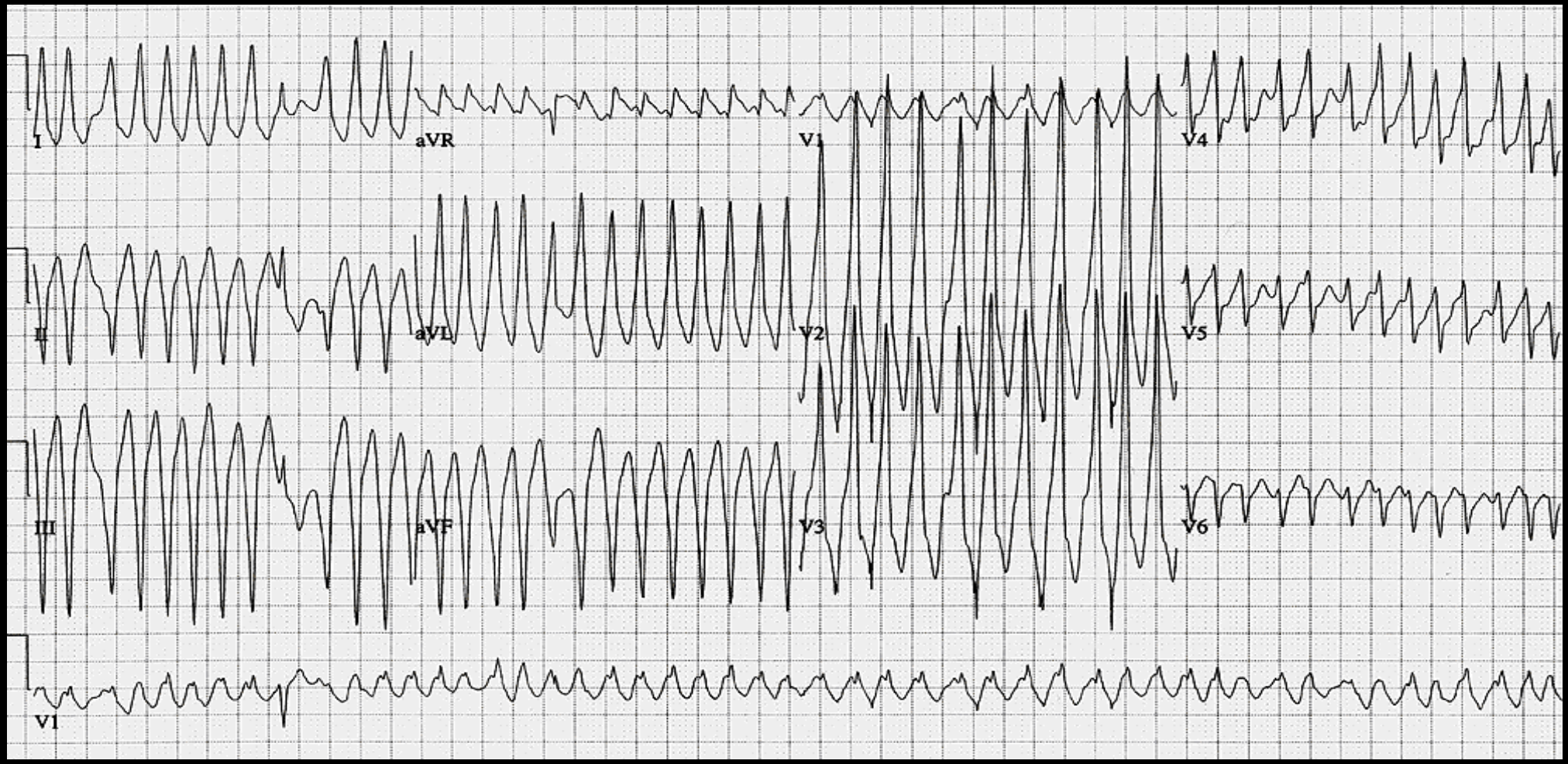
Patient with SVT just given adenosine – 1) what is rhythm?, 2) what is Mx?



The GP refresher course
16 June 2012

P Boon Lim
Acting Consultant Cardiologist and Electrophysiologist
Clinical Lead Syncope Unit
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Holter shows AF – who to refer?

SUMMARY

Date of report 16/02/2012 14:22:53

Hook-up date :	**09/02/2012 17:59:00**	Min Sinus HR (☀/☽) :	**48 (48/49) at 18:54:20**
Duration :	**22:53**	Mean Sinus HR (☀/☽) :	**71 (72/69)**
Recorder n° :	**VIA09001099**	Max Sinus HR (☀/☽) :	**185 (185/144) at 10:53:18**
Duration analysed :	**22:52:29**	SDNN :	**202 ms**
Number of QRS :	**115421**	PNN50 :	**38.34 %**

Bradycardias :	0
Pauses :	0 ; RR max 1.51 seconds (7:00:52)
Relative pauses :	192 ; longest 1.51 seconds (7:00:52)
AF :	3 ; longest 37832 QRS, 6:29:03 at 97 min-¹ (4:22:03)
AF burden :	41% (09:28); Mean HR 102 min-¹ (48/231 min-¹)
ST	0

Ventricular events				Supraventricular events	
VPB :	285 (0%)	Templates :	1	SVPB :	883 (0%)
Couplets :	6	Triplets :	0	Couplets :	173
bigeminy :	4 ; longest 6.98 seconds (3:40:21)			Triplets :	45
VT :	0			SVPT :	31 ; longest 6 QRS at 112 min-1 (1:34:01)
IVR/AIVR :	0				

Criteria for analysis : Min VT HR120 min-¹, SVPB 25%, Pause 2.50s, Brady HR 40 min-¹, Tachy HR 120 min-¹, Getting up time 07:00, Bedtime 23:00,
The values in italics in the tables have been modified manually

CONCLUSION

Technical Report:

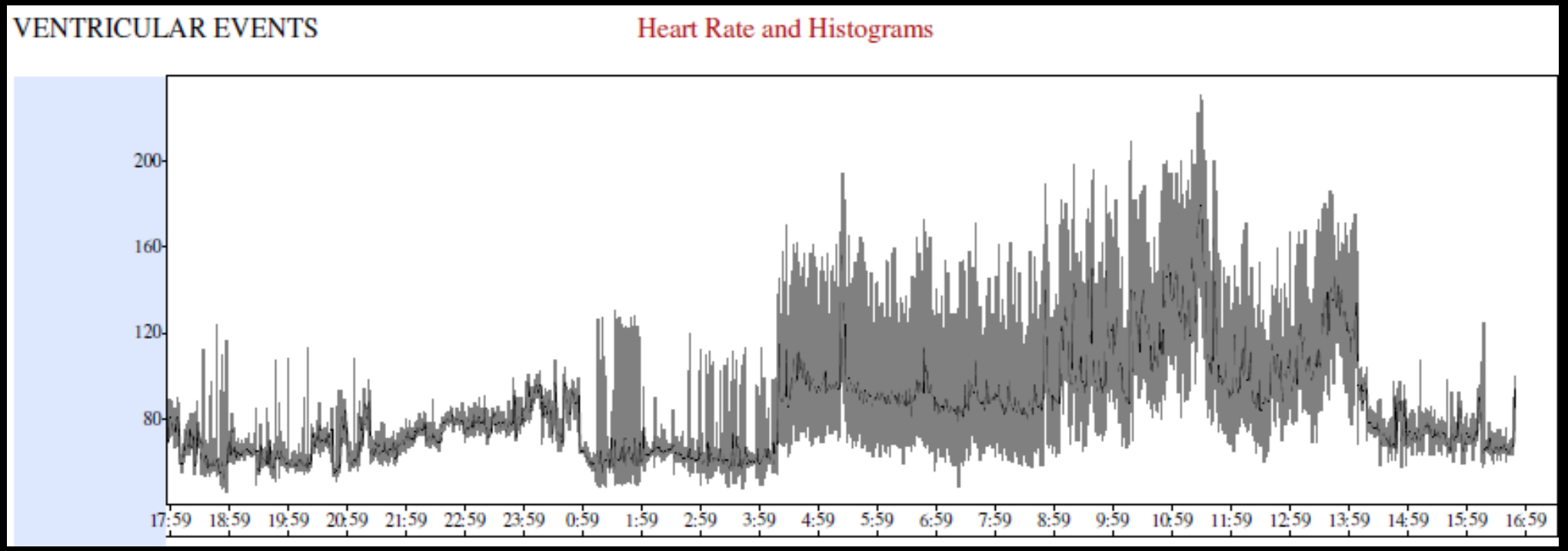
Sinus Rhythm with AF 41% of the duration of the recording (AF start 04:20:48 continues until 14:10:07)
Please see strips attached

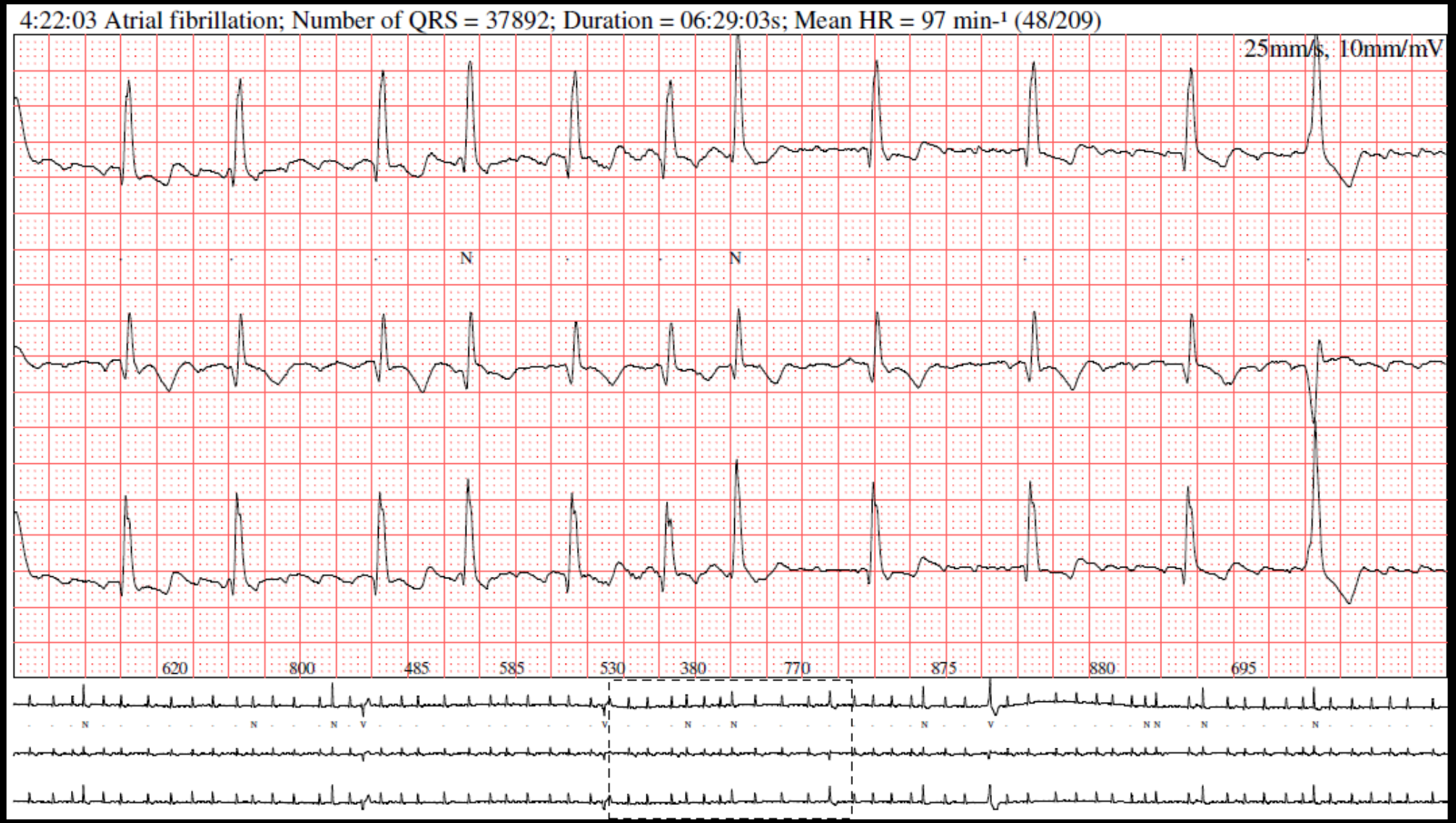
VE couplets x 6
VE singles x 260
VE bigeminy cycles x 4
SVE runs x 31
SVE triplets x 45
SVE couplets x 173
SVE singles x 225
SVE trigmeniy cycles x 6
SVE bigemeiny cycles x 1

Max HR: (AF) 231bpm at 11:31:52
Min HR: (SR) 48bpm at 18:54:20

No diary events entered

Sinus Rhythm with AF 41% of the duration of the recording (AF start 04:20:48 continues until 14:10:07)
Please see strips attached




4:22:03 Atrial fibrillation; Number of QRS = 37892; Duration = 06:29:03s; Mean HR = 97 min-¹ (48/209)
25mm/s, 10mm/mV
N
N
620
800
485
585
530
380
770
875
880
695

Definitions

2012 HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation: recommendations for patient selection, procedural techniques, patient management and follow-up, definitions, endpoints, and research trial design

March 2012, Europace, Heart Rhythm, J Interv Cardiac Electrophysiol

Paroxysmal	:	spontaneous termination <7 days
Persistent	:	AF > 7 days
Longstanding Persistent	:	AF > 1 year
Permanent	:	"State of mind" of physician/patient - acceptance of long term AF
Caveat, Post "early" DCCV	:	within 48h (Paroxysmal)
	:	> 48 hours (Persistent)

Case scenario 1

- 76 year old lady with HTN, DM, with persistent AF for 5 years and NYHA 2. Echo shows mildly impaired LV and LA size of 4.9cm. What is optimal management?

Case scenario 2

- 45 year old man with palpitations weekly, lasting up to 3 hours – unable to work during this time as highly symptomatic. Normal LV and LA 3.6cm. No underlying heart disease. ? Management

Case scenario 3

- 66 yo man with persistent AF for 12 months, LA size 4.6, Normal LV. Hypertensive. Fatigued but thinks it's "old age" catching up with him. ? Management

Management

Questions.

1. Rate vs Rhythm?
2. Anticoagulate or not?

Mechanisms of AF

Paroxysmal

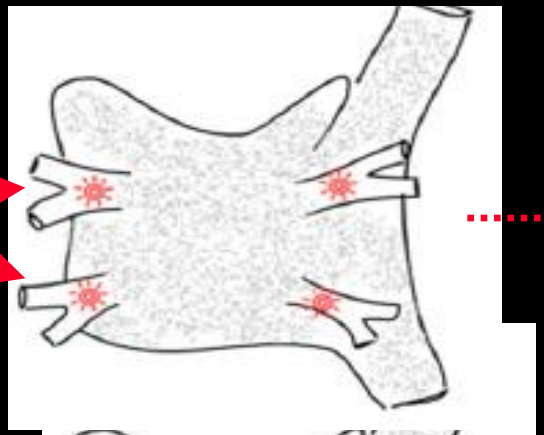
Persistent

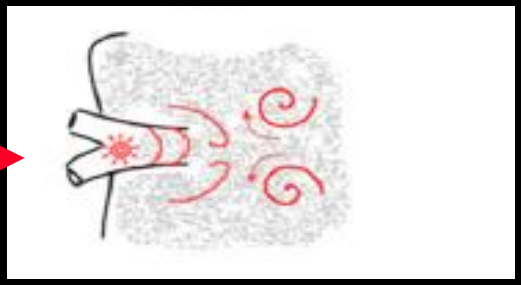
Permanent

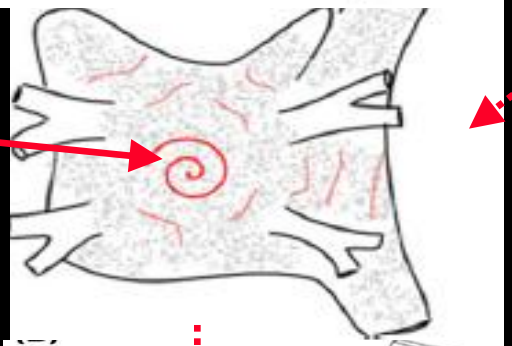
Focal Triggers

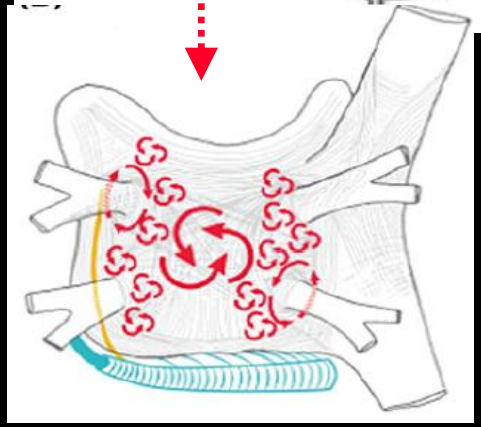
Re-entrant circuit

Multiple re-entrant circuits









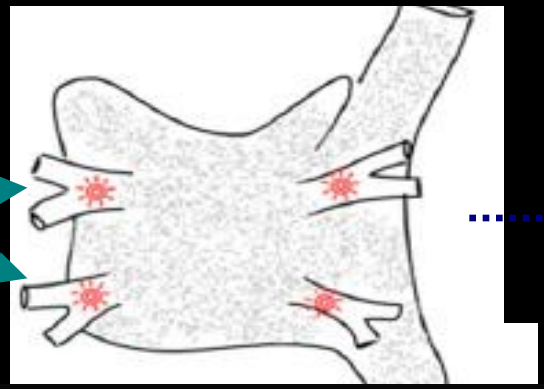
Rational for AF ablation – elimination of triggers

