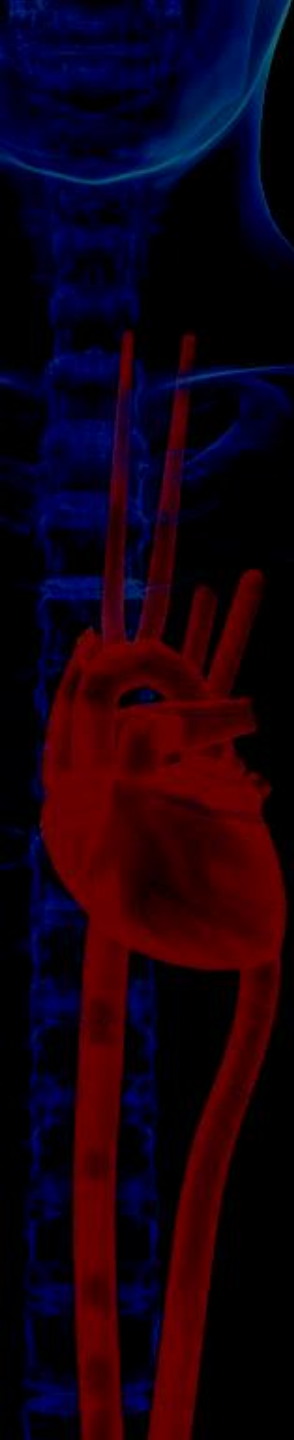
THIRD DEGREE (COMPLETE) AV BLOCK

Class I indications for temporary transvenous pacing after an acute MI

1. Asystole.
2. Symptomatic bradycardia.
3. Bilateral bundle branch block (BBB)
    a. Alternating BBB or right BBB (RBBB) with alternating left anterior fascicular block (LAFB)/ left posterior fascicular block (LPFB)
4. New or indeterminate age bifascicular block with first-degree AV block.
    a. RBBB with LAFB or LPFB
    b. Left BBB (LBBB)
5. Mobitz type II second-degree AV block.




TAKE A HOME MASSAGE



- Dealing with arrhythmia >>>like dealing with the fire>> immediate and definitive management >>can save many life.

- Review common and significant arrhythmias and EKG findings.

❑LOOK FOR:

- **YOUR MEDICATIONS.**
- **YOUR PATIENT:**

- If unstable >>> call Code Blue and follow ACLS.

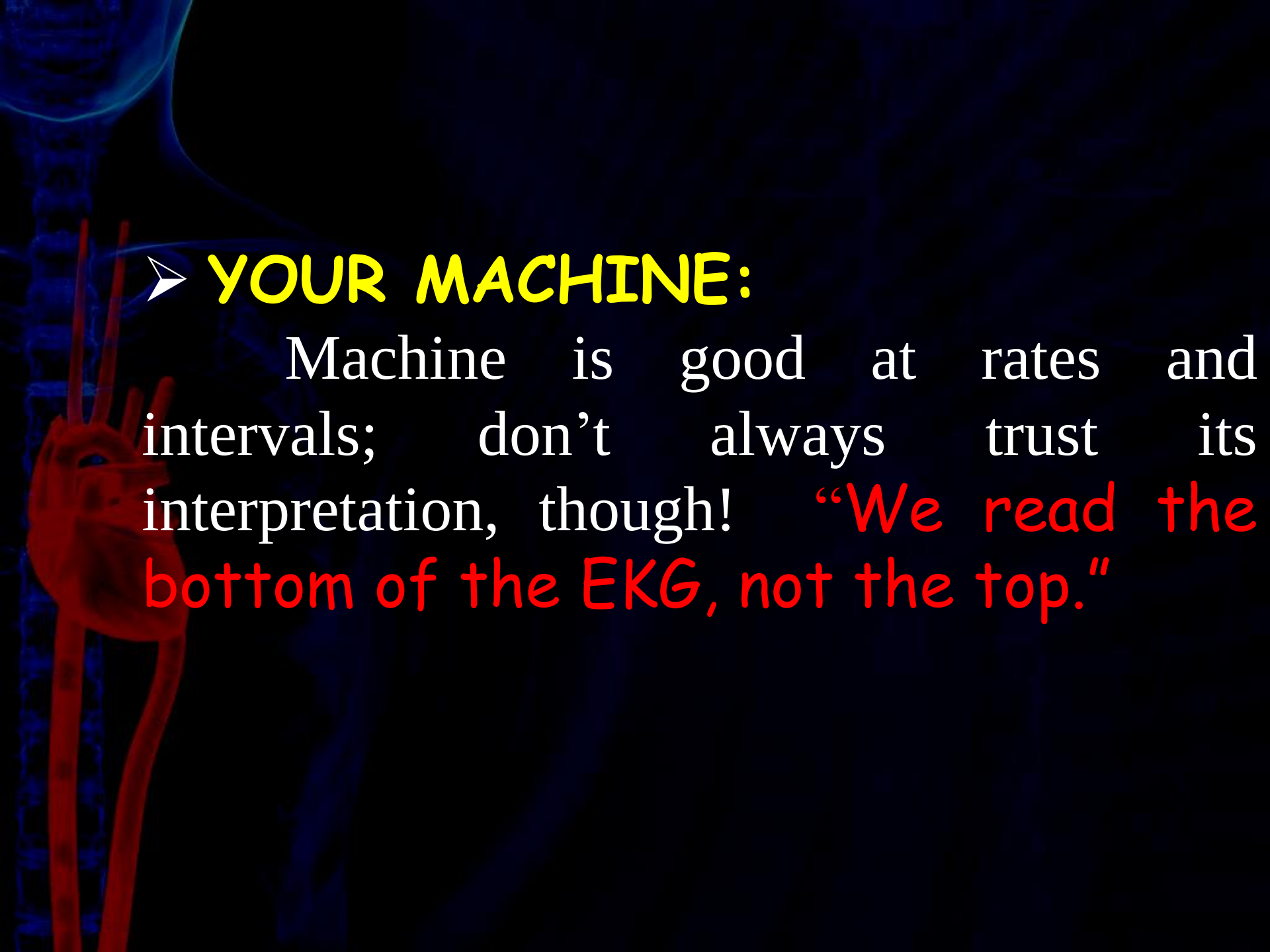
-If stable >>> 1- stop and think.

2- Call for help from your supervisor or staff.

3- Check code labs (CBC, CMP, Mg/Phos, cardiac enzymes, blood cx) and get a 12 lead EKG.

- **YOURSELF:**

If unsure, call a code—you'll get help fast, & the ICU team would rather have a "fake code" than a too-late code.



- **YOUR MACHINE:**

  Machine is good at rates and intervals; don't always trust its interpretation, though! "We read the bottom of the EKG, not the top."

➢ YOUR COLLEAGUE :

The best one who deal with arrhythmia is a cardiologist >> stabilize the patient and call me SOOOOON.


Thank You!