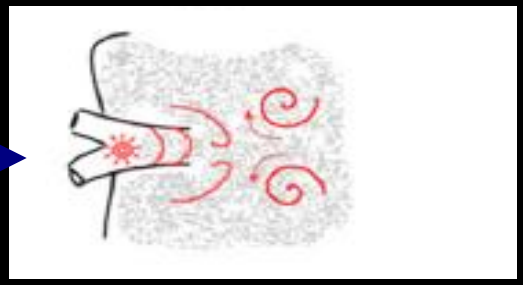
Paroxysmal

Persistent

Permanent

Focal Triggers

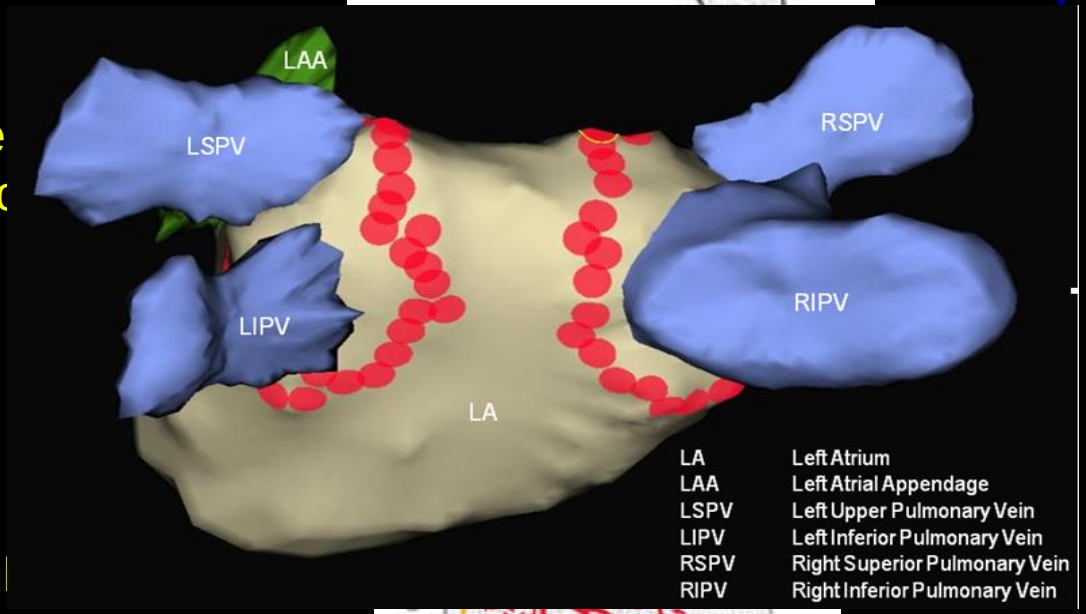


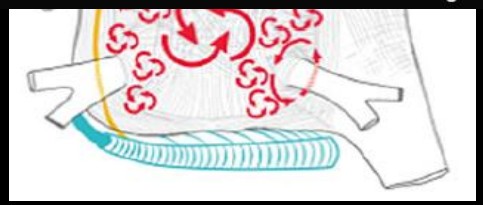


Re

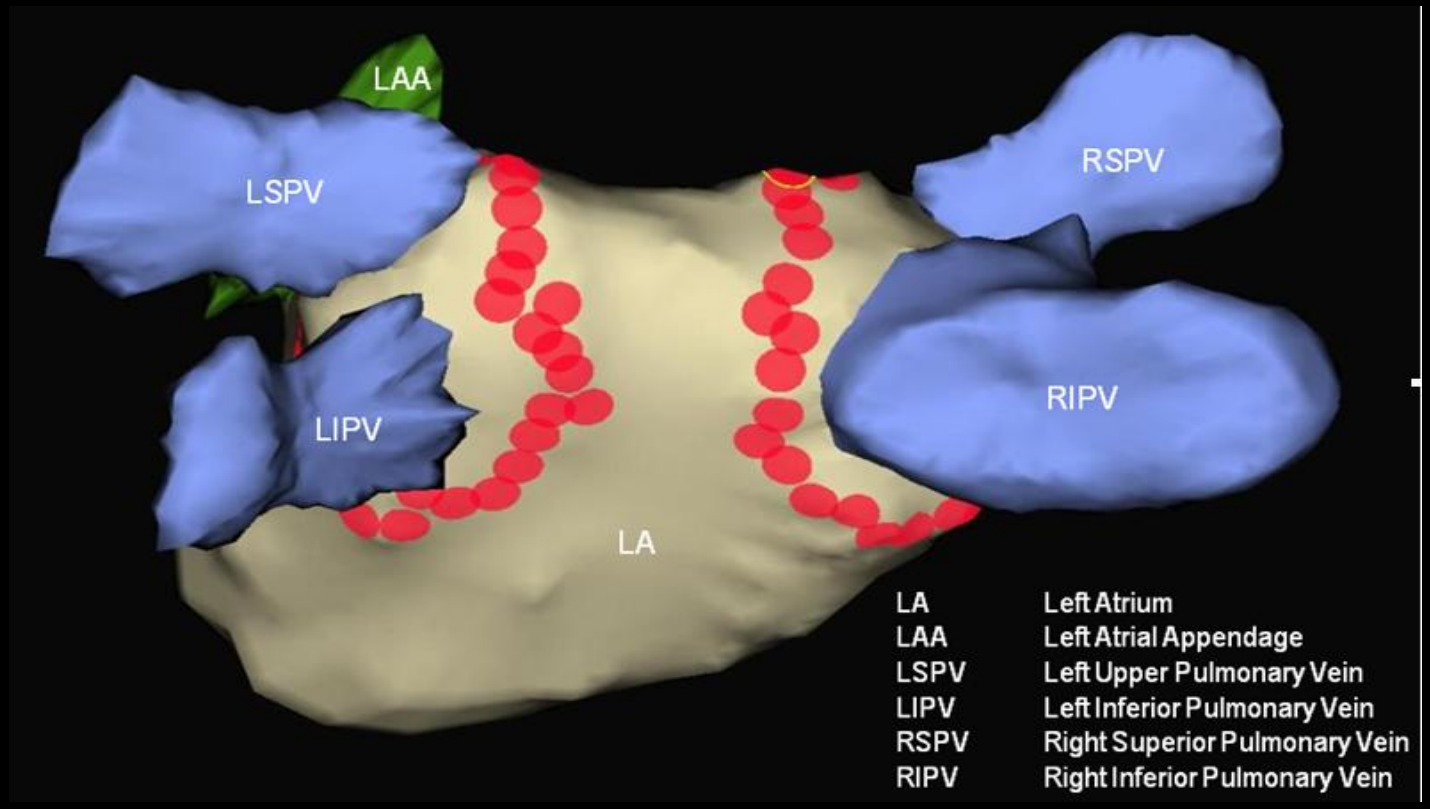
Mu

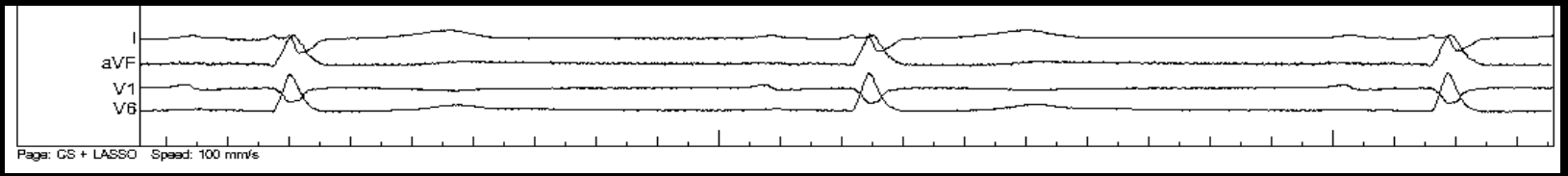
entrant circuits



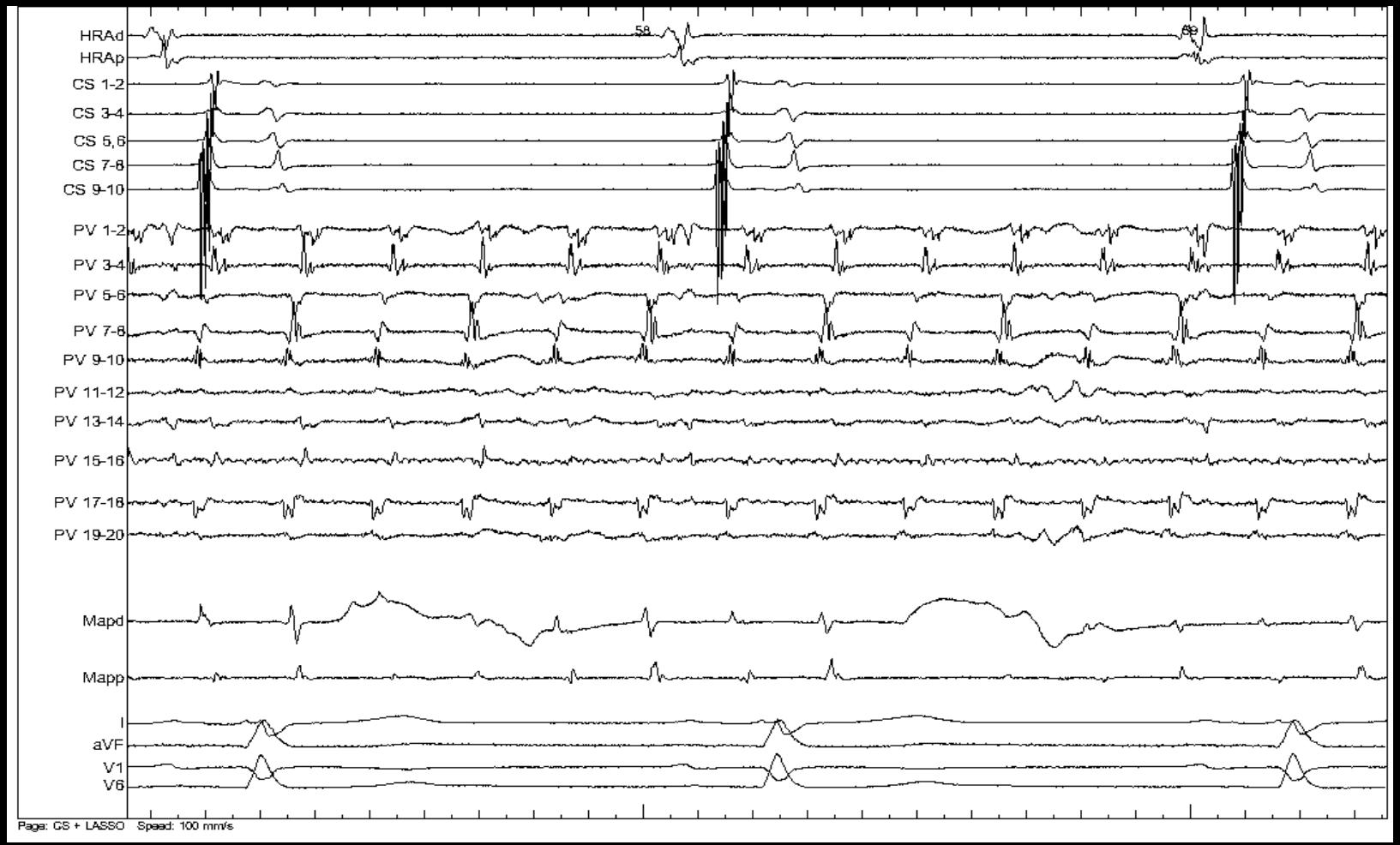


RUPV tachycardia in presence of SR after WACA





RUPV tachycardia in presence of SR after WACA



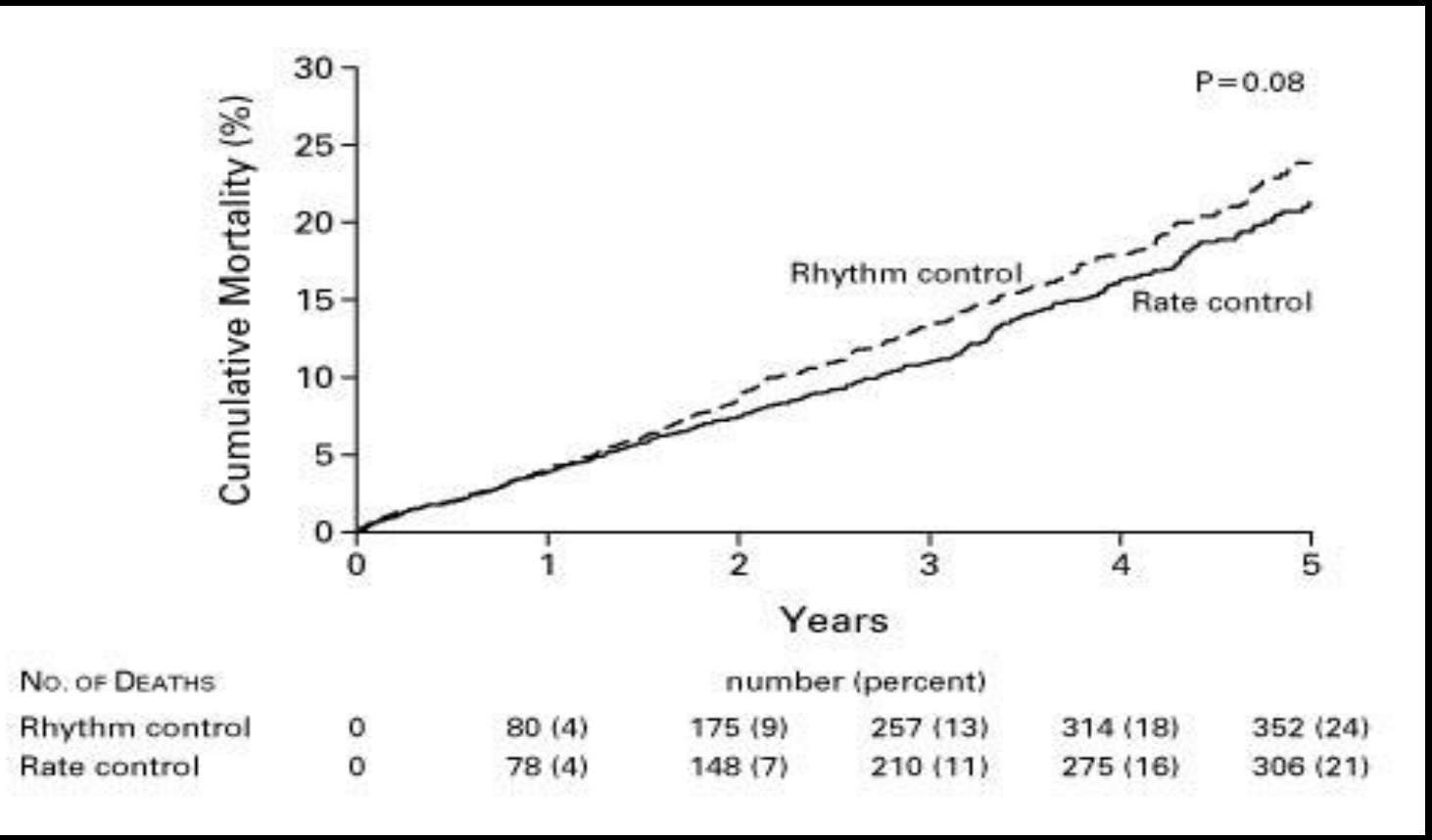
Rate vs rhythm

- **Decision based on**
  1. Symptoms, despite best drug therapy
  2. Likelihood of achieving sinus rhythm
     - » LA size < 5cm
     - » Duration of AF < 4 years
     - » Younger age
     - » Less atrial fibrosis (i.e. CMR imaging)

- **Options for treatment are**
  - » Anti arrhythmics: amiodarone, flecainide, propafenone, Sotalol
  - » AV nodal blocking: Digoxin, beta blockers, calcium antagonists
  - » DCCV – even if only to assess symptoms
  - » Ablation (earlier – and evolving indications)

Calkins et al. HRS Expert Consensus Statement, Heart Rhythm 2012

AFFIRM Study
Rate control versus anticoagulation in 4,000 patients



No. of Deaths		number (percent)				
Rhythm control	0	80 (4)	175 (9)	257 (13)	314 (18)	352 (24)
Rate control	0	78 (4)	148 (7)	210 (11)	275 (16)	306 (21)

AFFIRM NEJM 2002

Rate or rhythm? Medical Rx

- Maintenance of SR regardless of means associated with much better outcome
  - DIAMOND
    - » 506 patients with heart failure & AF
    - » Randomised to dofetilide vs. placebo
    - » Restoration of SR regardless of means was associated with a >50% reduction in mortality (after adjusting for covariates) (RR, 0.44; 95% CI, 0.30 to 0.64; p<0.0001)
  - AFFIRM sub-study
    - » Currently available AADs are not associated with improved survival, suggesting that any beneficial antiarrhythmic effects are offset by their adverse (?proarrhythmic) effects
    - » Sinus rhythm and warfarin use associated with reduced mortality

Circulation 2001; 104:292-296

Circulation 2004;109:1509-1513

Anti-coagulation

- Assessment of stroke risk, independent of arrhythmia
- CHADS2 and CHADSVASc score validated.
- Options for treatment
    1. Aspirin
    2. Warfarin
    3. Newer agents (dabigatran, rivaroxaban ,apixaban)
    4. Left atrial appendage closure devices.

What is the risk of CVA with AF?

- CHADS2
  - Congestive Heart Failure
  - Hypertension
  - Age >75yo
  - Diabetes
  - Previous CVA/TIA (scores 2)
- Score range 0-6

- CHADSVASc
  - Congestive Heart Failure
  - Hypertension
  - Age >75yo (scores 2)
  - Diabetes
  - Previous CVA/TIA (scores 2)
  - ADD IN 3 points
  - Sex (F scores 1)
  - Age >65 (scores 1)
  - Vascular disease (1)

Score range 0-9, being female =1

JAMA 2001

CHADS2 score

Score	Risk of Stroke Per Year
0	1.9%
1	2.8%
2	4.0%
3	5.9%
4	8.5%
5	12.5%
6	18.2%

Multiply CHADS2 score x2 and this is your rough estimate of annual CVA risk (unless full score = 18%). – starts at 2!

OR

2,3,4,6,8,12,18

JAMA 2001

Anticoagulation

- Key points

1. Decision to anti-coagulate made on risk factors for CVA alone, not on rate/rhythm strategy

2. Consideration of bleeding risk – often elderly who benefit from anticoagulation, also have highest risk of CVA

3. Evolving field, with new data/strategies for minimising CVA i.e new drugs, left atrial appendage closure

Case scenario 1

- 76 year old lady with HTN, DM, with persistent AF for 5 years and NYHA 2. Echo shows mildly impaired LV and LA size of 4.9cm. What is optimal management?

- Rate/Rhythm : unlikely to maintain SR (duration > 5 y, LA size almost 5.0cm, HTN) whatever the strategy. Likely therefore to assign “permanent AF” state. For rate control (including ? AV nodal ablation + pace)

- CVA risk: CHADS2 = 3 (i.e approx 6% annnual CVA risk), therefore for full anticoagulation.

- Heart failure management : ACE, Beta blockers, frusemide, etc.

Case scenario 2

- 45 year old man with palpitations weekly, lasting up to 3 hours – unable to work during this time as highly symptomatic. Normal LV and LA 3.6cm. No underlying heart disease. ? Management?

- Rate/Rhythm : likely to maintain SR (PAF, duration unspecified, LA size normal). For rhythm control.
  - Drugs vs ablation?
    - 8 prospective randomised trials now comparing success rates
      - AAD (9-40%) vs Ablation (66-89%)
    - Improved QOL in ablation arms in all trials

- CVA risk: CHADS2 = 0 (low annual CVA risk), not for anticoagulation

Calkins et al. HRS Expert Consensus Statement, Heart Rhythm 2012

Case scenario 3

- 66 yo man with persistent AF for 12 months, LA size 4.6, Normal LV. Hypertensive. Fatigued but thinks it's "old age" catching up with him. ? Management

- Rate/Rhythm : ? likely to maintain SR (duration 12 months, LA size 4.6, large but not too large for ablation, ? symptomatic).
  - Start on AAD
  - Consider DCCV with full anticoagulation to assess symptoms
    - » If symptomatic with AF, and failed AAD therapy, can have AF ablation.

- CVA risk: CHADS2 = 1 (HTN), medium risk. "Grey area", but would opt for full anticoagulation if ablation is considered.

- CHADS2 score evolve with time: Old patients get older! / HTN / DM / HF

Holter shows VT – who to refer?

Case of 56 yo woman with HTN and obesity only

REFER?

SUMMARY *Date of report 19/12/2011 15:46:19*

Hook-up date :	**16/12/2011 18:25:00**	Min Sinus HR (☀/☽) :	**62 (66/62) at 4:27:20**
Duration :	**24:00**	Mean Sinus HR (☀/☽) :	**86 (90/78)**
Recorder n° :	**VIP10000894**	Max Sinus HR (☀/☽) :	**129 (129/122) at 12:09:53**
Duration analysed :	**23:39:42**	SDNN :	**90 ms**
Number of QRS :	**119242**	PNN50 :	**0.60 %**

Bradycardias :	0
Pauses :	0 ; RR max 1.35 seconds (5:58:52)
Relative pauses :	0
AF :	0
AF burden :	
ST	0

Ventricular events				Supraventricular events	
VPB :	390 (0%)	Templates :	1	SVPB :	19 (0%)
Couplets :	1	Triplets :	0	Couplets :	1
bigeminy :	0			Triplets :	0
VT :	2 ; longest 44 QRS at 179 min-1 (1:29:58)			SVPT :	2 ; longest 5 QRS at 134 min-1 (21:21:18)
IVR/AIVR :	0				

Criteria for analysis : Min VT HR120 min-¹, SVPB 25%, Pause 2.50s, Brady HR 40 min-¹, Tachy HR 120 min-¹, Getting up time 07:00, Bedtime 23:00,
The values in italics in the tables have been modified manually

CONCLUSION

Technical report: 72HR monitor day 2

Sinus rhythm with two episodes of VT runs

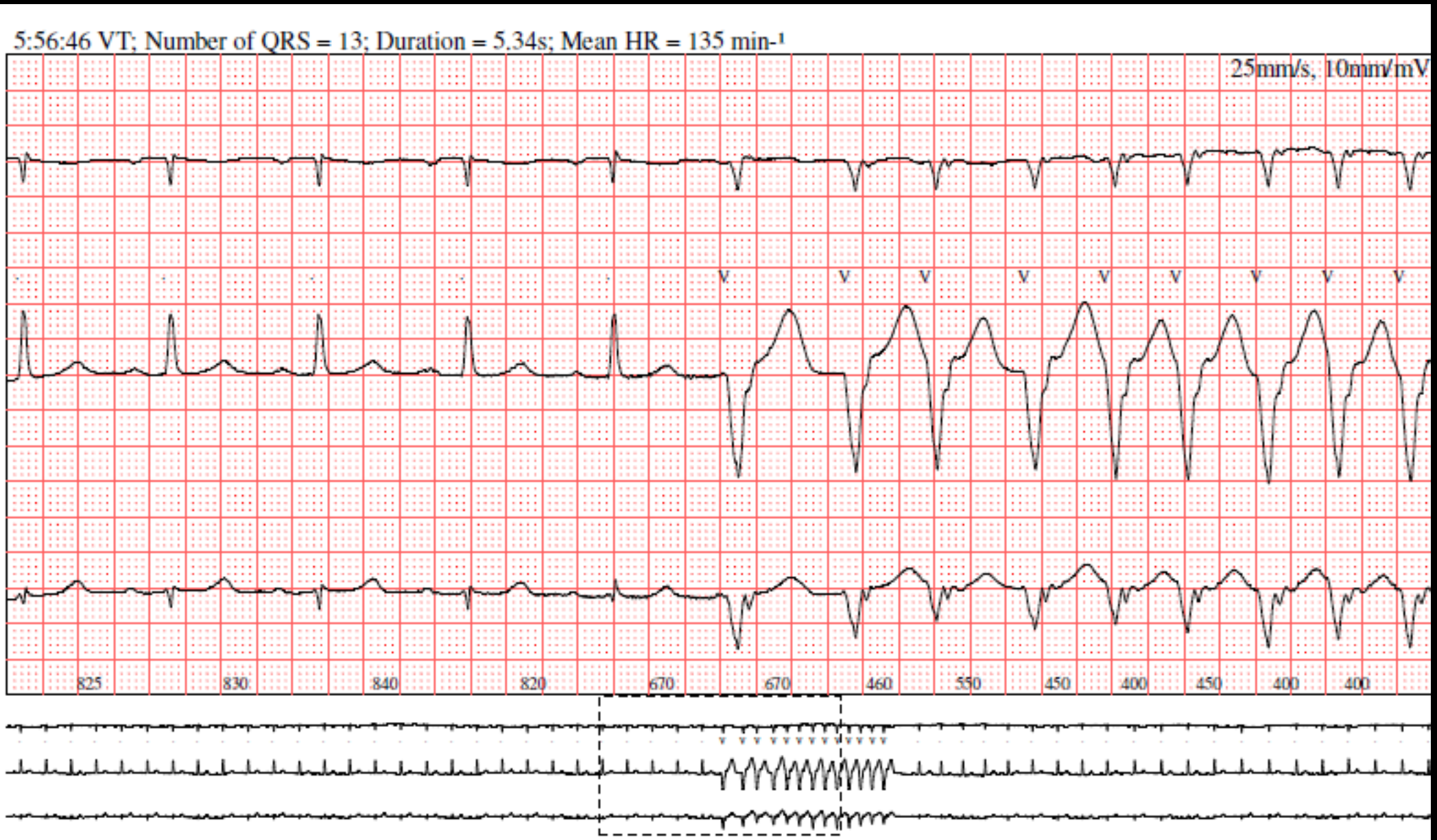
VT run of 13beats @ 135bpm at 05:56:46
Longest and fastest VT run of 44beats @217bpm at 01:30:00

VE singles 331
SVE runs x 2
SVE couplets x 2
SVE singles x 6

Max R-R: 1.35sec at 05:58:52

Max HR: 217bpm (VT) @ 1.:30:11
Max HR: 129bpm at 12:09:53
Min HR: 62bpm at 04:27:20

No diary retunred


5:56:46 VT; Number of QRS = 13; Duration = 5.34s; Mean HR = 135 min-1
25mm/s, 10mm/mV
825
830
840
820
670
670
460
550
450
400
450
400
400

Management of non-sustained BCT

- **Refer** if uncertain
- If VT + syncope → will need consideration for ICD
- Fhx sudden death is relevant.
- IHD = high likelihood that this is ischaemic VT (commonest)
- If no IHD,Will need work up for Arrhythmogenic right ventricular cardiomyopathy, Brugada, Hypertrophic cardiomyopathy, Long QT syndrome, Early repolarisation syndromes
- At tertiary centre : thorough Hx, CMRI, ajmaline and adrenaline challenge, VT stimulation study, Reveal device,

Holter shows VT – who to refer?

Case of 30 yo woman with palps, but no syncope. Normal echo

REFER?

Hook-up date :	**03/05/2012 14:41:00**	Min Sinus HR (☀/☽) :	**55 (56/55) at 6:49:18**
Duration :	**24:00**	Mean Sinus HR (☀/☽) :	**82 (83/80)**
Recorder n° :	**VIP10000899**	Max Sinus HR (☀/☽) :	**127 (127/111) at 8:13:03**
Duration analysed :	**23:07:38**	SDNN :	**98 ms**
Number of QRS :	**113109**	PNN50 :	**18.90 %**

Bradycardias :	0
Pauses :	0 ; RR max 1.36 seconds (23:43:31)
Relative pauses :	0
AF :	0
AF burden :	
ST	0

Ventricular events				Supraventricular events	
VPB :	*12027 (10%)*	Templates :	1	SVPB :	*98 (0%)*
Couplets :	0	Triplets :	0	Couplets :	2
bigeminy :	31 ; longest 11.24 seconds (10:49:22)			Triplets :	1
VT :	0			SVPT :	0
IVR/AIVR :	0				

Criteria for analysis : Min VT HR120 min-¹, SVPB 25%, Pause 2.50s, Brady HR 40 min-¹, Tachy HR 120 min-¹, Getting up time 07:00, Bedtime 23:00,
The values in italics in the tables have been modified manually

CONCLUSION

Technical Report:

Sinus Rhythm with frequent VE's

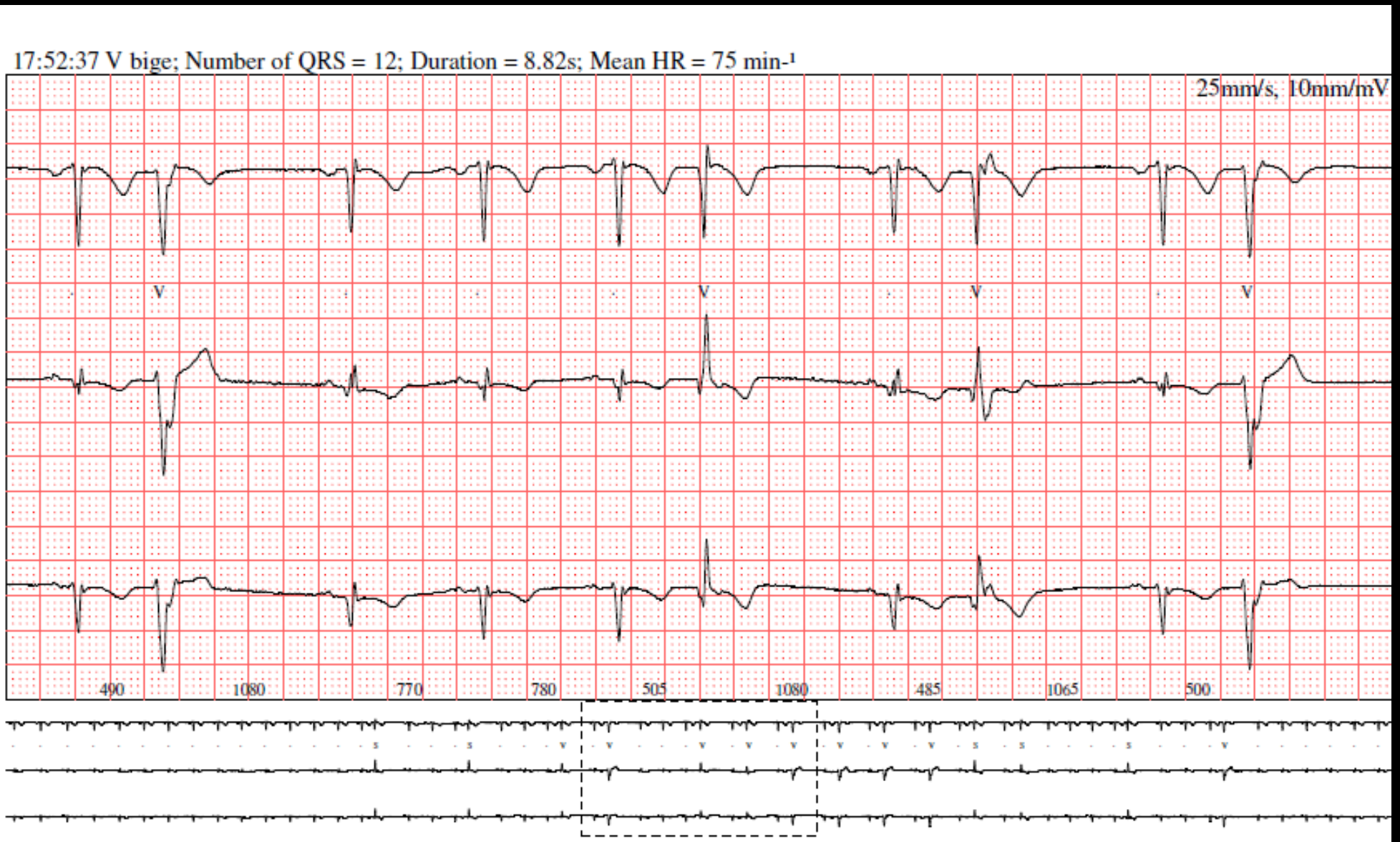
VE singles x 12027
Occasional VE trigeminy and bigeminy cycles

SVE triplets x 1
SVE couplets x 2
SVE singles x 98

Max R-R: 1.36sec at 23:43:31

Max HR: 127bpm at 08:13:03
Min HR: 55bpm at 06:49:18

No diary returned


17:52:37 V bige; Number of QRS = 12; Duration = 8.82s; Mean HR = 75 min-1
25mm/s, 10mm/mV
V
V
V
V
490
1080
770
780
505
1080
485
1065
500

Other clinical cases – the HOLTER report, what does it mean?

- Echo – Normal
- VE > 10,000, but no sustained VT

Challenging / Unanswered questions

- what is natural progression of disease?
- What burden of ectopy is considered “high” enough to warrant therapy?
- At what stage do you start therapy?
- Drugs or ablation ?

The GP refresher course
16 June 2012

P Boon Lim
Acting Consultant Cardiologist and Electrophysiologist
Clinical Lead Syncope Unit
Imperial College Healthcare NHS Trust

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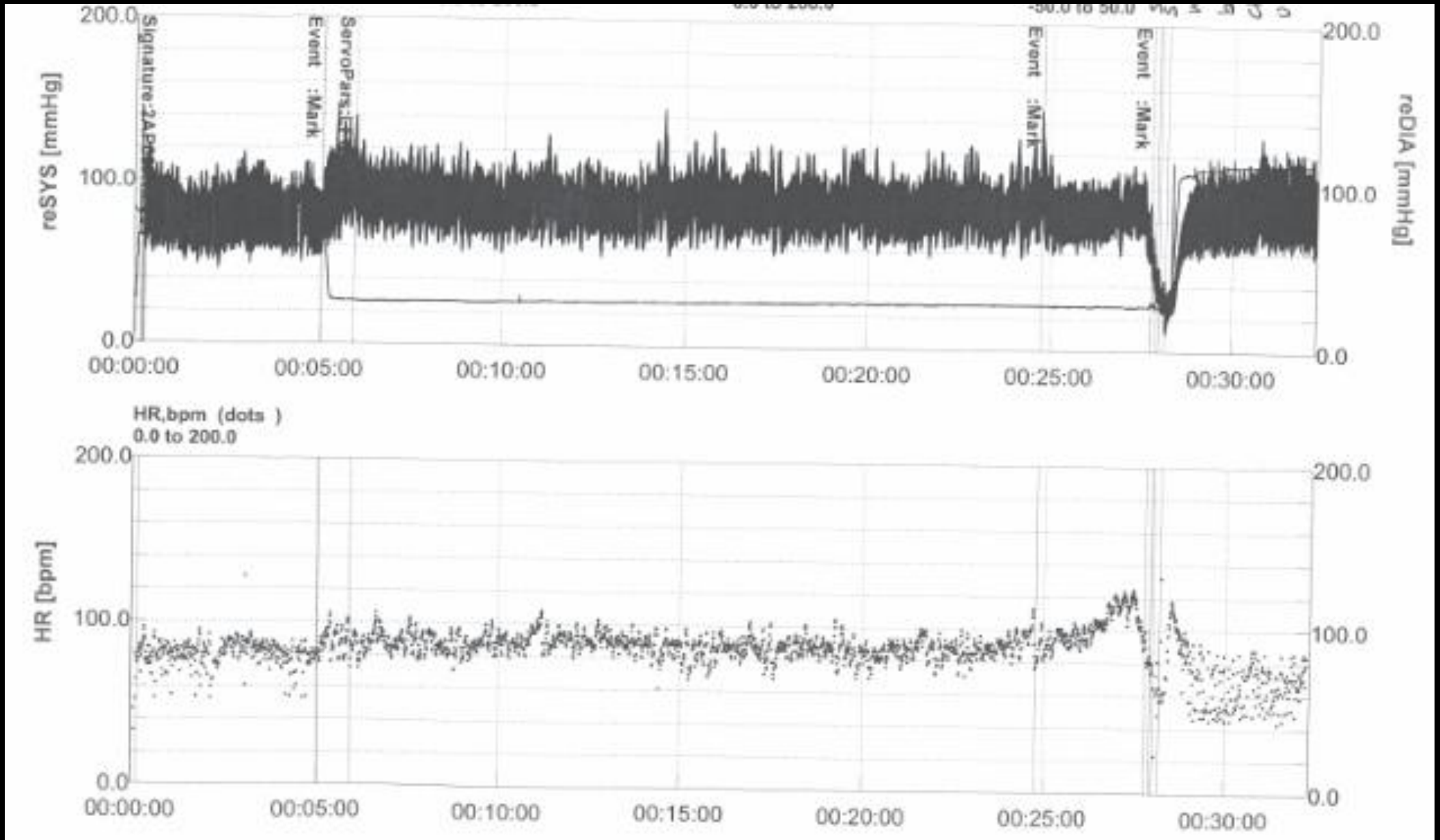
The tilt report – what does it mean?

- 32 yo lady with infrequent palpitations, followed by collapses with loss of consciousness. Sometimes feels lightheaded on standing
- Normal echo, normal 24 hour tape, normotensive



(C) 2002 TNO-TPD-BMI

03/04/2012 17:14:00

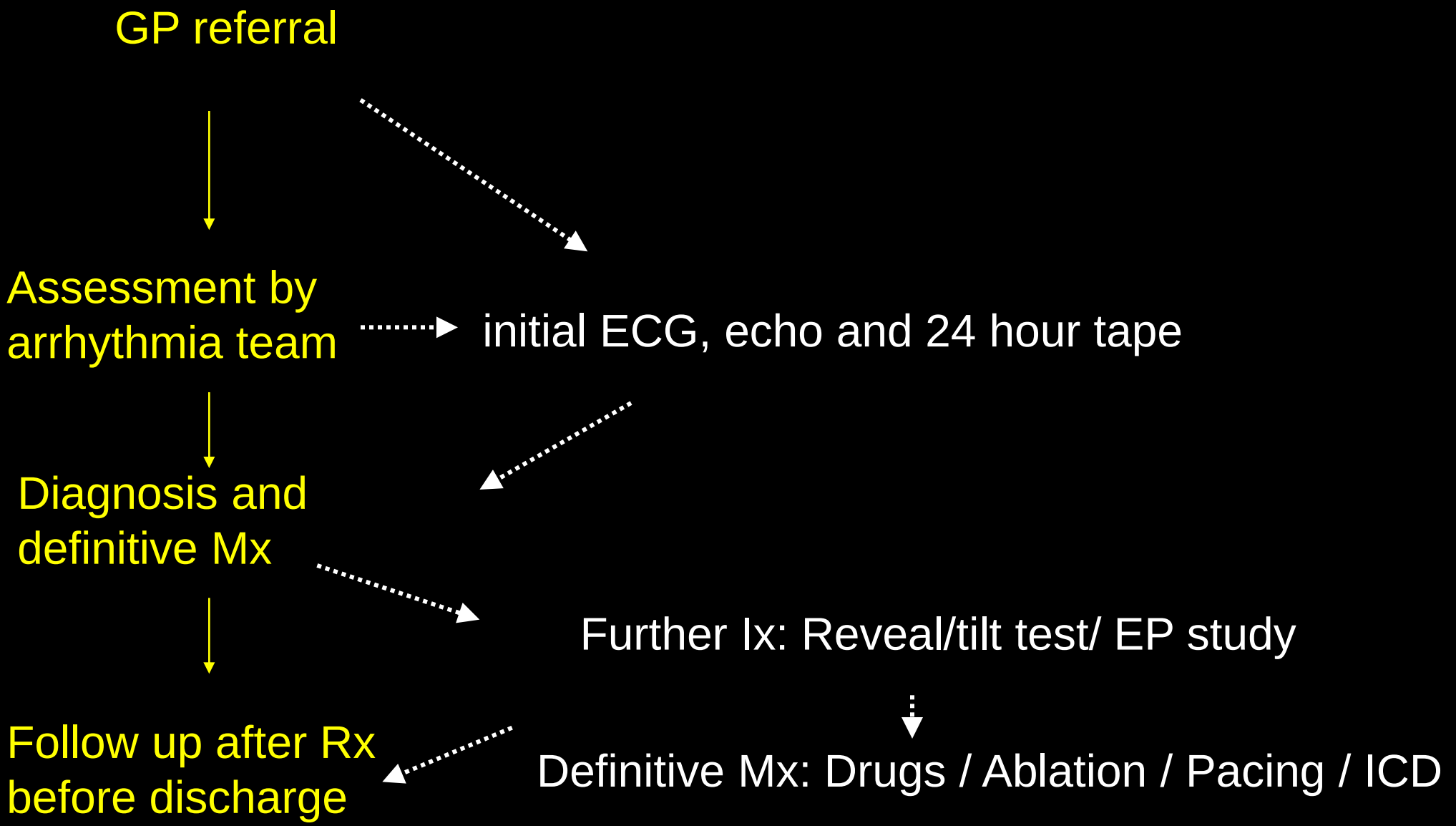
Report and management

- Vasovagal syncope with a VASIS 1 (mixed cardioinhibitory and vasodepressor pattern)

- Management is conservative first
  - Isometric counter-pressure manoeuvres
  - Increase fluids ± salt
  - Evasive action
  - Regular meals
  - Avoid caffeine
  - Pharmacotherapy 2nd line (midodine, ivabradine, fludrocortisone, salt tablets)
  - PPM? ILR? Ablation??

The rapid access arrhythmia clinic

- Aim: To provide access to specialist arrhythmia service with full investigations to support early Dx and Mx of arrhythmias

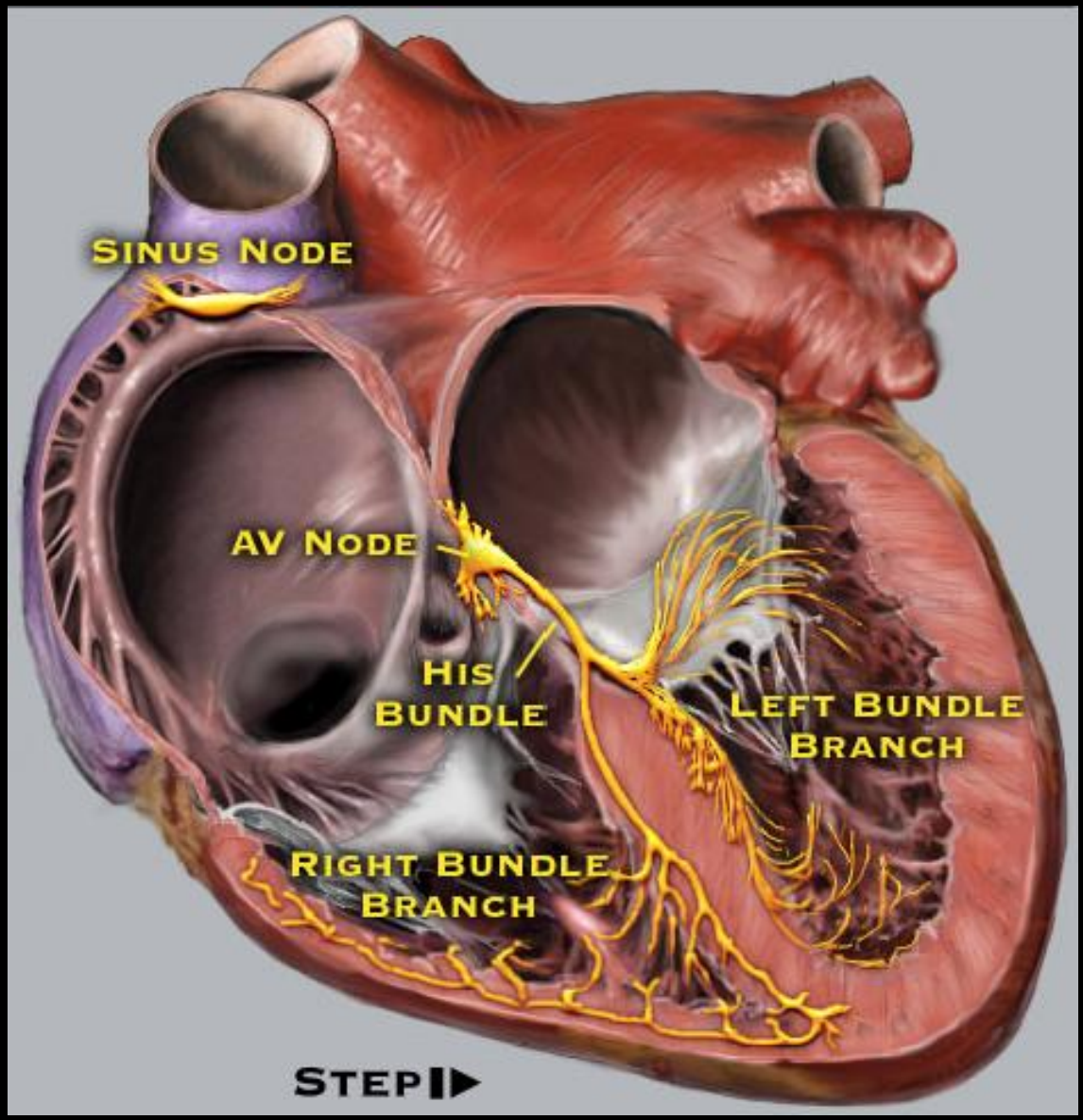

GP referral
Assessment by arrhythmia team
initial ECG, echo and 24 hour tape
Diagnosis and definitive Mx
Further Ix: Reveal/tilt test/ EP study
Definitive Mx: Drugs / Ablation / Pacing / ICD
Follow up after Rx before discharge

Imperial College Healthcare EP and Pacing Dept

- 6 EP and pacing consultants in academic unit
- 2 cath labs running high volume, >300 complex (AF/VT ablation), >700 total EP cases pa, and >300 pacing cases pa
- Syncope service, and clinic + tilt dept~ 600 head-up tilts pa
- Weekly outreach arrhythmia clinics in Lister + Stevenage, Teddington, Roehampton, Maida Vale, West Middlesex
- Local clinics in St Mary's, Charing Cross, and Hammersmith.
- Innovation, with first-in-man studies of several applications: Robotic catheter technology, cryoablation, 256 ECG jacket (ECGvue), novel multipoint electroanatomical mapping technology (Rhythmia, and Ripple mapping), syncope pacemaker algorithm development and trials (ISSUE3)

Anatomy of the conducting system - Bradycardia

Sinus bradycardia
Sinus arrest
Sick sinus syndrome
Carotid sinus hypersensitivity



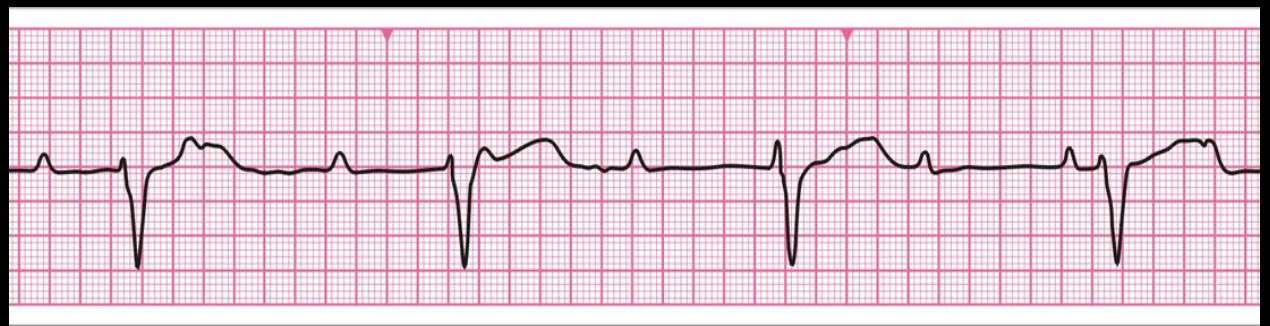
1st degree heart block

2nd degree heart block
- Wenkebach
- Mobitz II

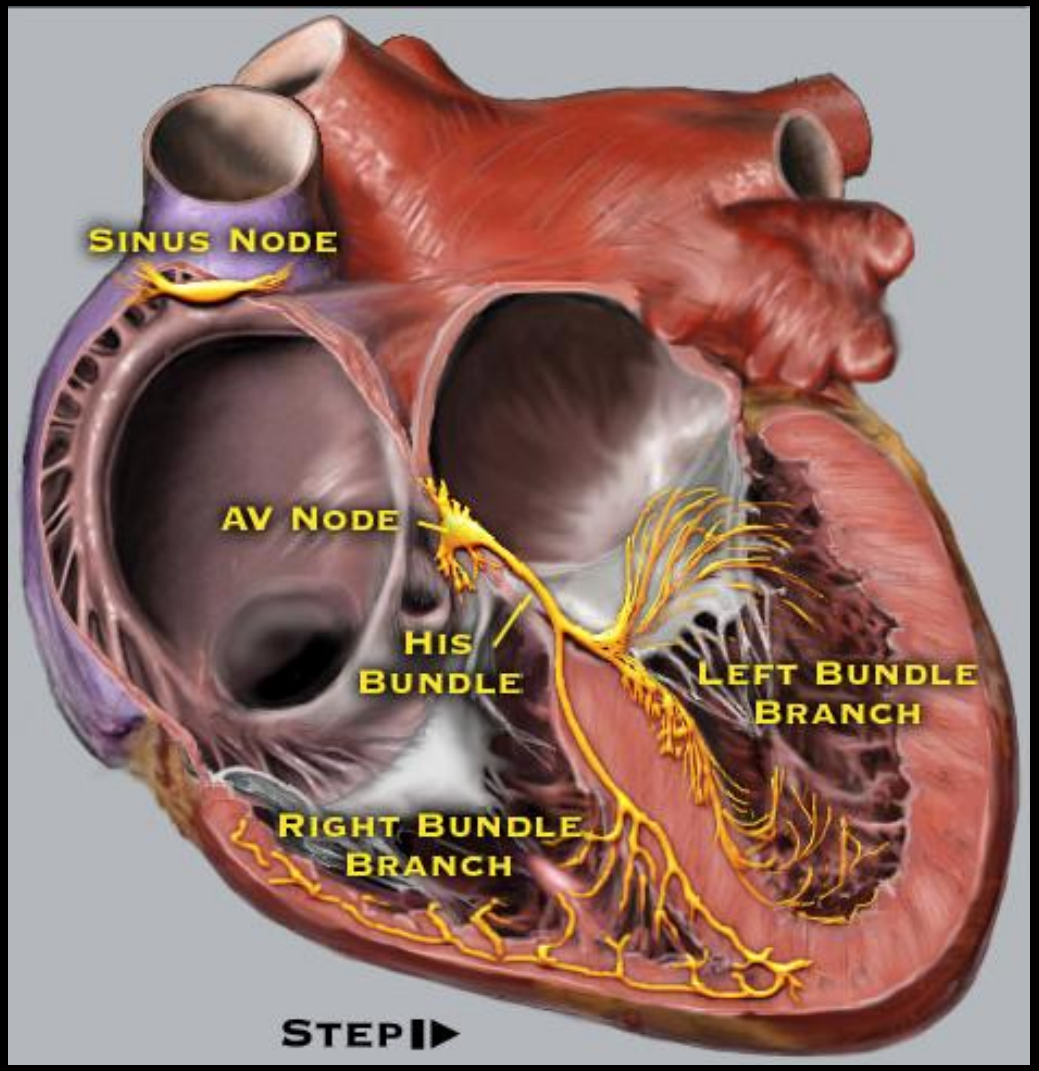
3rd degree heart block

Trifascicular block

Questions (for you)



Anatomy of the conducting system - Tachycardia



AV Node independent:

- Atrial fibrillation (AF)
- Atrial flutter
- Atrial tachycardia (AT)
- Sinus tachycardia
- Inappropriate sinus tachycardia
- Atrial premature beats

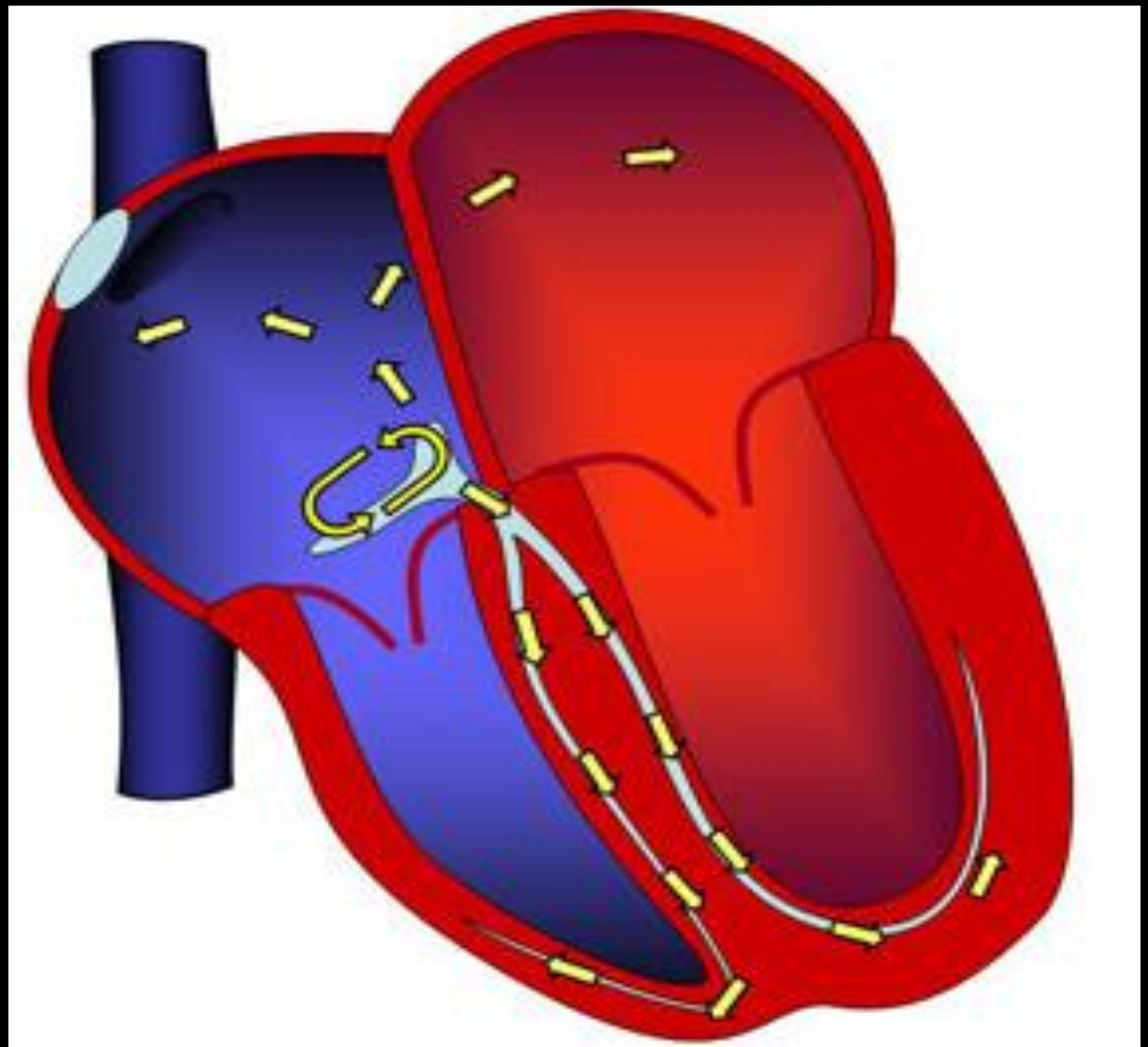
AV Node dependent:

- Atrio-ventricular nodal re entrant tachycardia (AVNRT)
- Atrioventricular re-entrant tachycardia (AVRT)

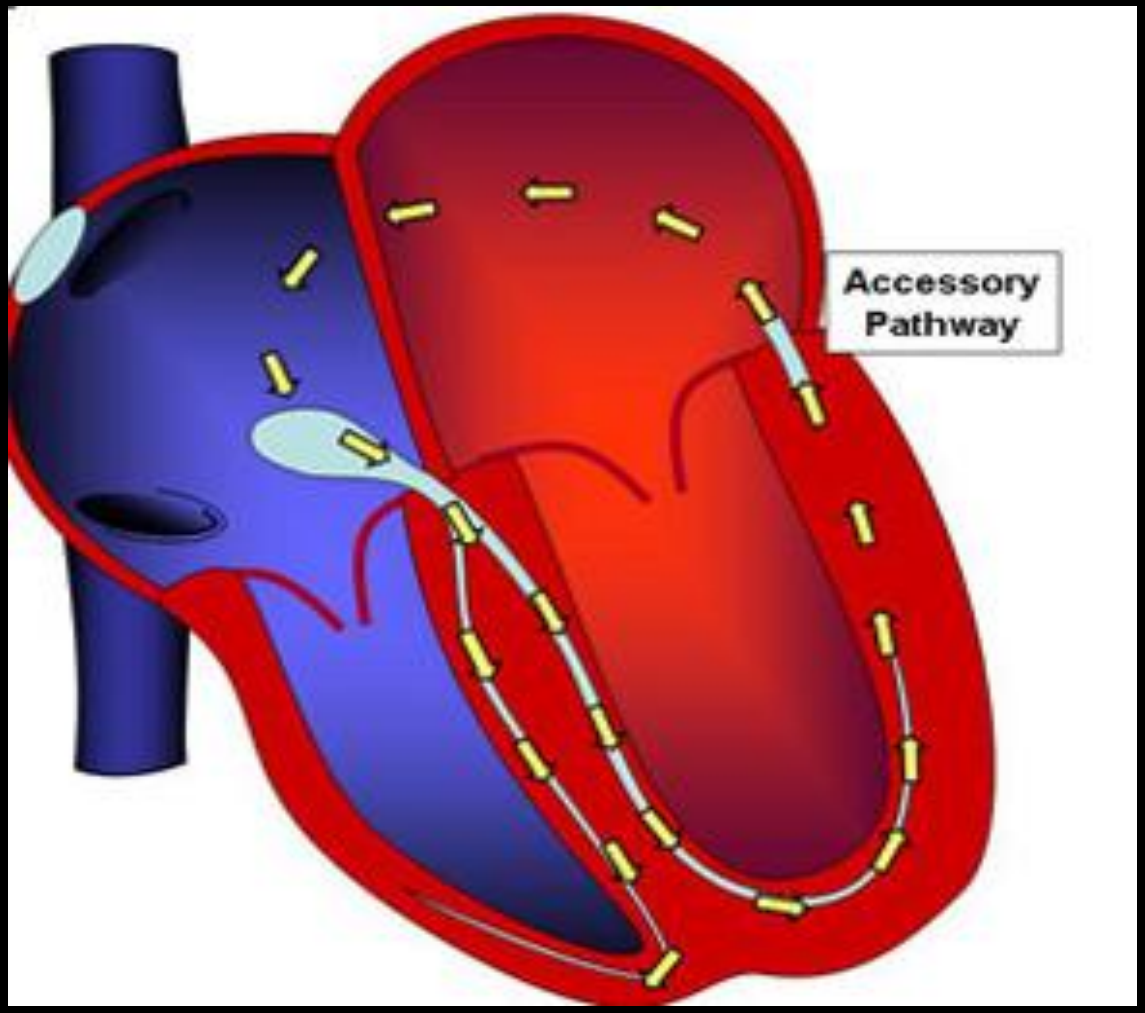
Ventricular

- Ventricular tachycardia (VT)
- Ventricular fibrillation

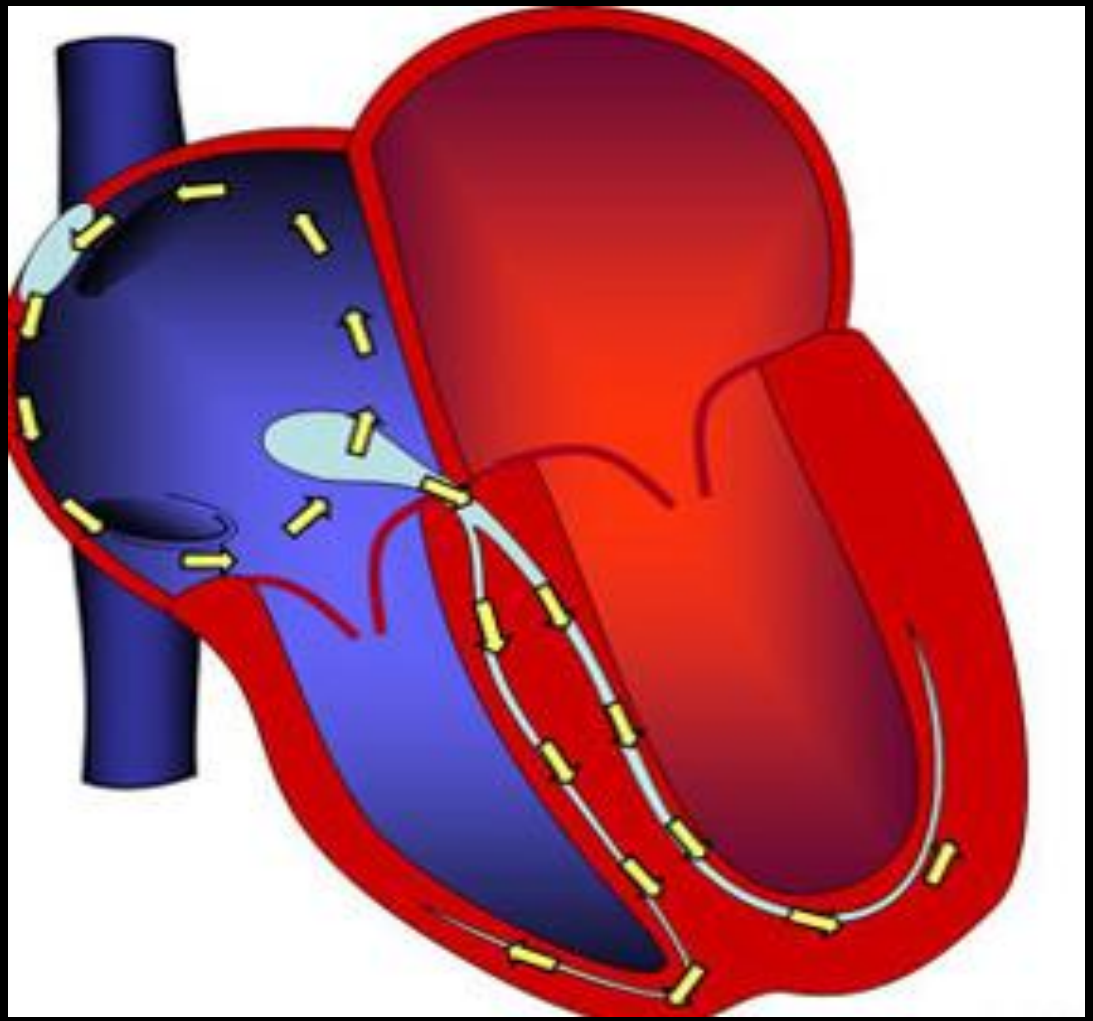
AVNRT



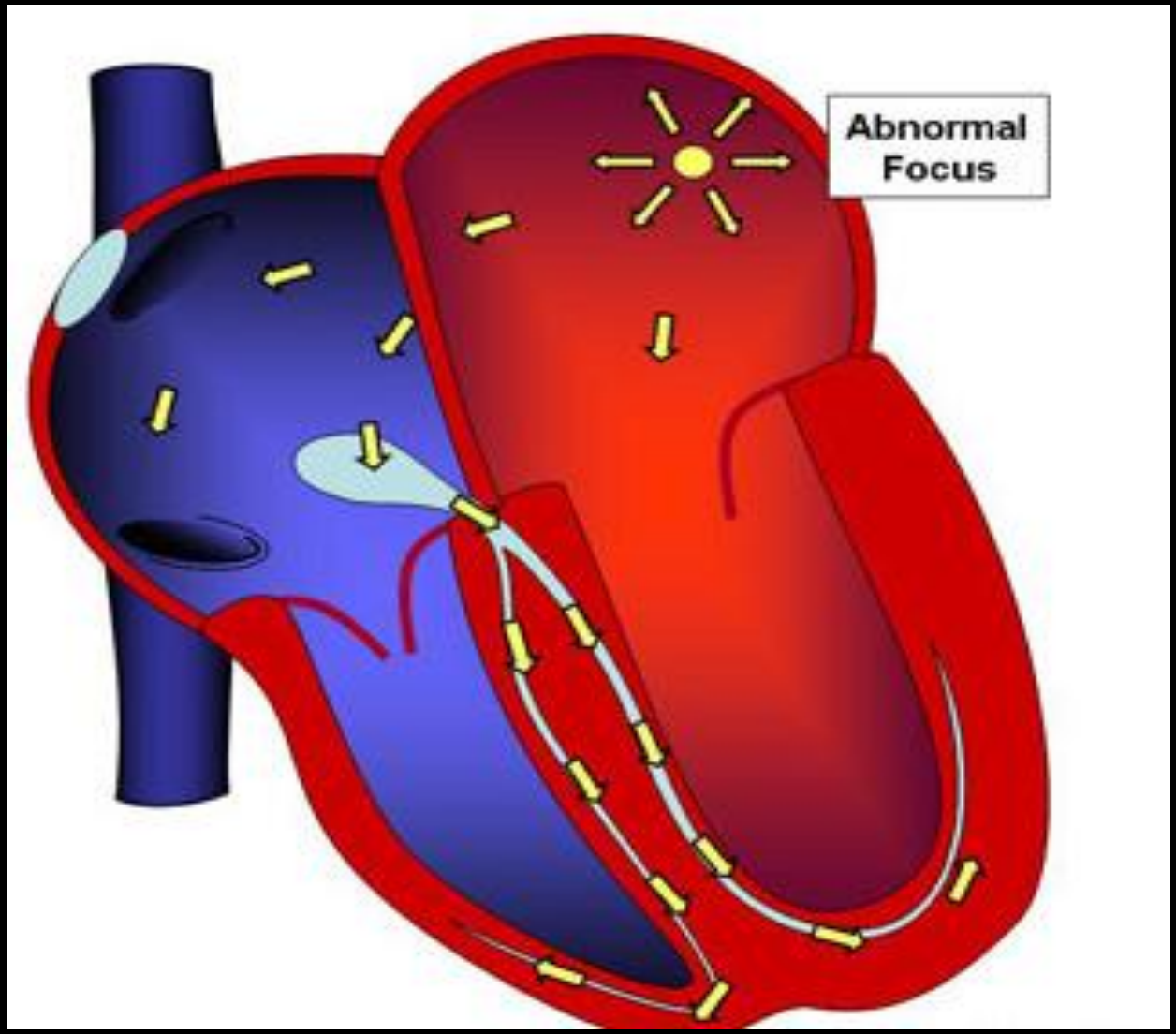
AVRT



Atrial flutter

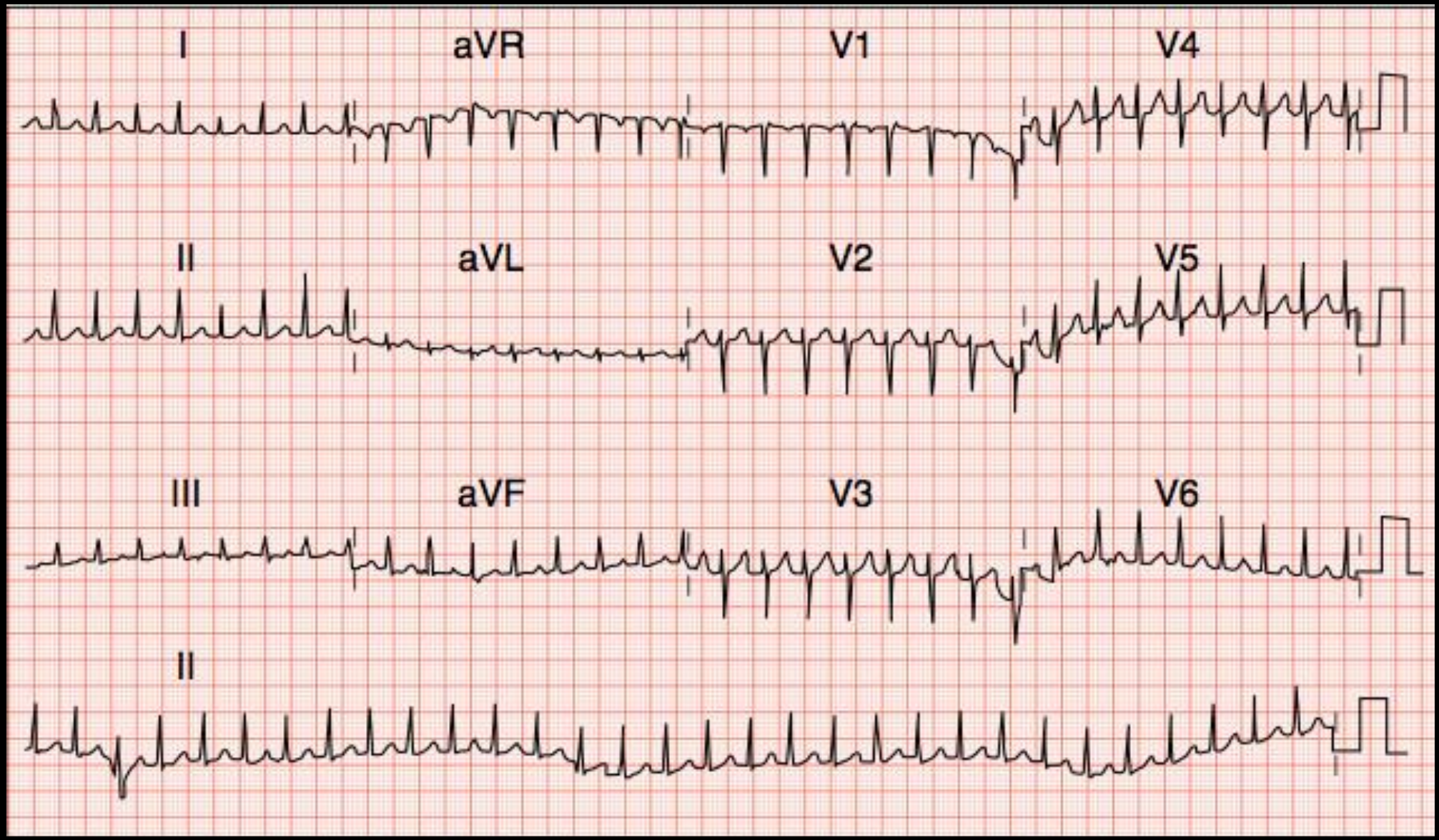


Atrial tachycardia



Question 1

- 45 yo lady with CP and palpitations
- Infrequent palpitations


I
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III
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V3
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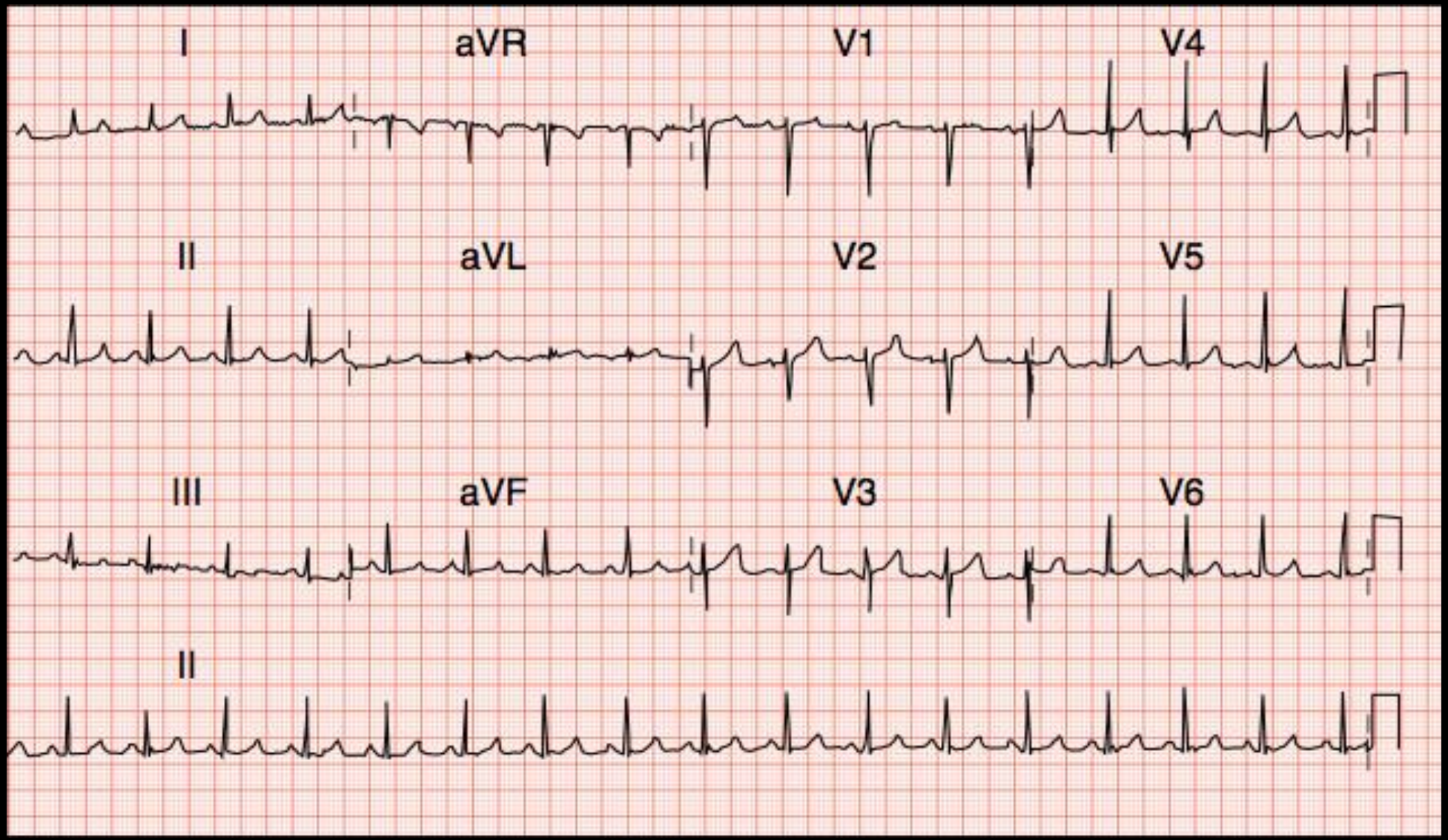
II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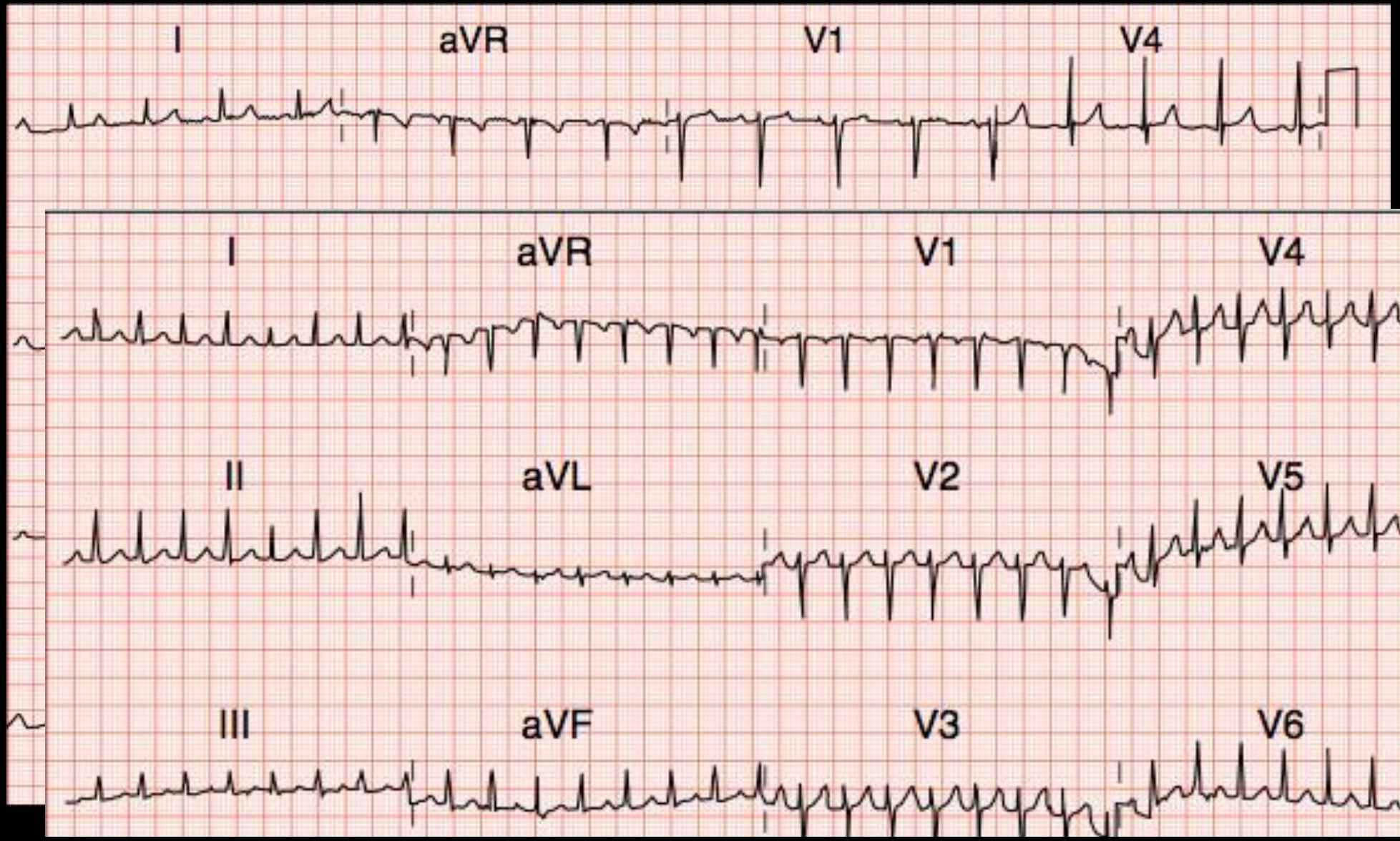
II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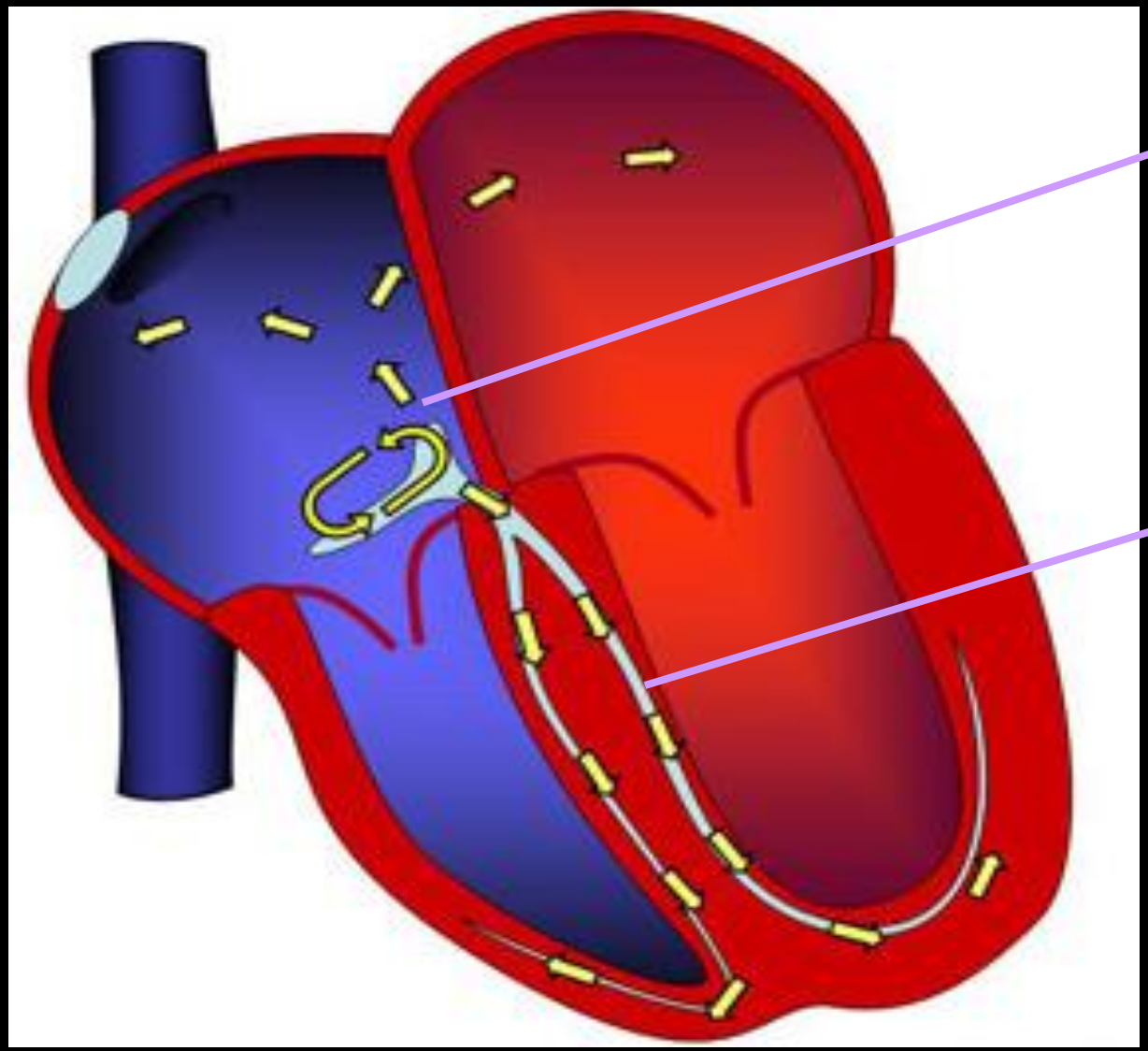
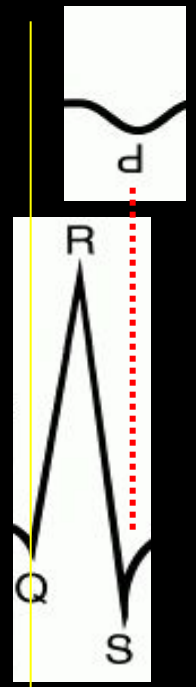
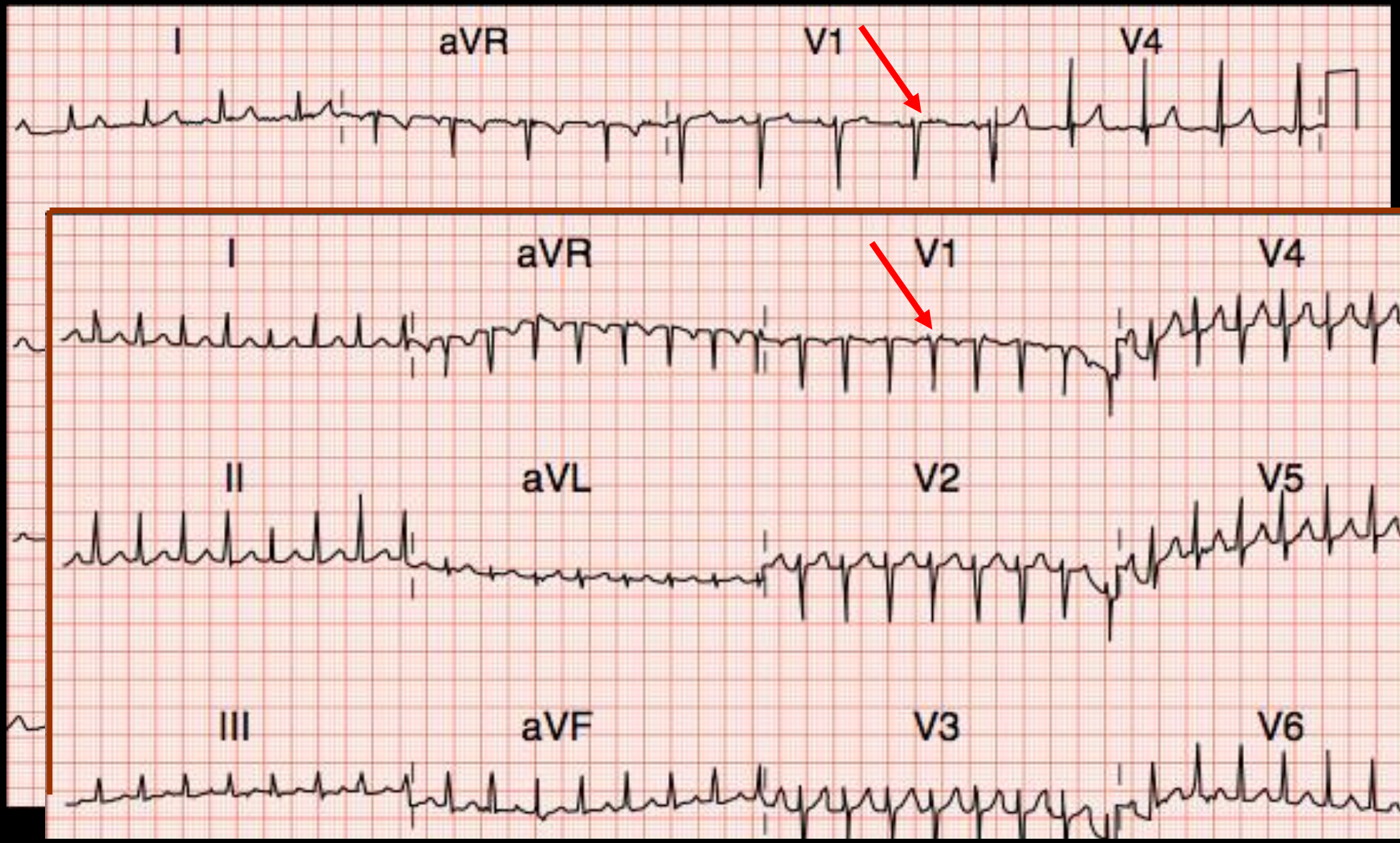
AVNRT






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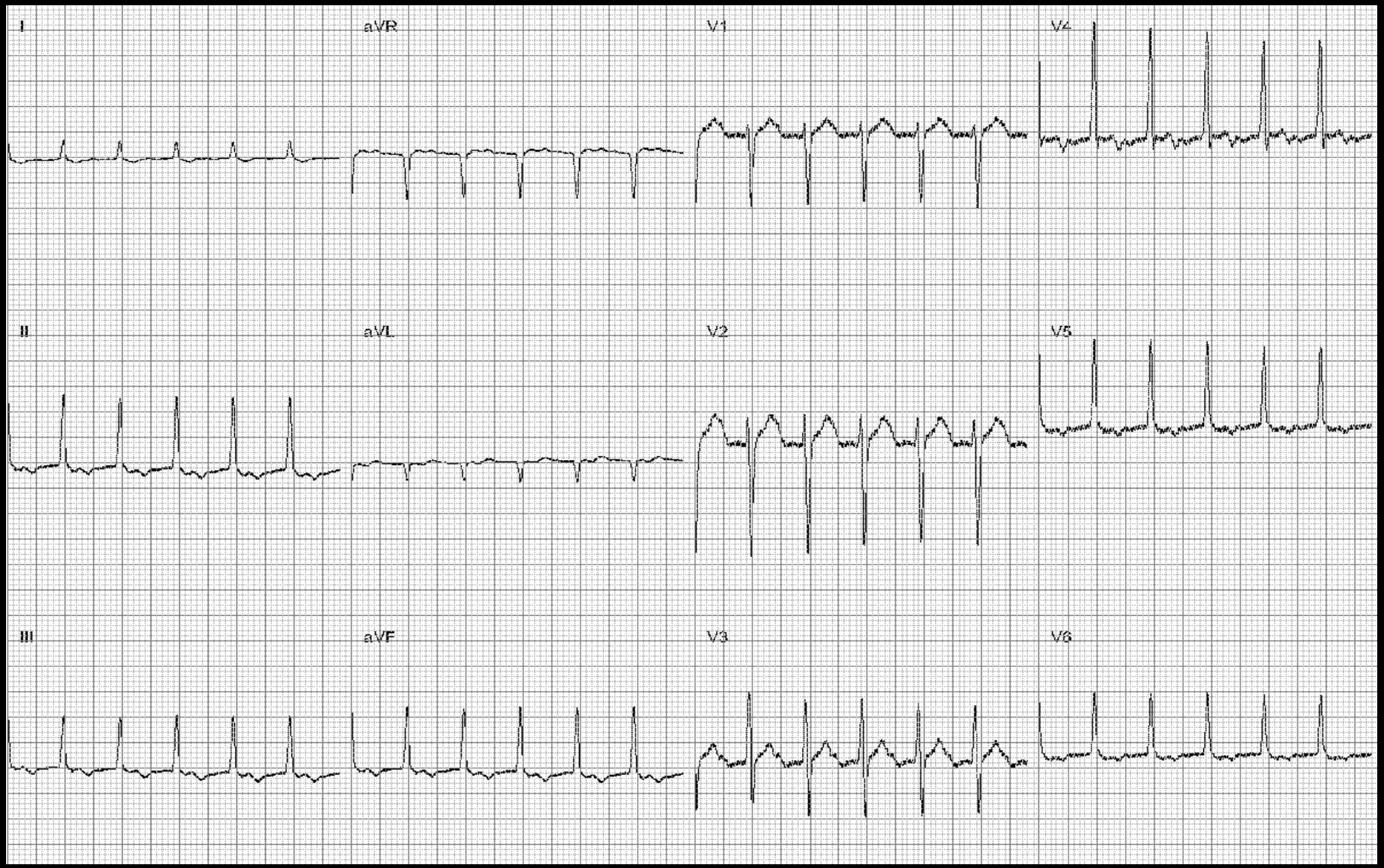
III
aVF
V3
V6

Question 2

- 25 yo woman with palpitations since childhood
- Worse during pregnancy
- Palpitations can last from seconds to minutes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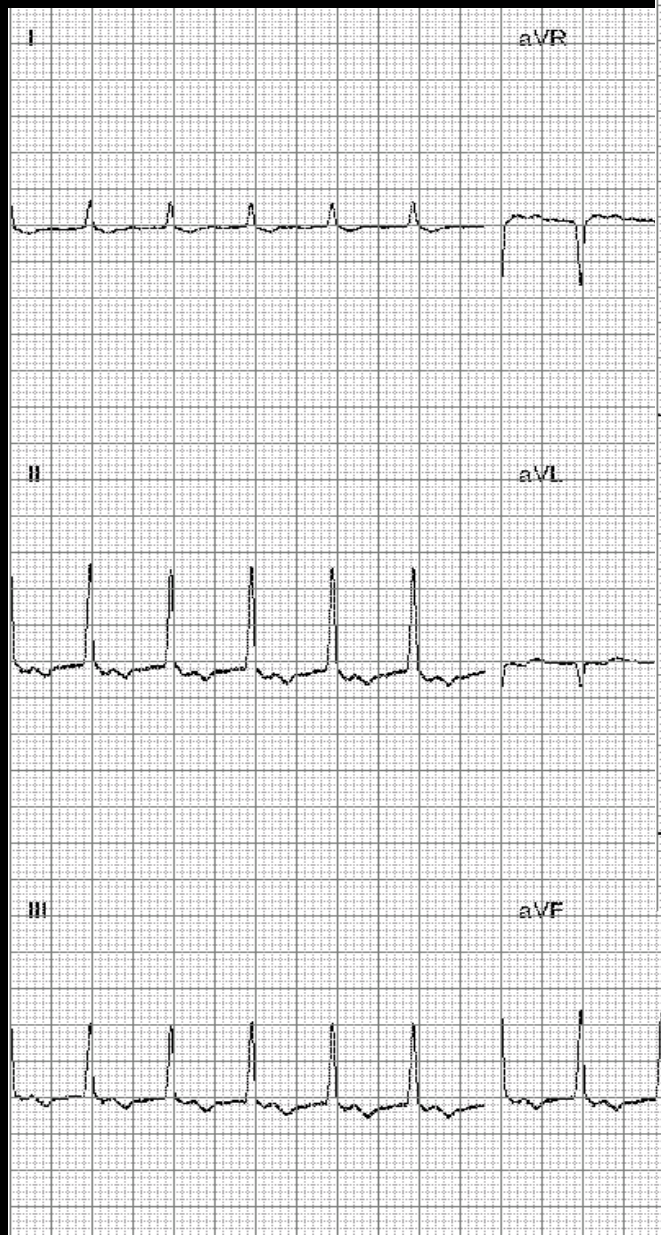
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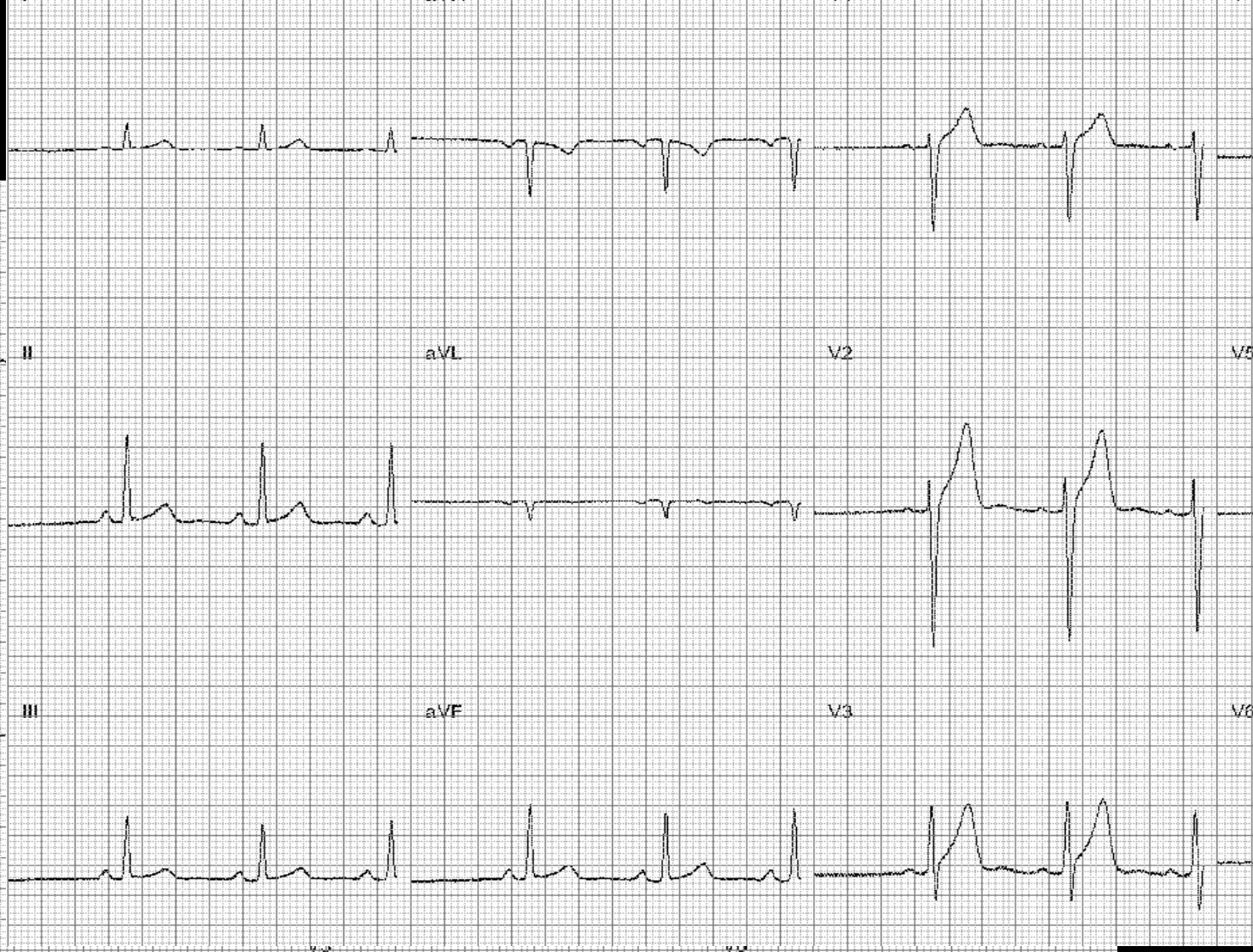
II
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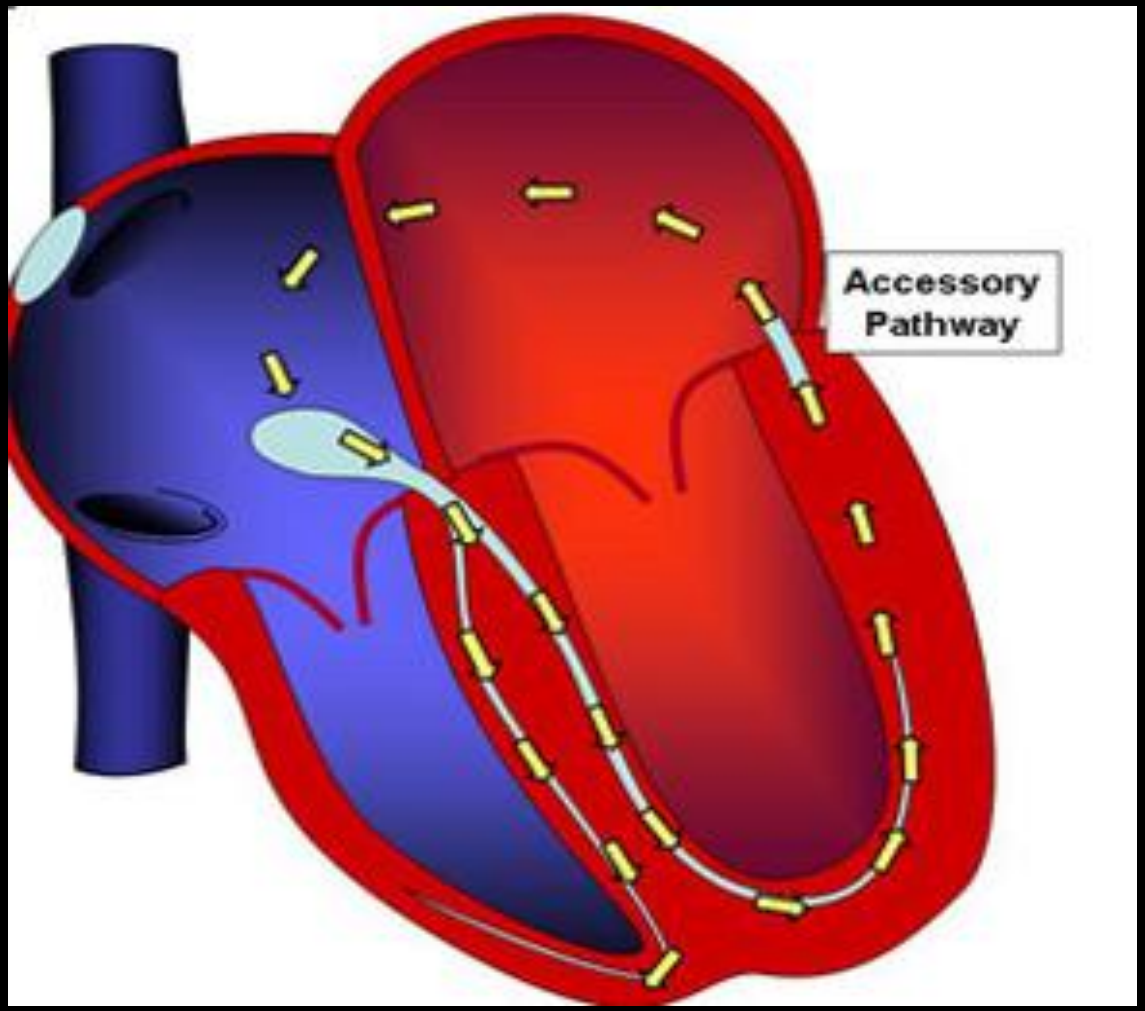
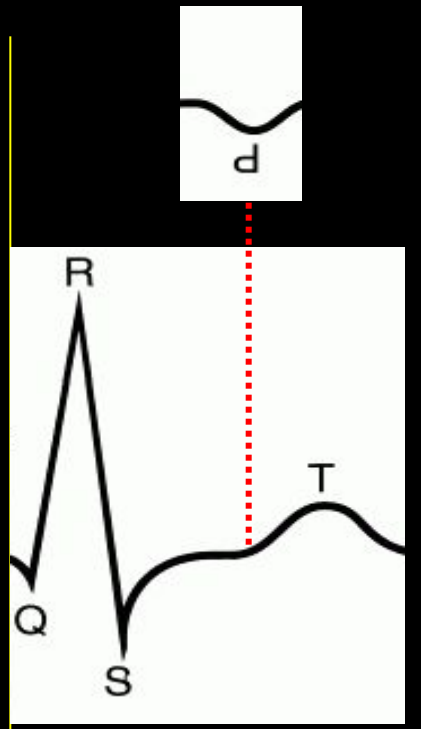
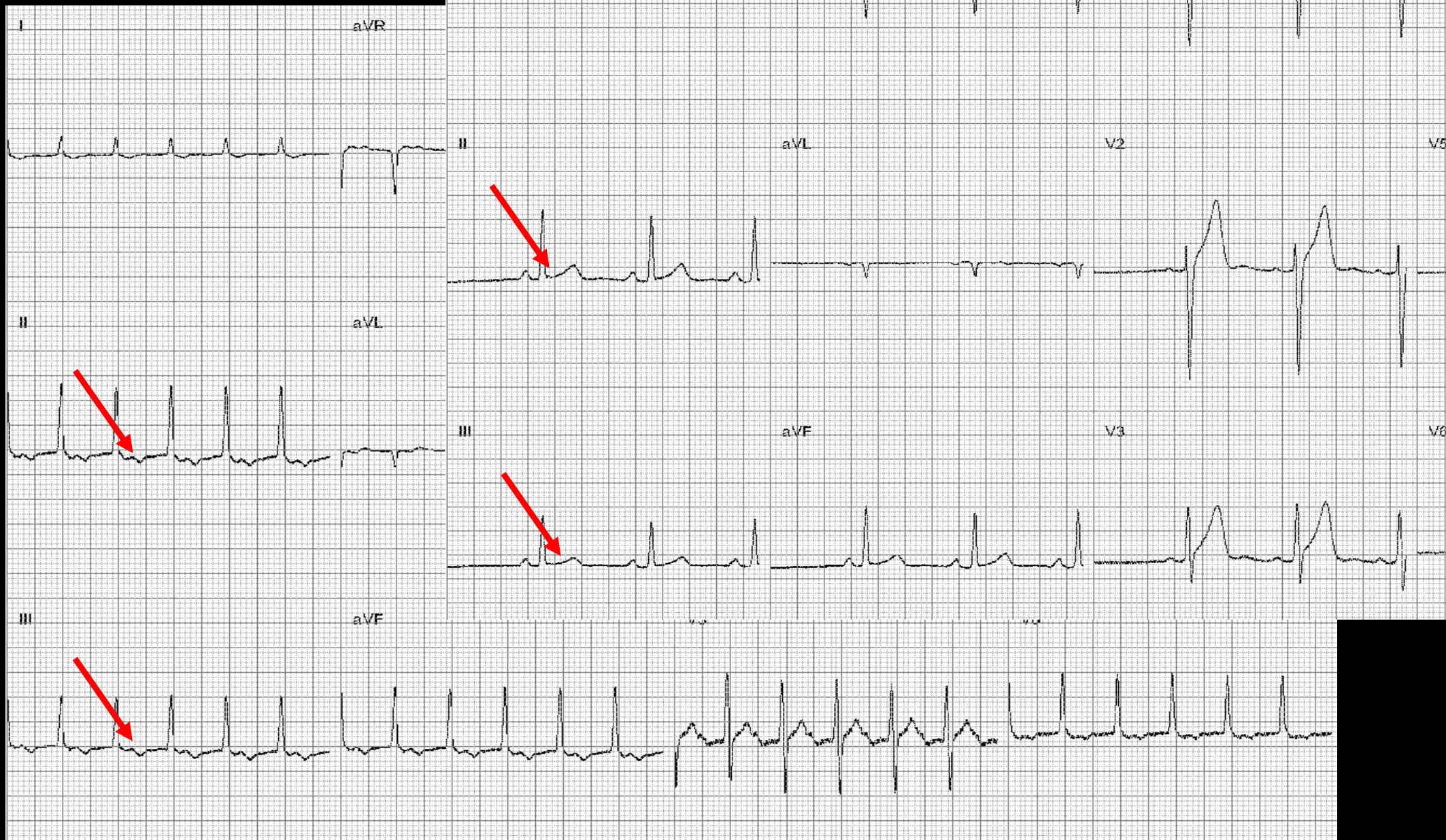
V2
III
aVF
V3

AVRT






I
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Spectrum of pre-excitation

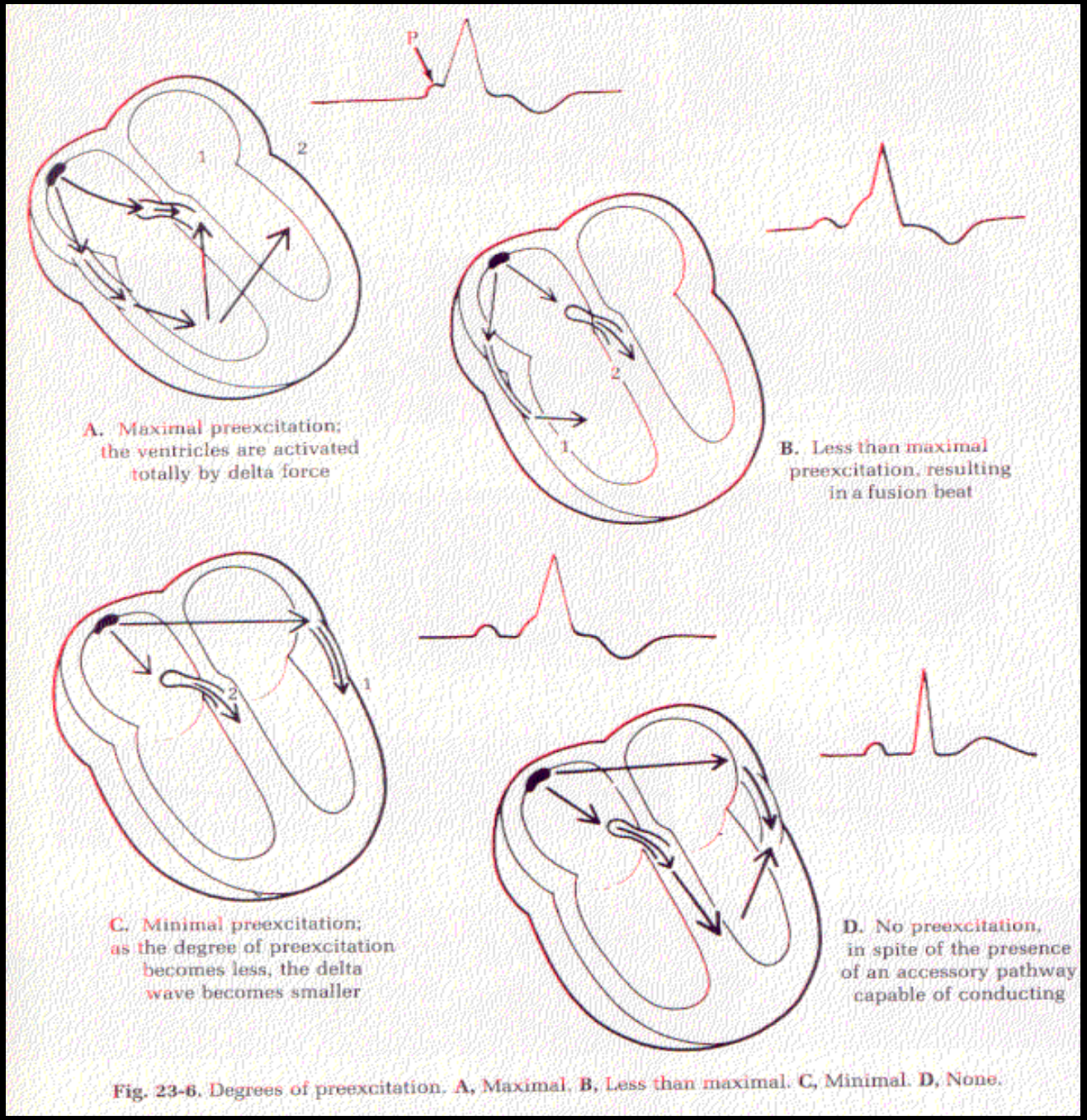
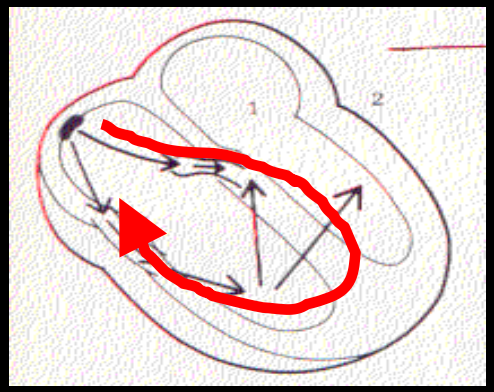


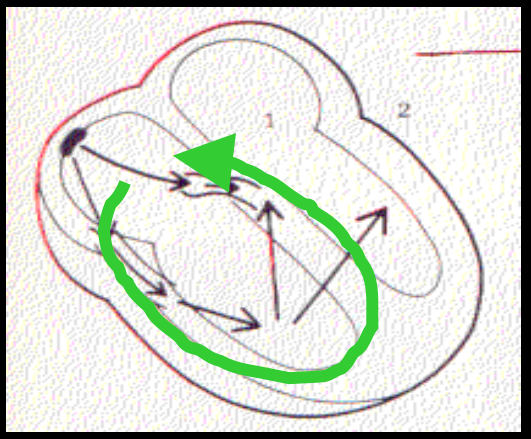
Fig. 23-6. Degrees of preexcitation. **A**, Maximal. **B**, Less than maximal. **C**, Minimal. **D**, None.

Mechanisms of AVRT



Orthodromic

Antidromic



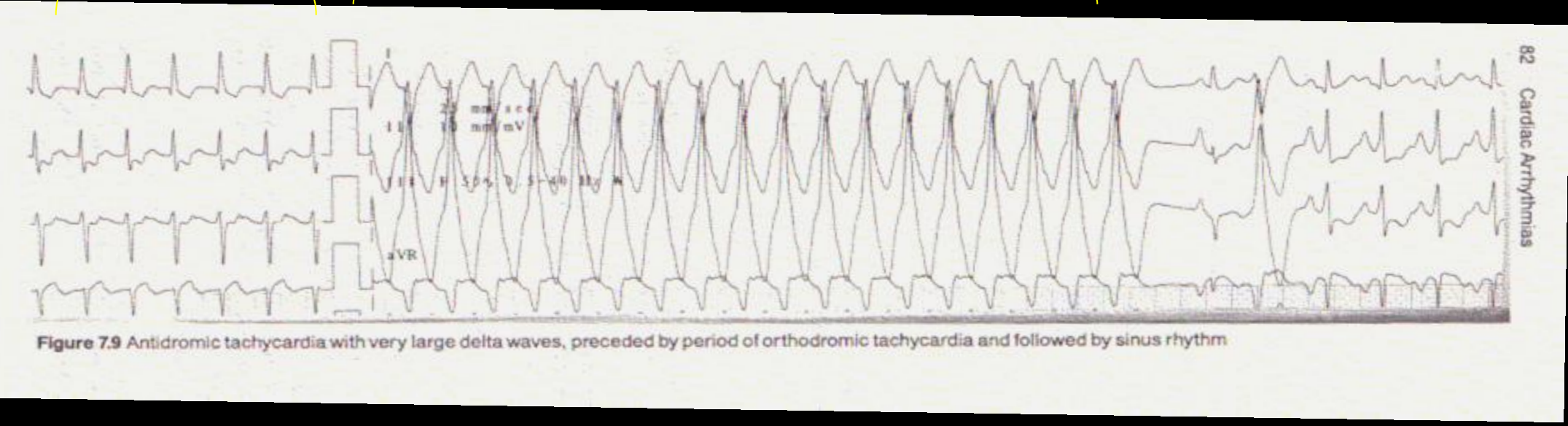
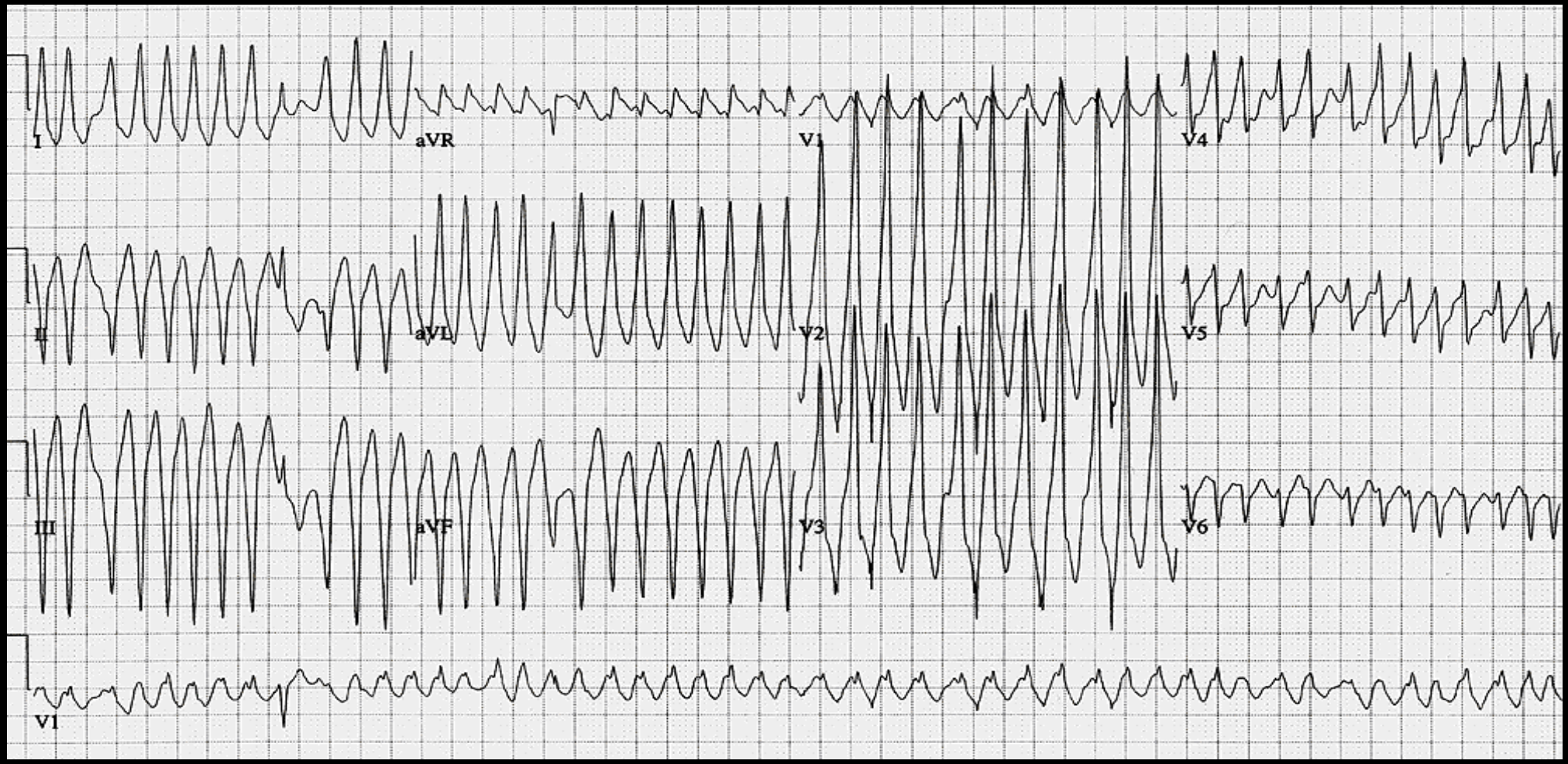
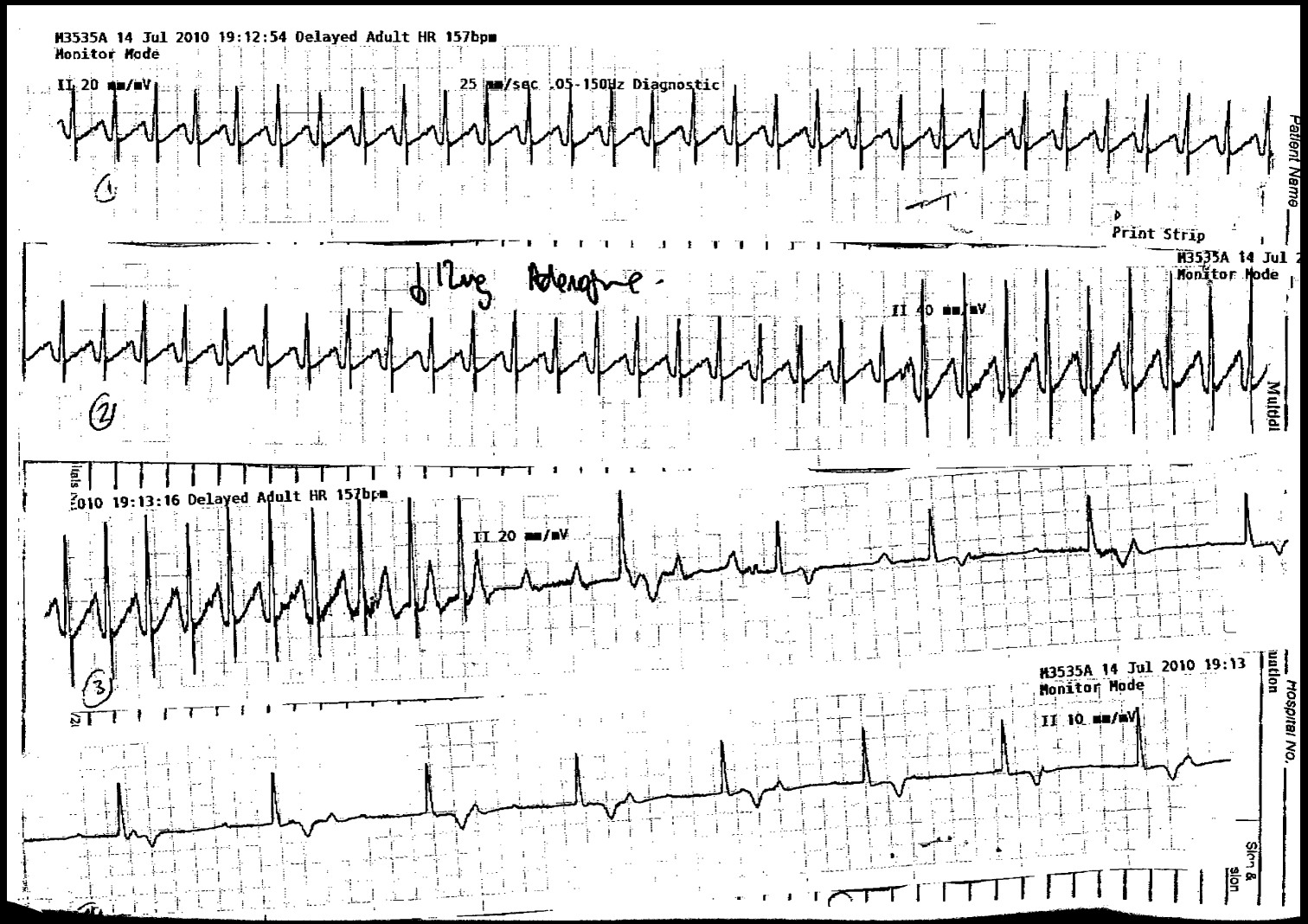


Figure 7.9 Antidromic tachycardia with very large delta waves, preceded by period of orthodromic tachycardia and followed by sinus rhythm

Post adenosine – what is rhythm?



Q3: 35 yo with palps, terminated with adenosine



For every arrhythmia, think of…

Age of onset

Duration of symptoms

Frequency of symptoms

Mode of presentation : triggers, relieving factors

Previous medical history

Family history

Unique features

Key points from clinical history

- Age
- Symptoms
  - Asymptomatic/ Syncope/ Palpitations/ Chest pain/ Dyspnoea
- 1st time or recurrent?
- Situation
  - Anger / Fright/ Exercise/ Sleep/ Micturition
- Mode of onset
  - Gradual or rapid
- Mode of termination
  - With a valsalva/ vagal manouevres
- Drug history
  - Anti-arrhythmics/ Stimulants/ Antibiotics- consult the BNF
  - Toxicity- accidental overdose
- Family history
- History of structural heart disease
- History of previous cardiac surgery/ablation

Management of narrow complex tachy

- File rhythm strip in notes (write patient name/DOB/date on strip)
- Drugs are ineffective! But try beta blockers, CCB, flecainide.
- Most arrhythmias should be referred on to an EP centre
  - High success rates for ablation for Aflutter, A tach, AVNRT, AVRT (95+ %)
  - Lower rates for AF (50-80%)

Lim-PB et al, Electrophysiology and ablation of arrhythmias, Br J Hosp Med 2012 (in press)

Take home messages

1. Symptomatic bradycardia = pacing indication
2. Tachycardias can be diagnosed in most cases from history + ECG
3. Most SVTs should be considered for ablation if refractory to drug therapy
4. AF Management = 1) Rate vs Rhythm and 2) CHADS-2

Managing palpitations
The GP refresher course
11 May 2012

P Boon Lim
Acting Consultant Cardiologist and Electrophysiologist
Imperial College Healthcare NHS Trust

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Diagnosis and management of Broad Complex Tachycardia

Differential diagnosis of BCT

- VT
- SVT + aberrant conduction
- Antidromic AV reentrant tachycardia
- VF
- Any pre-excited tachycardia (i.e. an accessory pathway in presence of atrial tachycardia/flutter/AF).

Differentiating VT vs SVT with aberration

- ECG criteria is difficult to remember at 2am with unwell patient – and mostly non-absolute in making diagnosis.

- If uncertain, treat as VT

- Key features
  - Ischaemic heart disease (assume VT always)
  - Structural heart disease
  - Sympathomimetics
  - Beware electrolytes.
  - Known history of VT

Differentiating VT vs SVT with aberration (for medical finals)

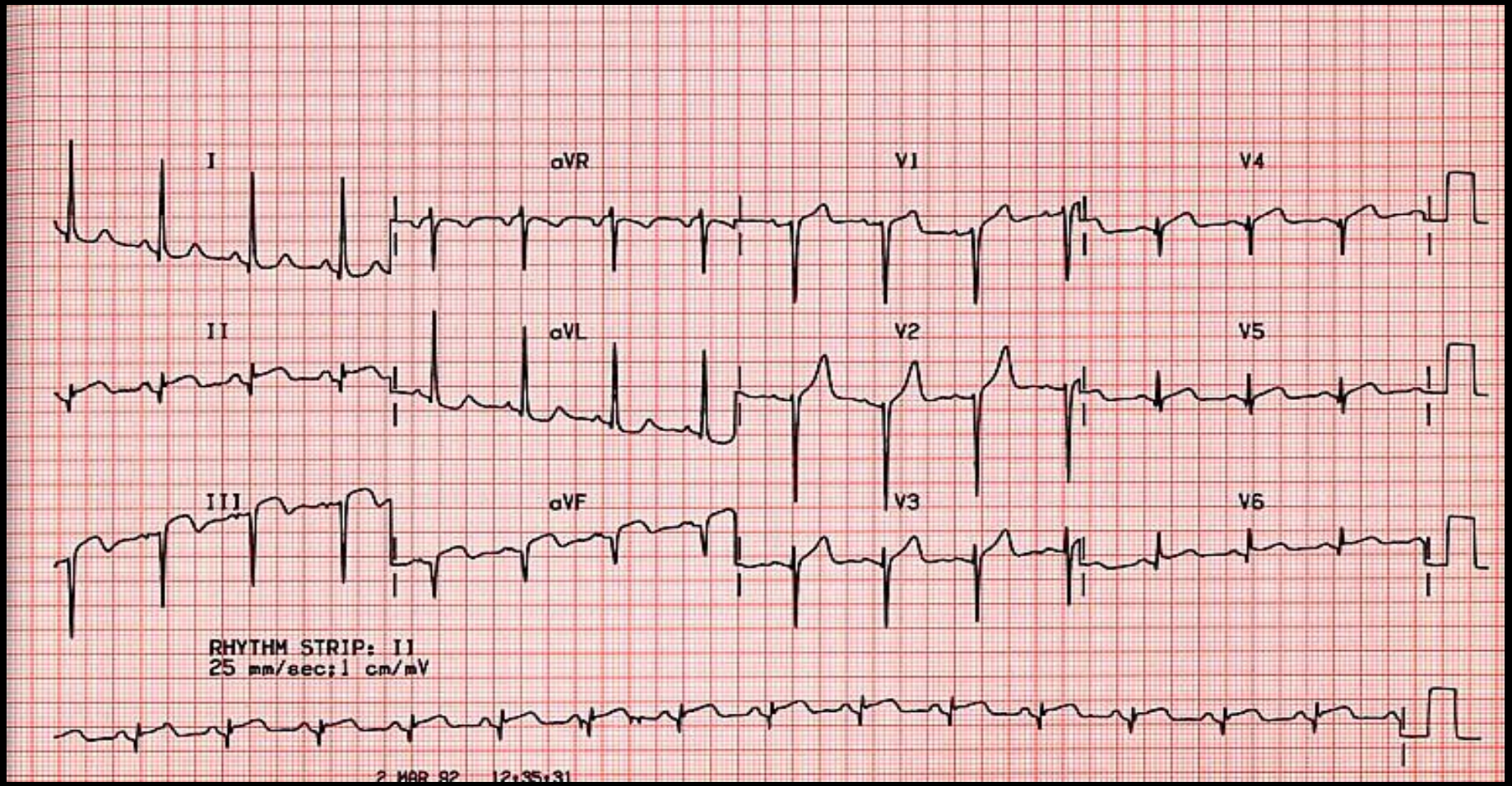
- Fusion/capture beats
- Dissociated P waves
- Broad QRS (>140ms)
- Concordance
- Extreme axis deviation

Best ECG discriminators

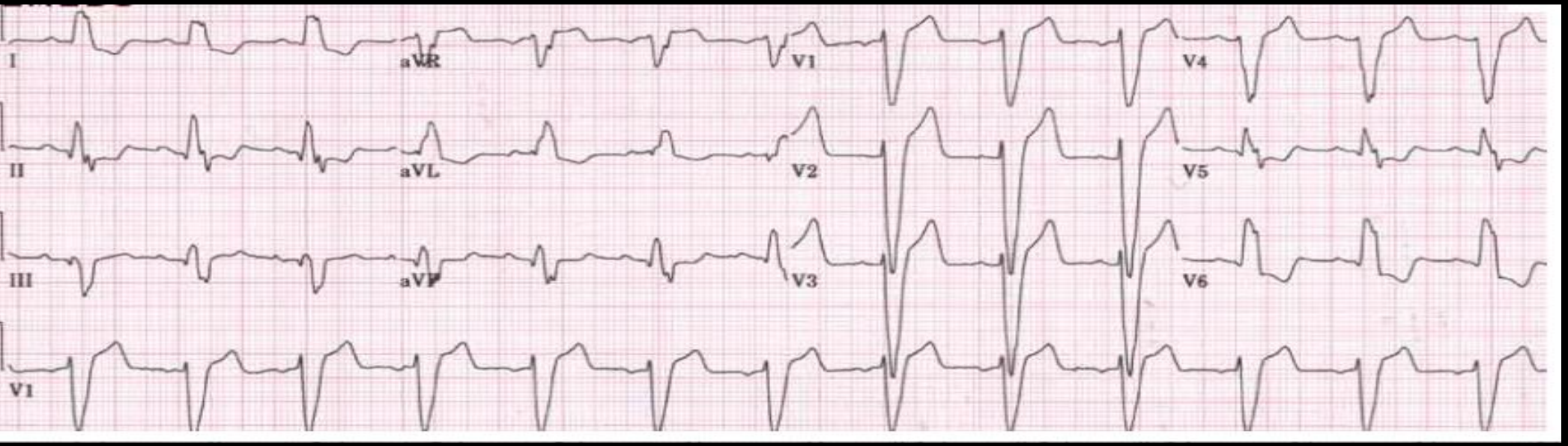
Differentiating VT vs SVT with aberration (for 2am in ER!)

- Does it look like a typical RBBB or LBBB?

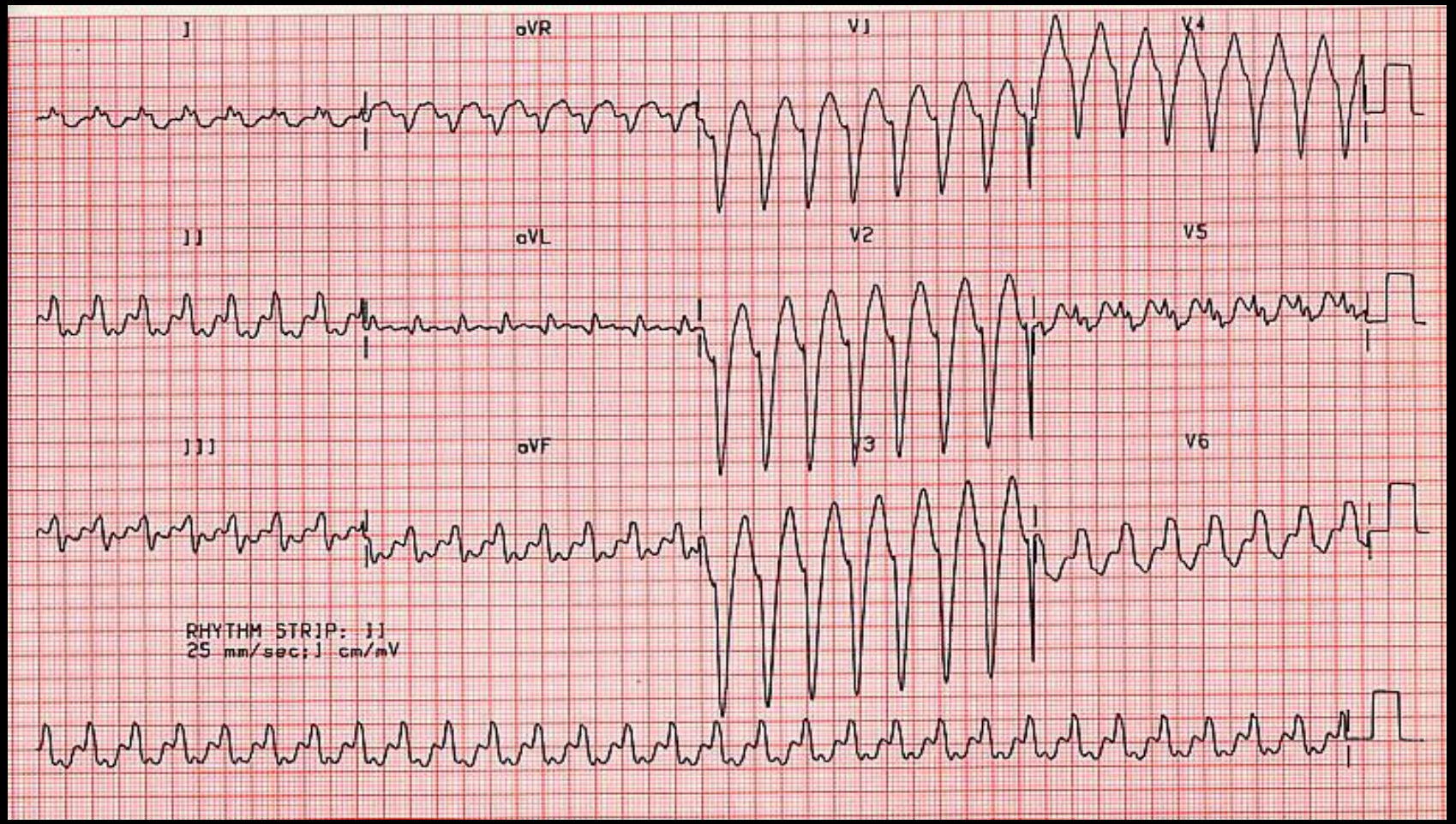
Normal QRS complex – observe onset QRS



Typical LBBB – observe the onset of QRS



VT or SVT?


I
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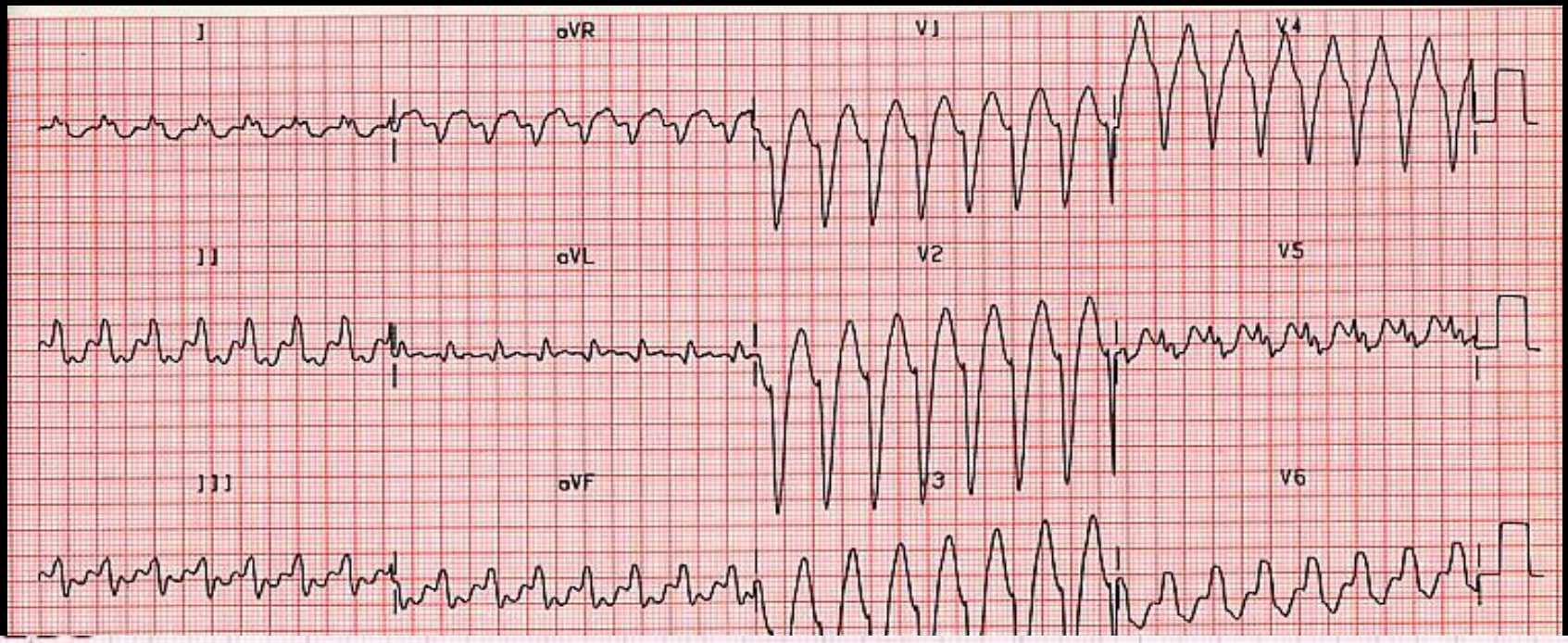
V6
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV

VT or SVT?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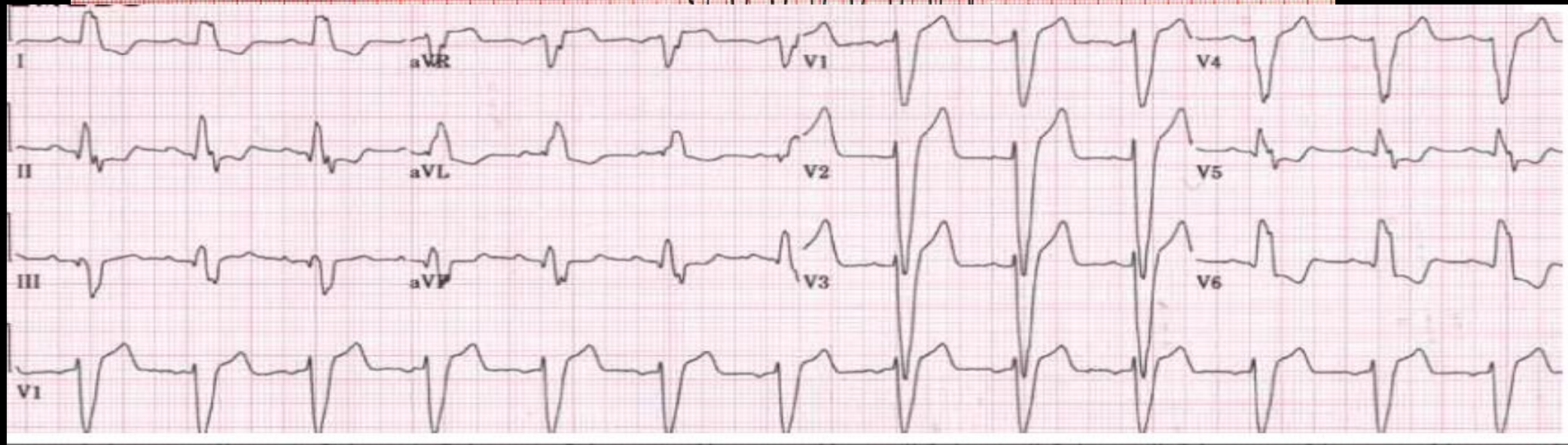
I
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V1
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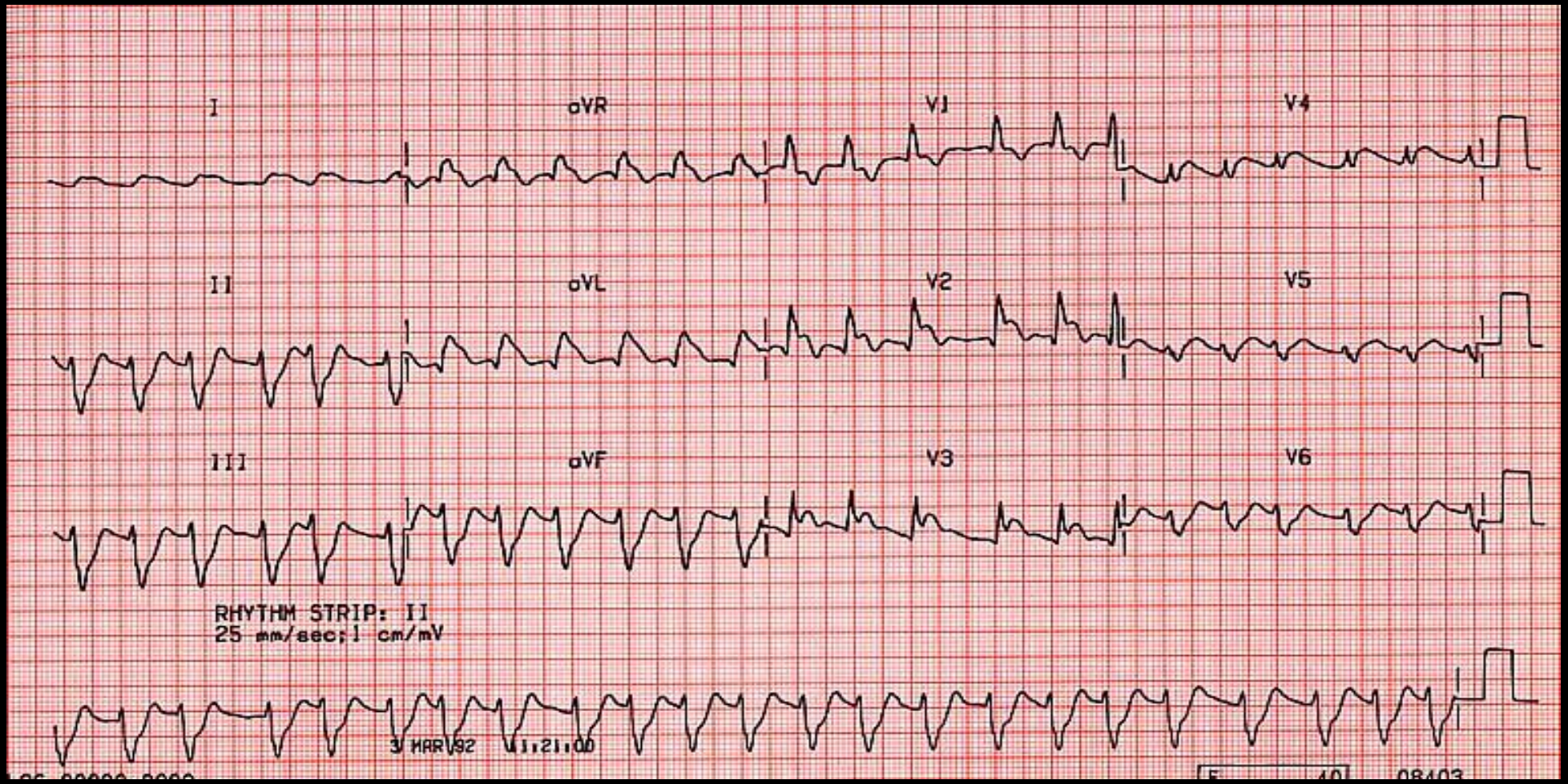
V3
V6
V1

VT or SVT?


I
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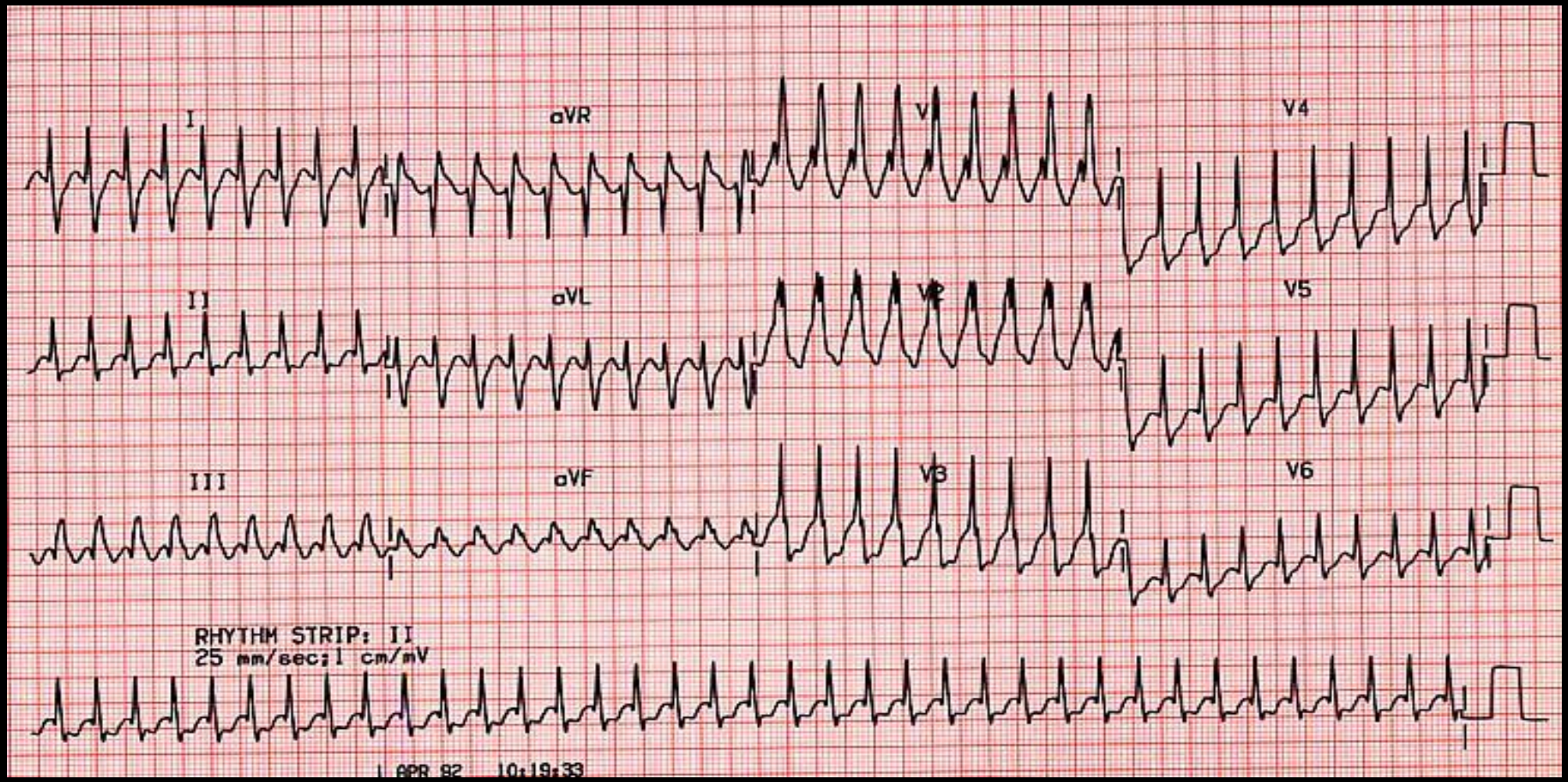
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV

VT or SVT?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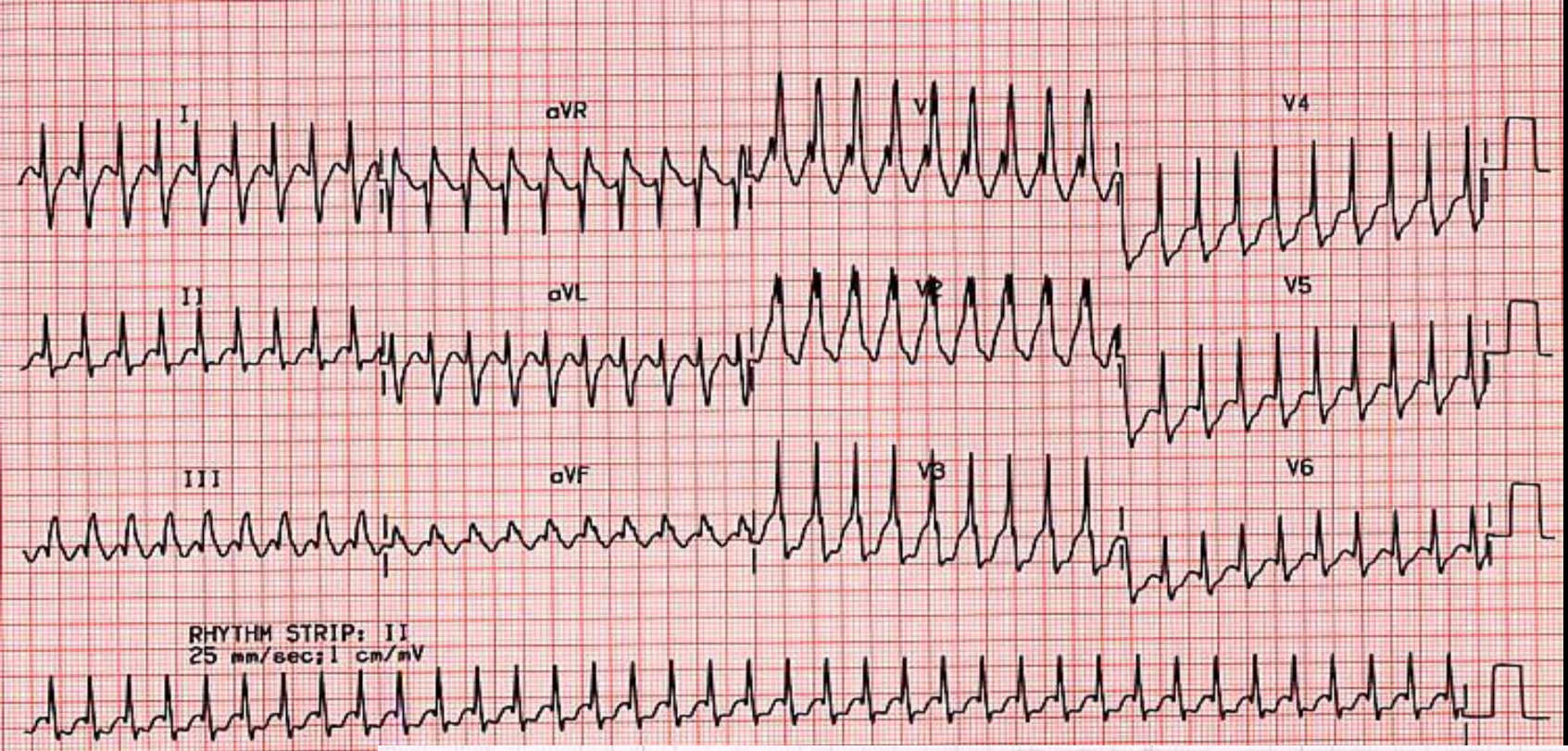
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV


I
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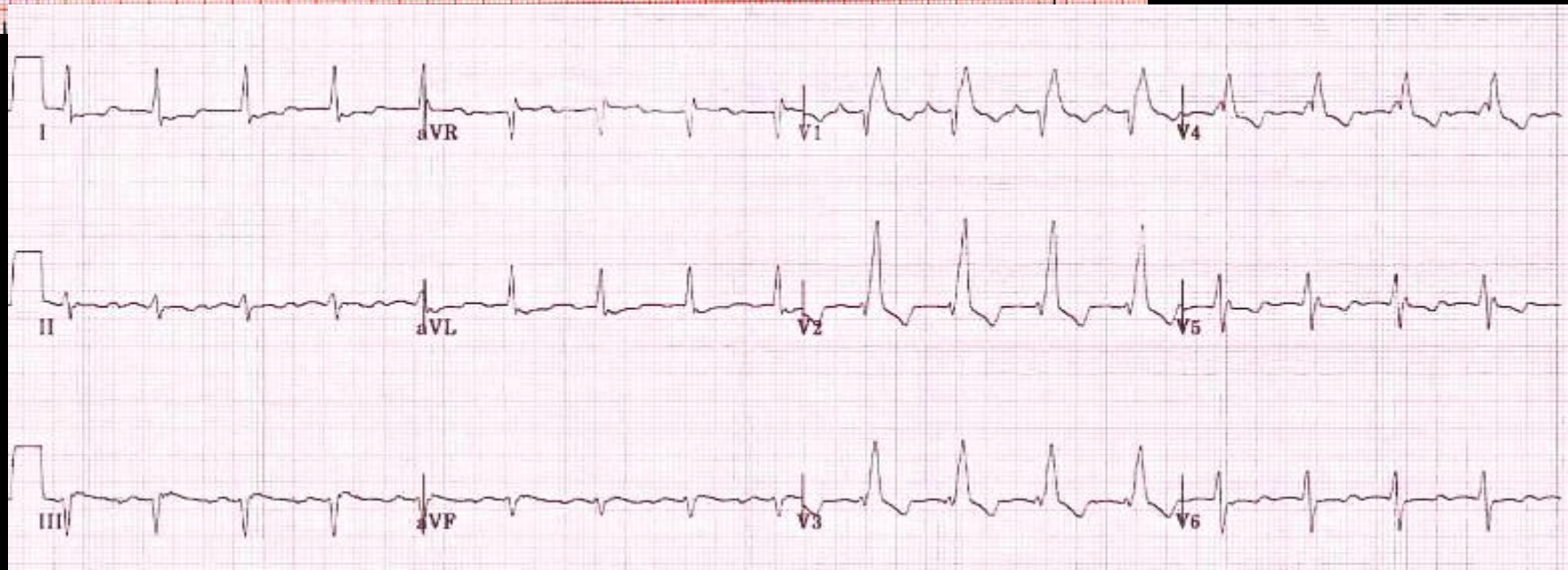
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV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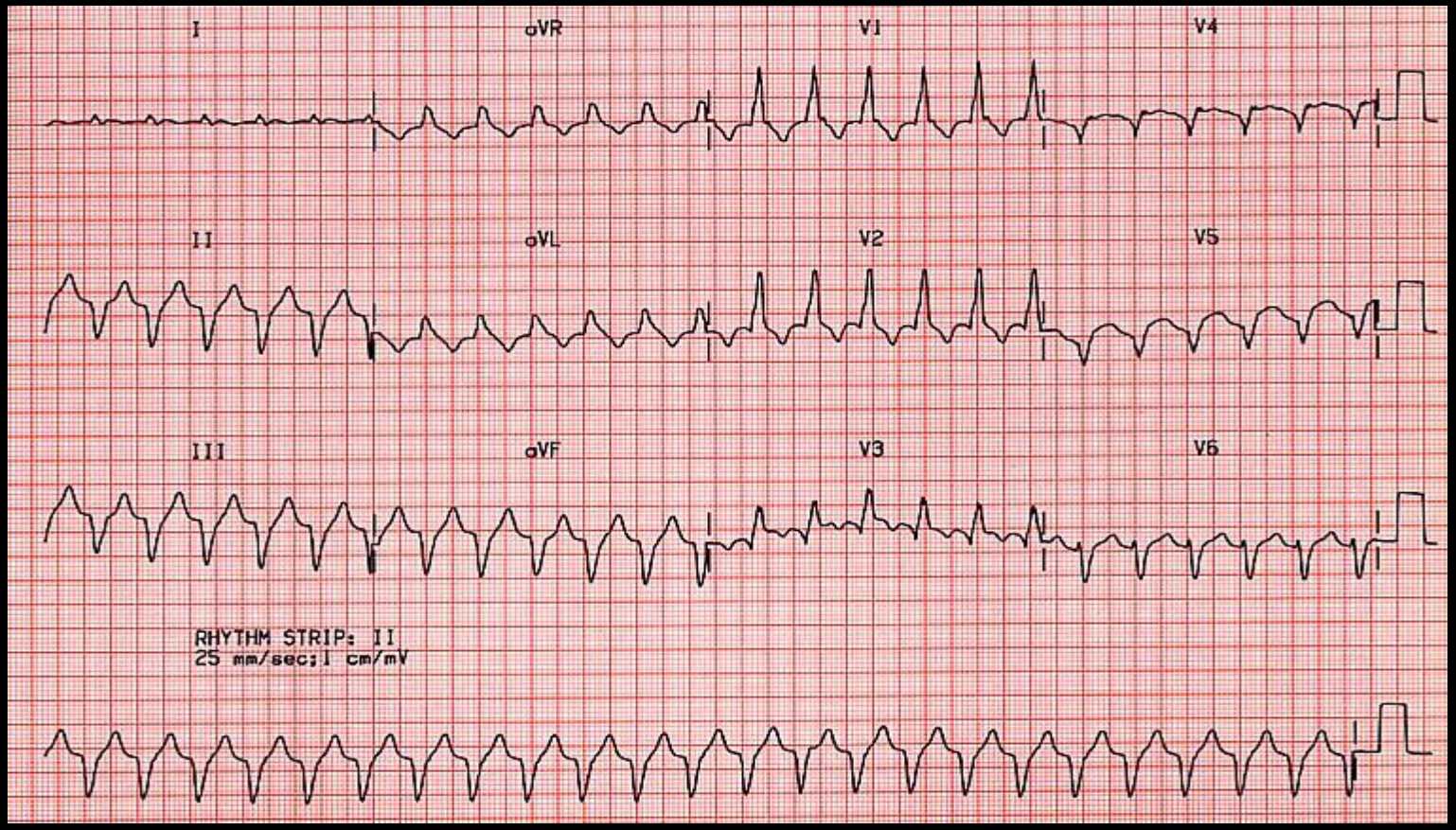
VT or SVT?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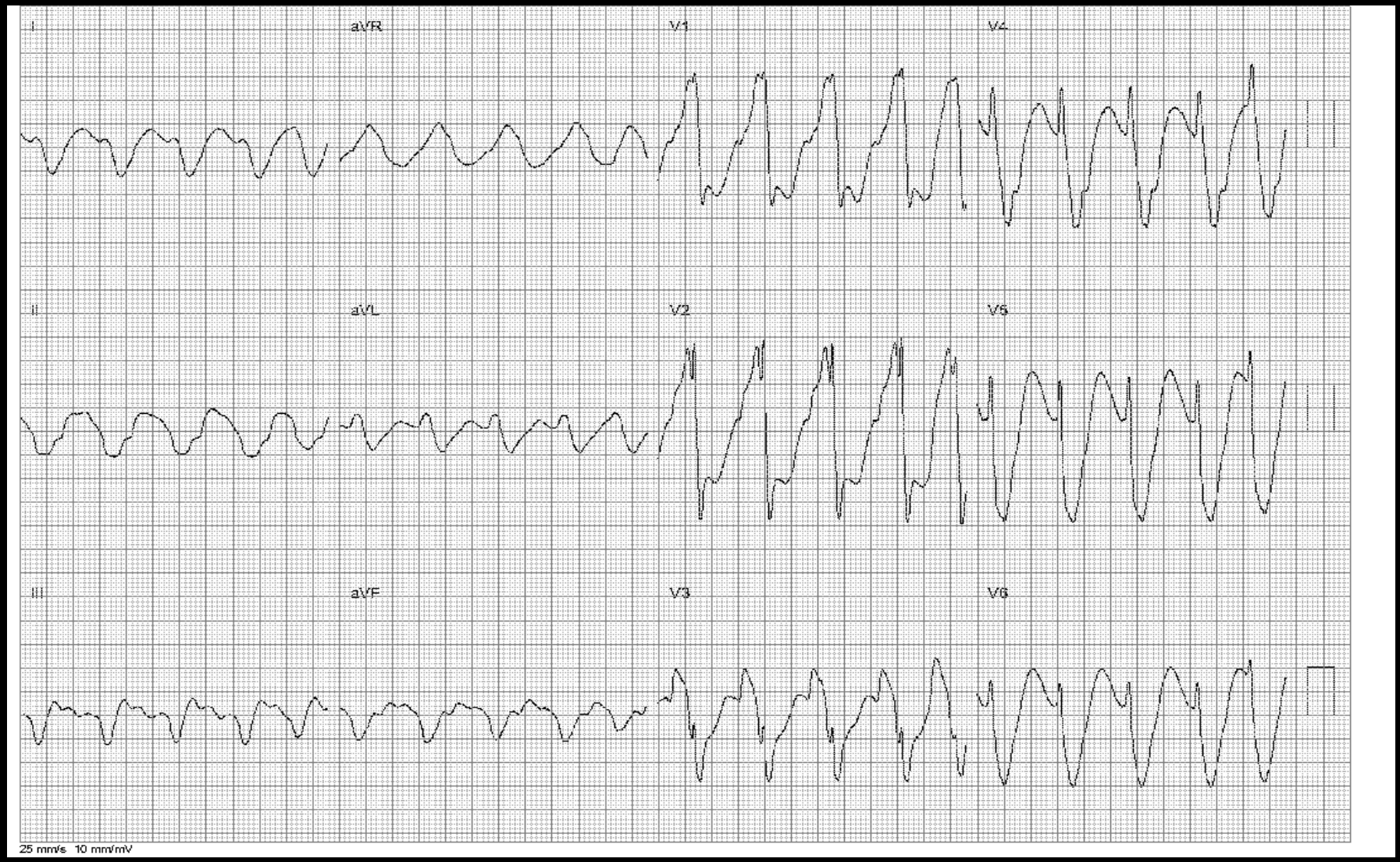
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV

VT or SVT?



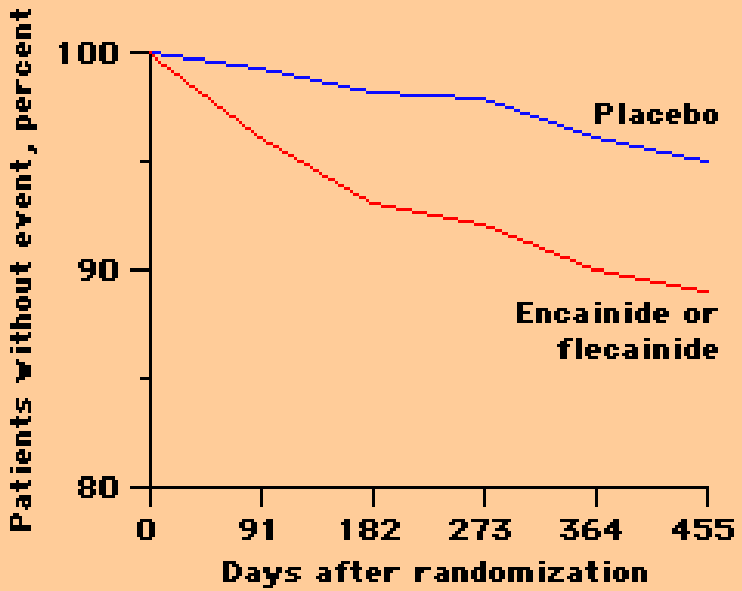
VT or SVT?

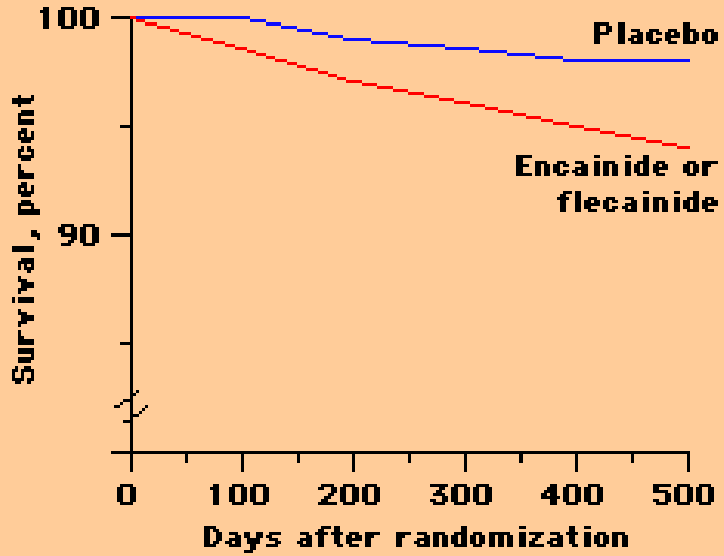
Acute management of BCT

- Haemodynamic instability – DCCV
- If not unstable, time to think
  - Accurate/relevant history/ drugs
  - Check electrolytes (K/Mg)
  - Attempt to make accurate diagnosis – 12 lead ECG
  - Acute ischaemia (primary PCI call ? Thrombolysis)
- Pharmacology (VT)
  - Magnesium/ K+ correction
  - Amiodarone
  - Beta blockers
  - Lignocaine
  - Procainamide
- Non pharmacology
  - Overdrive pacing
  - DCCV

Chronic management of VT

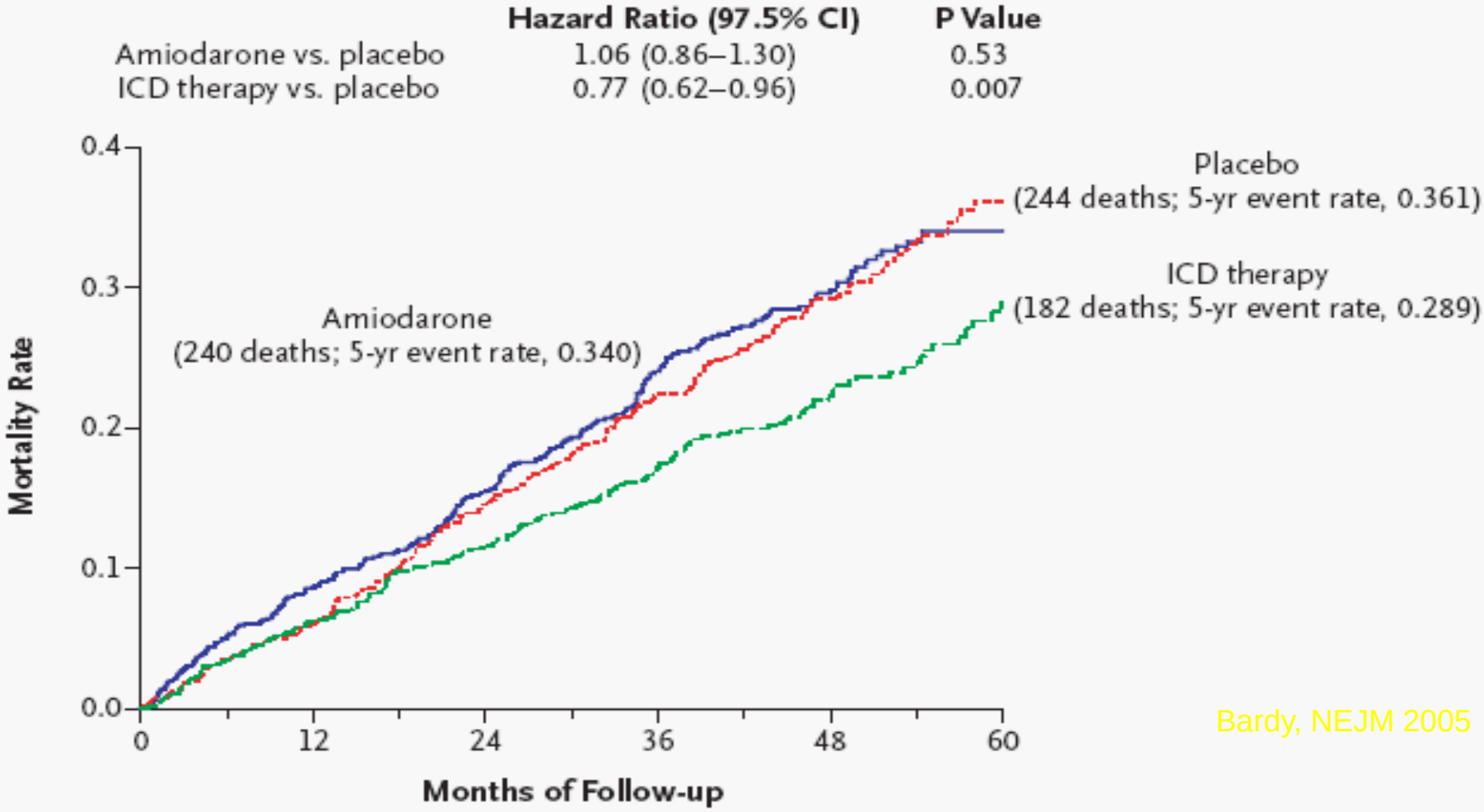
Why drugs have a bad name in VT?





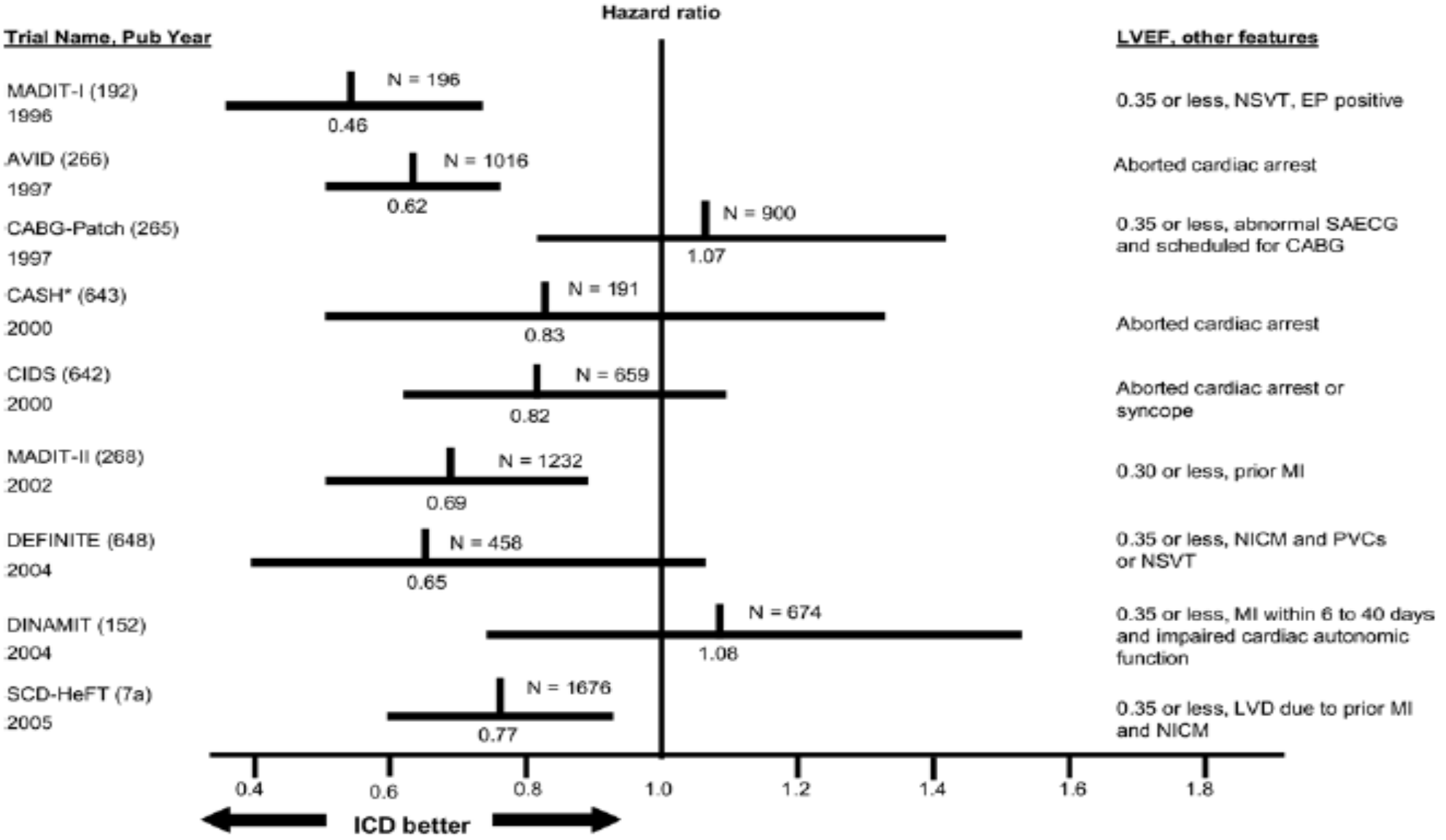
Encainide and flecainide increase cardiac mortality Results of the Cardiac Arrhythmia Suppression Trial (CAST) in patients with ventricular premature beats after myocardial infarction. Patients receiving encainide or flecainide had, when compared to those receiving placebo, a significantly lower rate of avoiding a cardiac event (death or resuscitated cardiac arrest) (left panel, p = 0.001) and a lower overall survival (right panel, p = 0.0006). The cause of death was arrhythmia or cardiac arrest. (Data from Echt, DS, Liebson, PR, Mitchell, B, et al, N Engl J Med 1991; 324:781.)

Drugs don’t prevent SCD



Bardy, NEJM 2005

Only ICD saves lives!



Future challenges

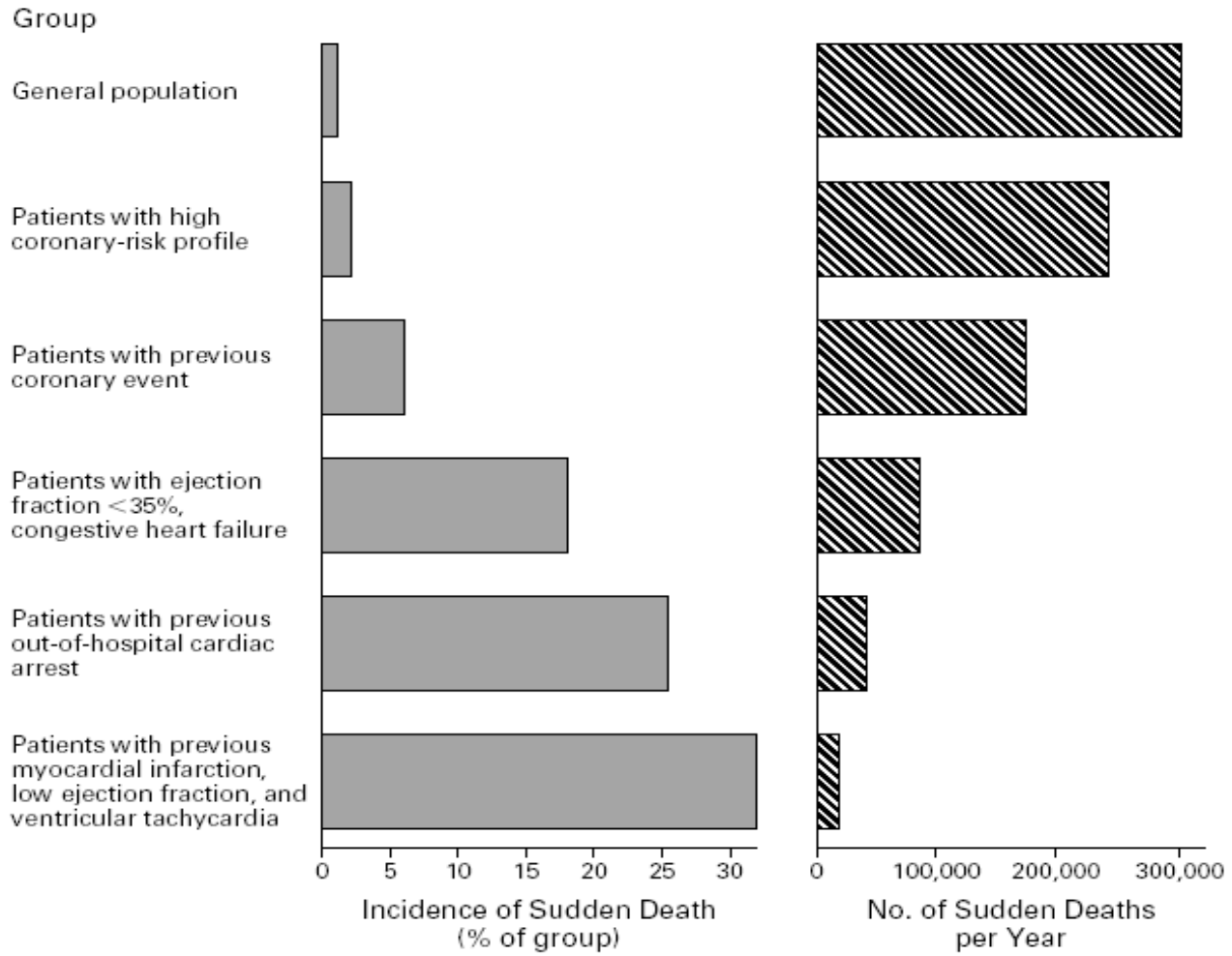


Figure 1. The Incidence of Sudden Death in Specific Populations and the Annual Numbers of Sudden Deaths in Those Populations. Most of the deaths occur in the larger, lower-risk subgroups. Modified from Myerburg et al.[10] with the permission of the publisher.

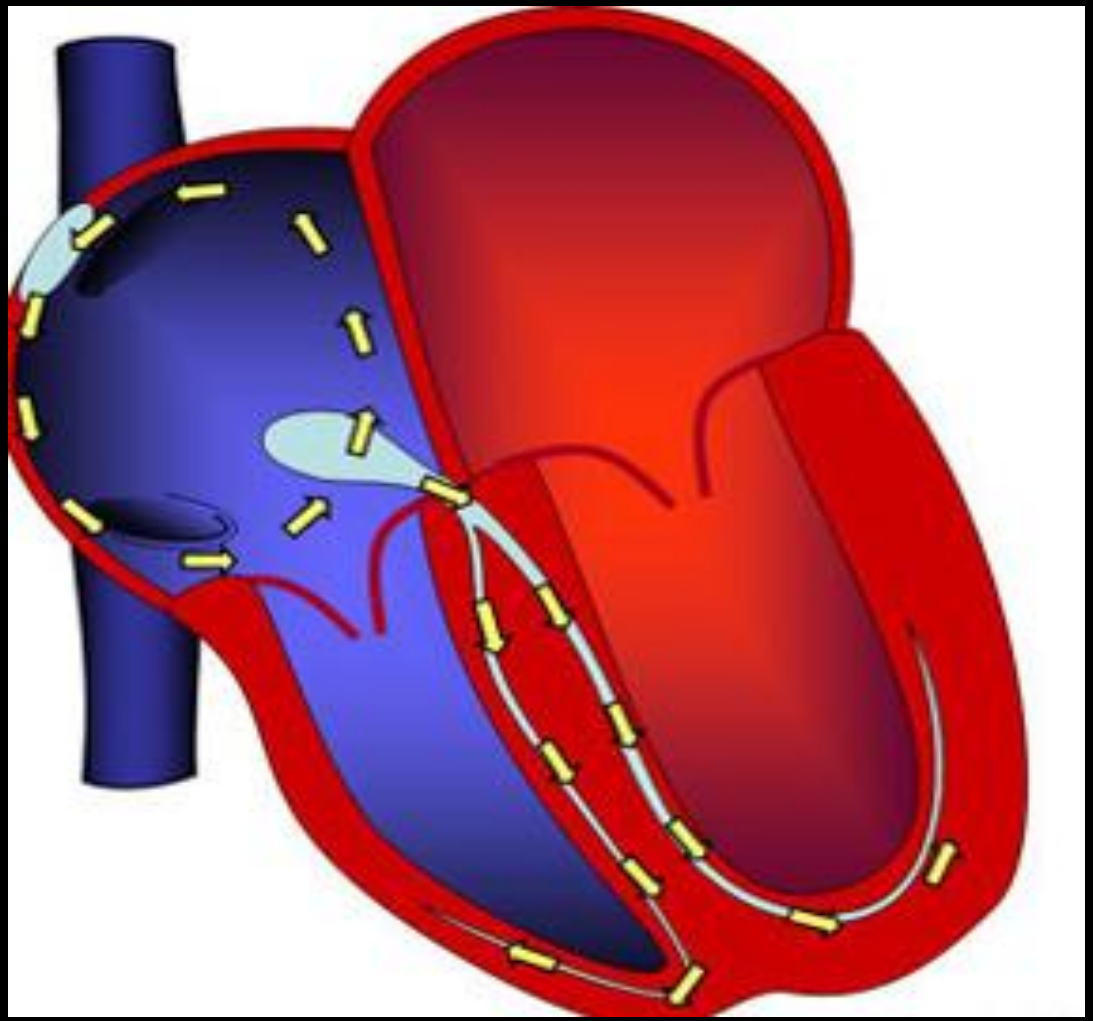
Atrial Flutter

- A macro re-entrant arrhythmia
  - Anatomical barrier
  - Zone of slow conduction

- Typical atrial flutter
  - Contained within the right atrium
  - Constrained anteriorly by the tricuspid valve
  - Constrained posteriorly by the crista terminalis and eustachian ridge
  - Travels in a counterclockwise direction around the atrium

- Atypical atrial flutters
  - Counterclockwise flutter
  - ASD/ scar related flutter
  - Perimitral flutter

Atrial flutter



Atrial Flutter

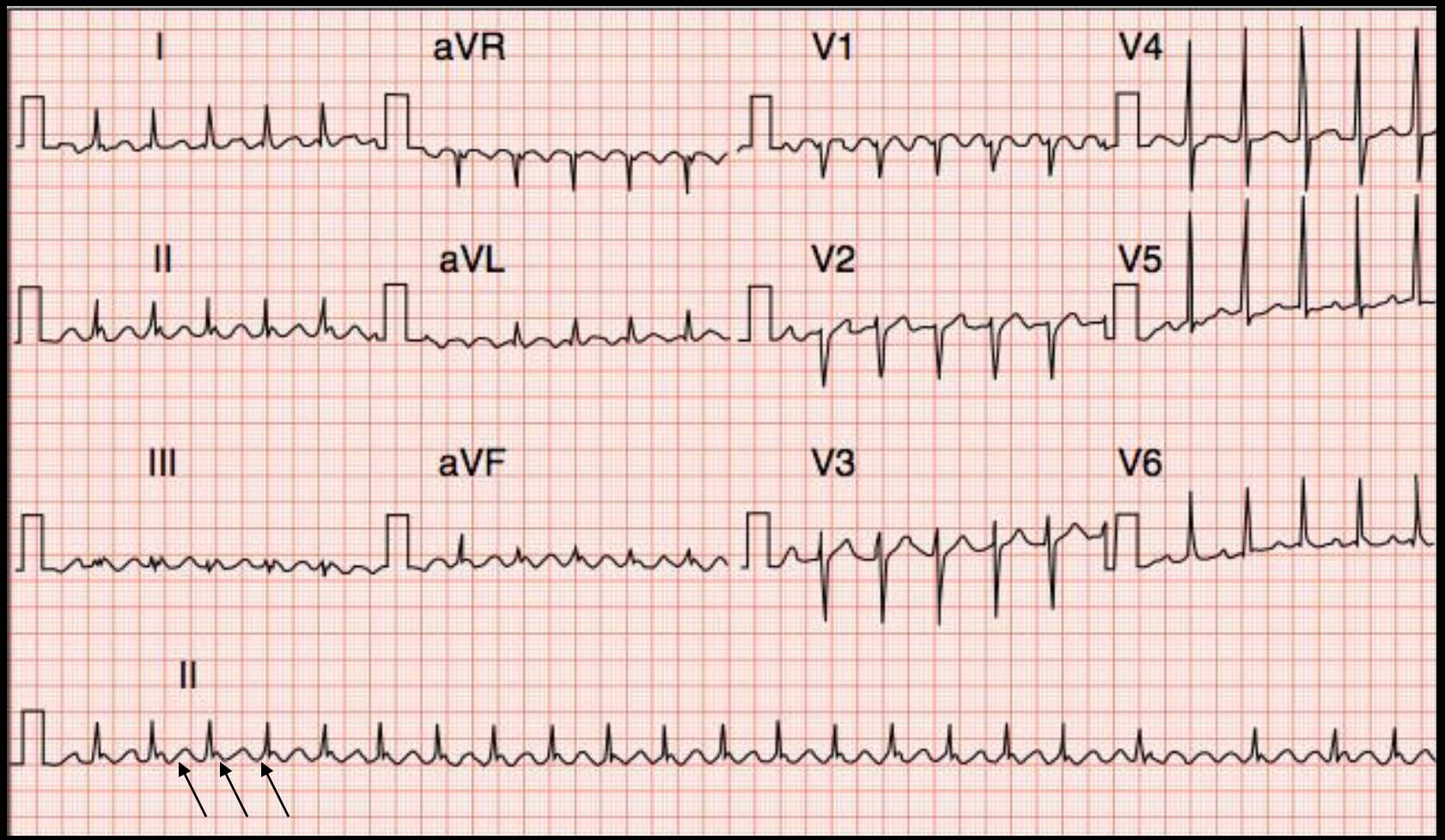
- Tends to occur in middle age
  - Probably due to atrial dilatation
- Pulmonary embolism
  - Commonly presents with a sinus tachycardia
- Associated valve disease
  - Mitral or Tricuspid disease
  - Atrial septal defects
  - Chronic ventricular failure
- Toxic and Metabolic conditions
  - Alcohol/ thyrotoxicosis/ pericarditis
- Previous ablation

Atrial Flutter

Examination

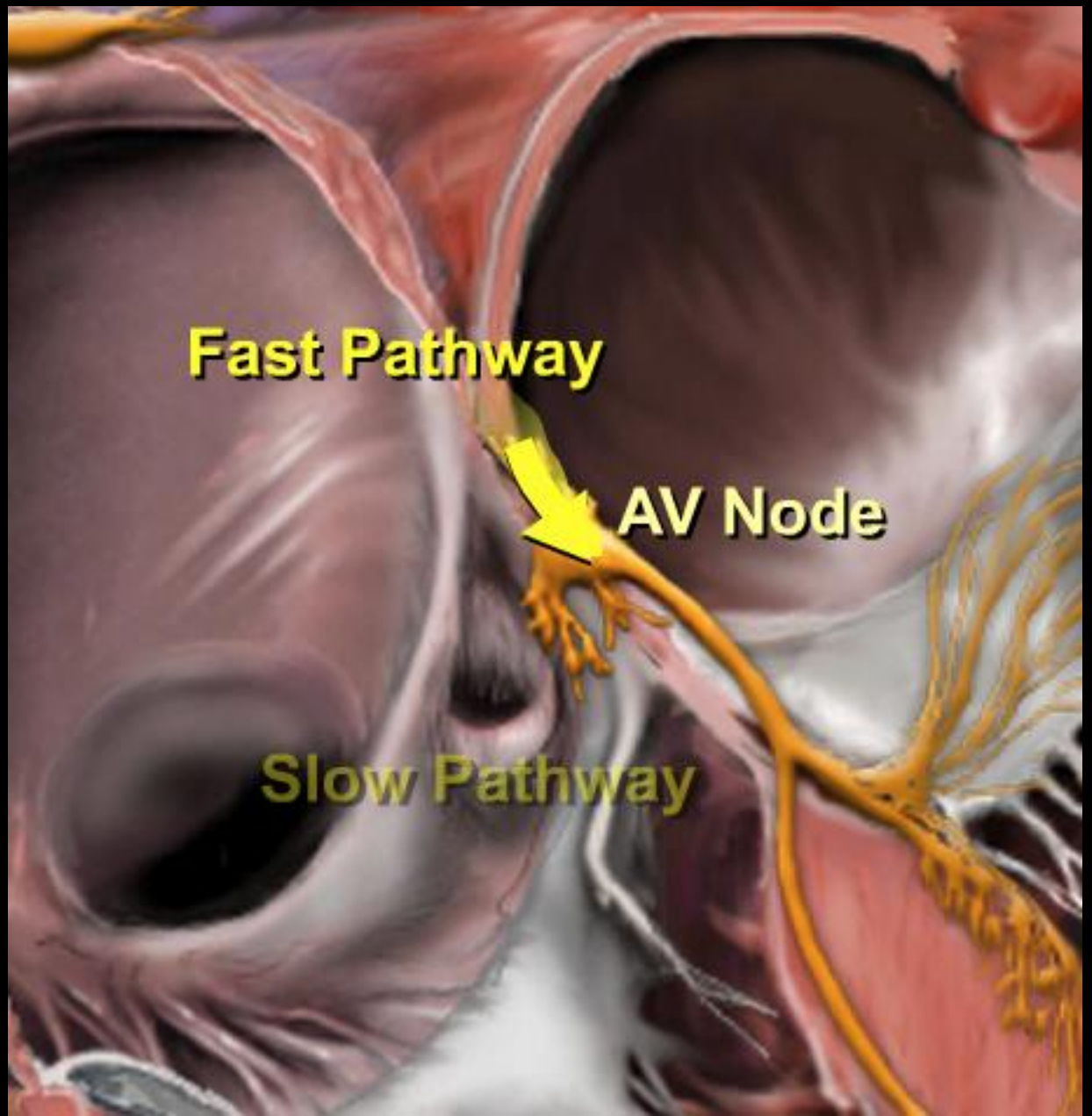
- Rarely helpful in establishing the diagnosis
- Regular pulse (150bpm- 2:1, 75bpm 4:1- can be slower)
- May see rapid, regular flutter waves in the JVP
- Heart sounds
  - Constant intensity of S1 *if* relationship of flutter waves to QRS is constant
- Carotid massage or adenosine
  - Allows flutter waves to be seen more easily
  - Ventricular rate will increase when CSM is stopped

Atrial Flutter

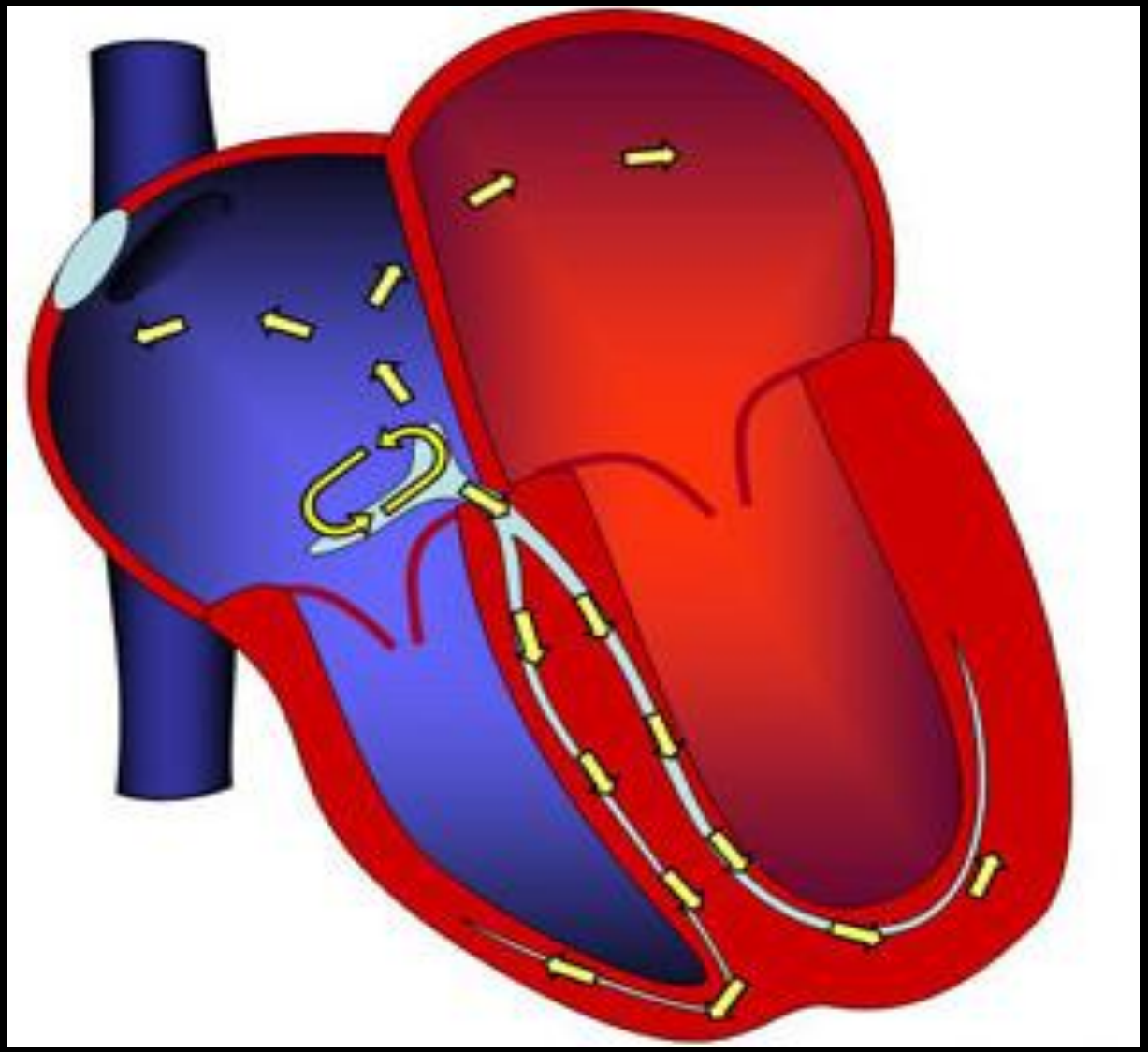


AVNRT

- Commonest supraventricular arrhythmia
  - ie dependent upon the AV node
  - AV Node with Dual (or more) physiology: Fast and slow pathway


Fast Pathway
AV Node
Slow Pathway

AVNRT



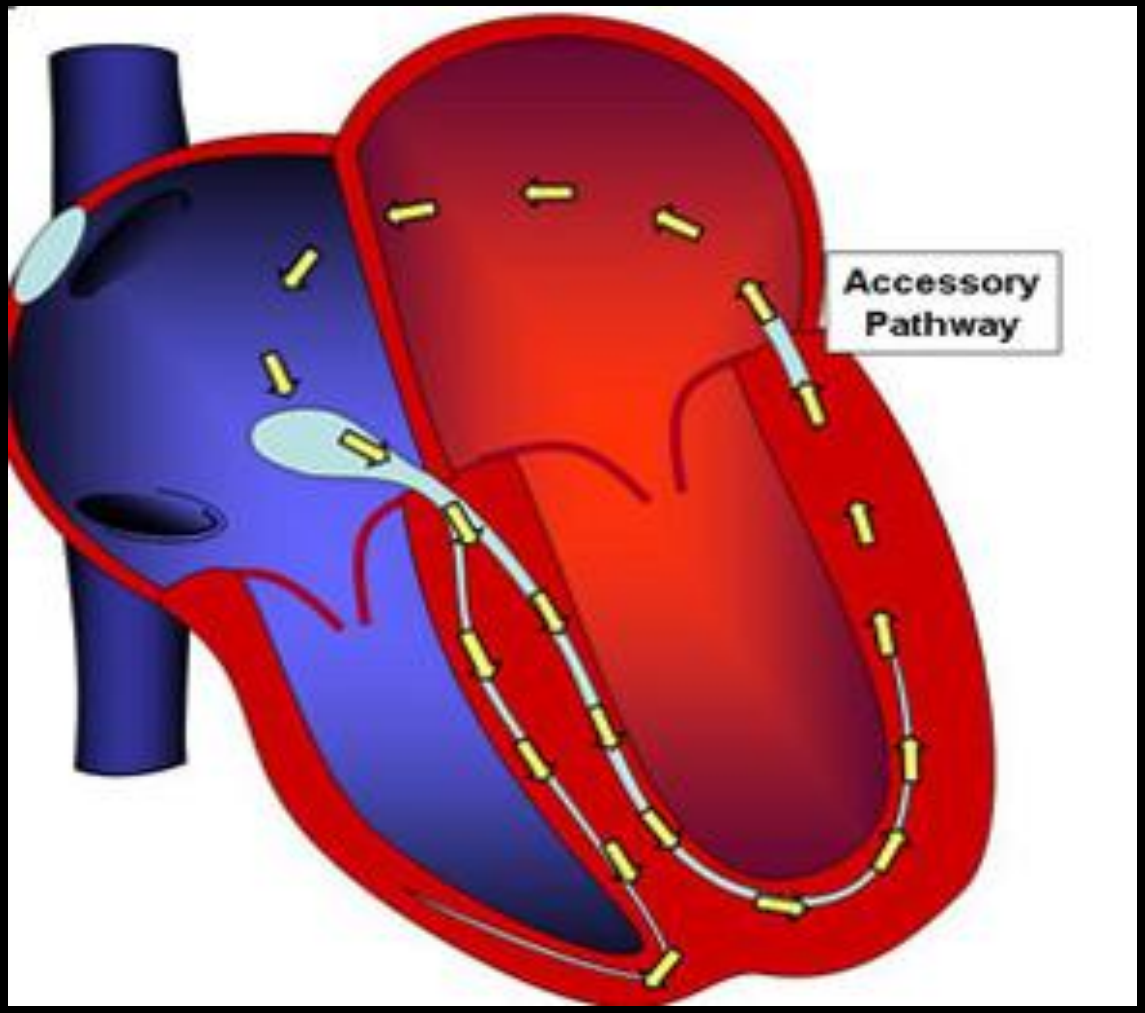
AVNRT

- Typically 3rd and 4th Decade
- Recurrent palpitations
- RAPID onset and RAPID offset
- Patient may feel an ectopic beat to initiate/ terminate the arrhythmia
- Vagal manoeuvres to terminate the arrhythmia
- Anxiety/ breathless/ palpitations
  - Syncope (due to high rate or due to transient asystole at termination)

AVRT

- Due to accessory pathway – abnormal electrical AV conduit
  - Patients can have multiple pathways
- Accessory pathways may conduct
  - Antegradely
  - Retrogradely
  - Combination of the two
- Wolf- Parkinson -White Syndrome
  - Short PR interval (<120ms)
  - Delta wave
  - Palpitations and narrow complex tachycardia

AVRT



Definitions

- Orthodromic
  - Conduction travels in the normal direction (ie A to V)
- Antidromic
  - Conduction travels in an abnormal direction (ie V to A)

AVRT

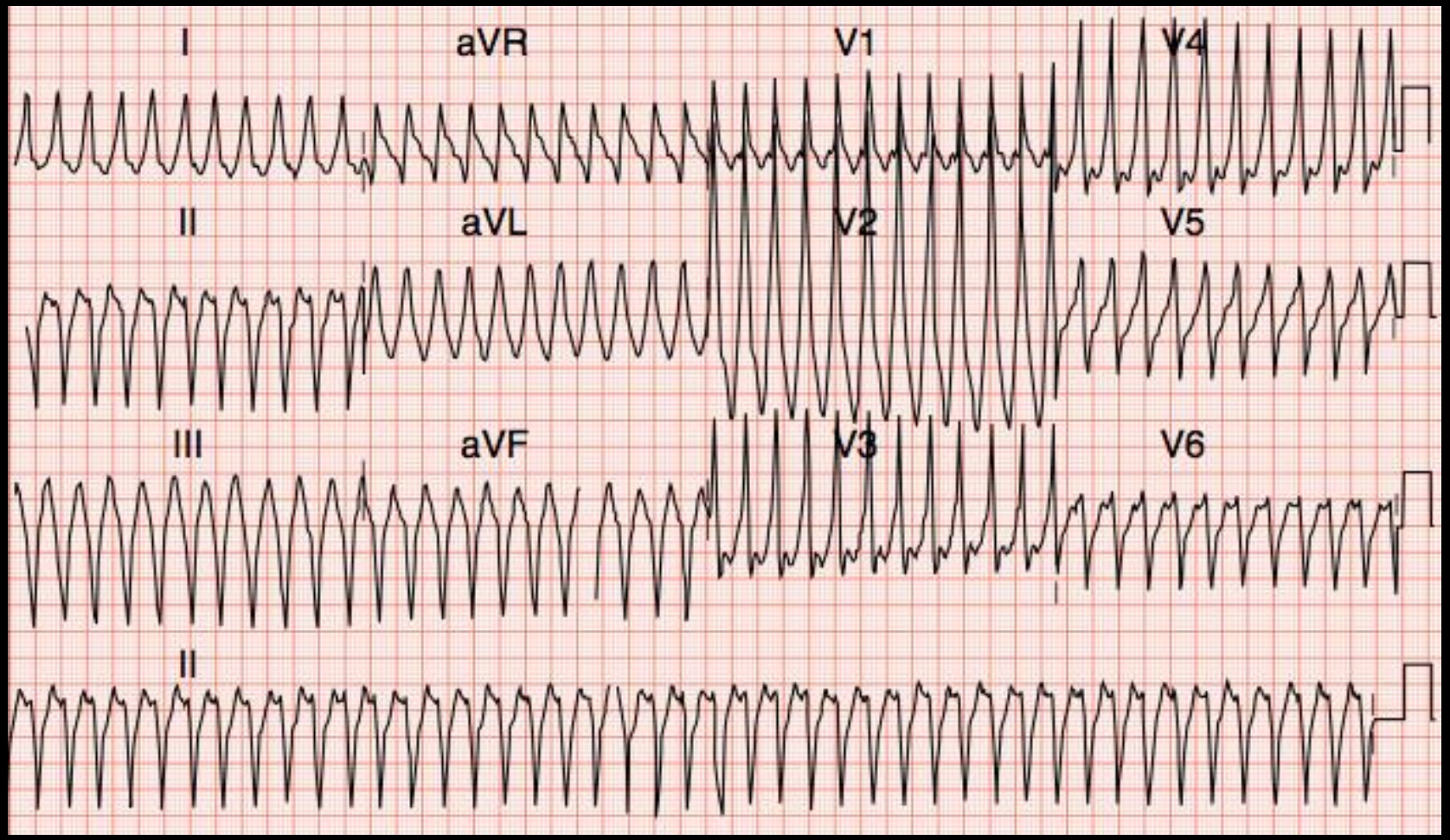
Presentation

- Young patient typically 3rd to 4th decade
- May be asymptomatic- part of a medical
- RAPID onset and RAPID offset
- Patient may feel an ectopic beat to initiate/ terminate the arrhythmia
- Vagal maneuvres to terminate the arrhythmia
- Anxiety/ breathless/ palpitations
  - Syncope (due to high rate or due to transient asystole at termination)

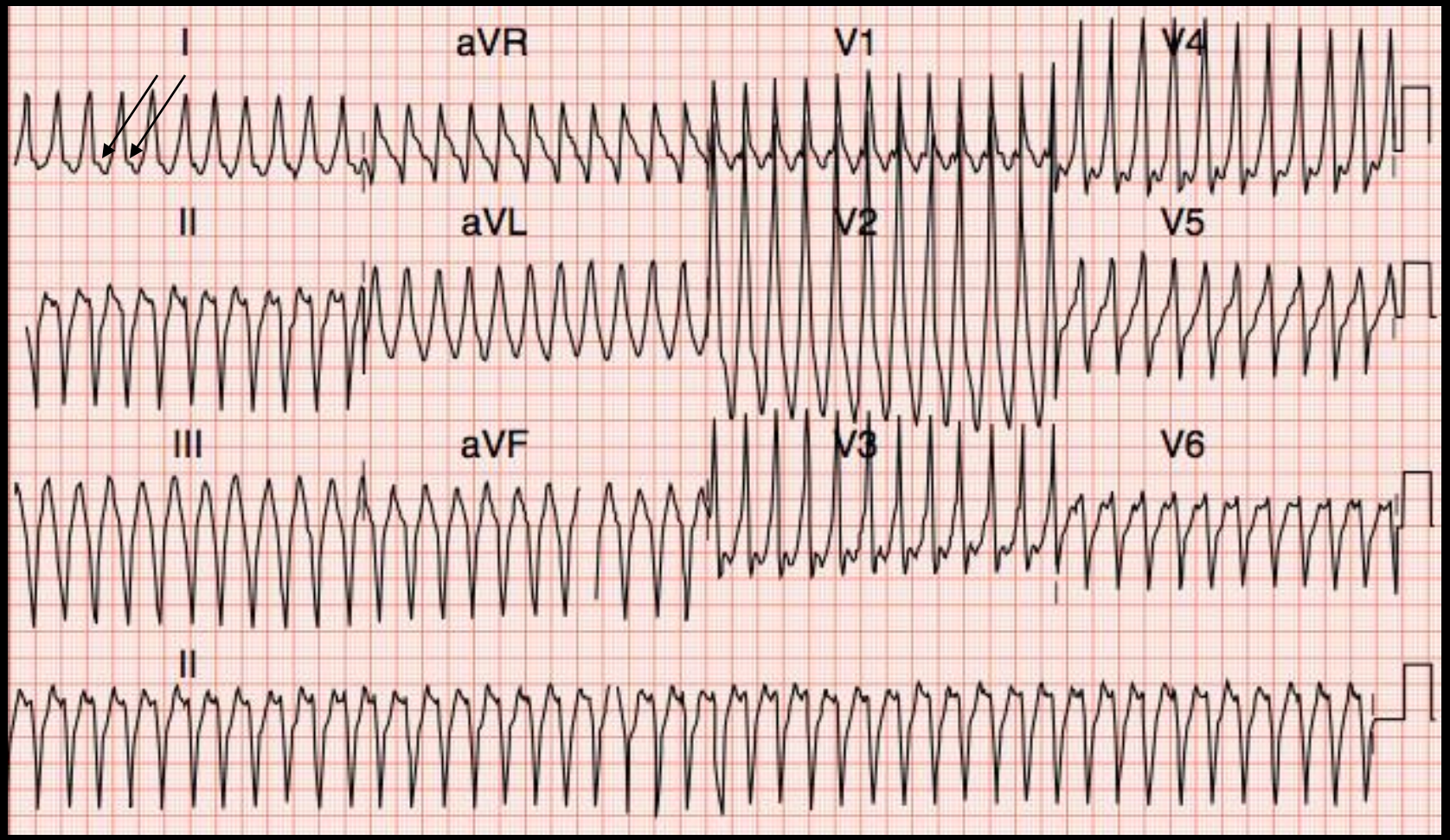
AVRT

- History of structural heart disease
  - Ebstein's anomaly
    - » Multiple right sided accessory pathways
- Family history
  - Higher prevalence in the children; especially if multiple accessory pathways
- Examination
  - Frequently normal

AVRT



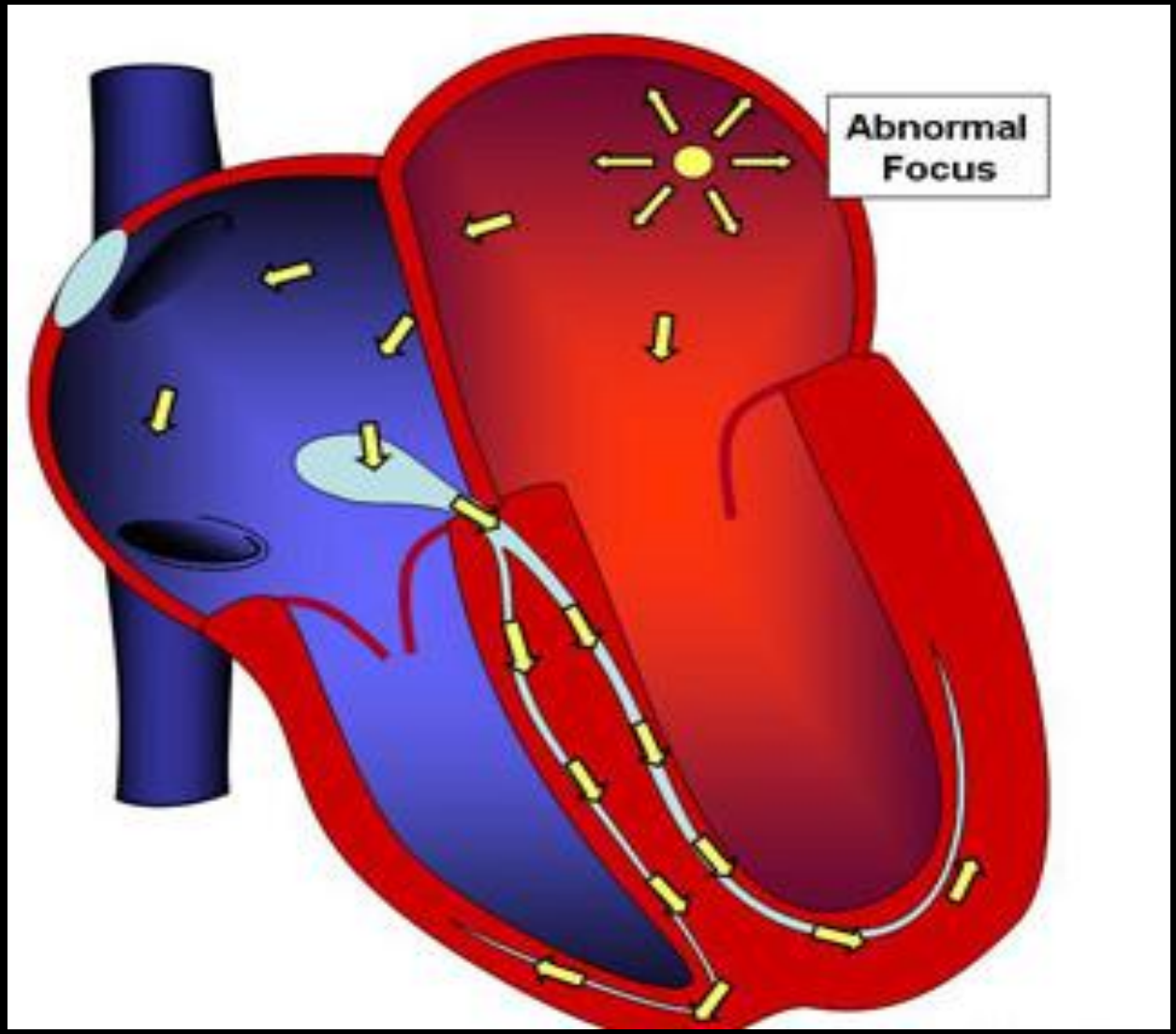
AVRT



Focal Atrial Tachycardia

- Typically older patients >6th decade
- Frequently have structural heart disease, pulmonary disease
- Symptoms are related to
  - Rate (120-250bpm)
  - Underlying heart disease
- Rapid initiation
  - Rate can increase over a few beats as the AV node “warms up”
- No consistent effect with vagal maneuvres
- Digoxin / Alcohol/ Lung disease/ Metabolic derangements

Atrial tachycardia



Focal Atrial Tachycardia

- Regular pulse
  - Exceptions
    - » If atrial tachycardia is fast the AV node may Wenckebach (Mobitz Type I)
    - » If more than one focus (Multifocal atrial tachycardia)
- Check for signs of pulmonary disease
- Cannon A waves
- Variable S1

Atrial Tachyarrhythmias -review

AV Node dependent:

- Atrio-ventricular nodal re-entrant tachycardia (AVNRT)
- Atrioventricular re-entrant tachycardia (AVRT)

AV Node independent:

- Atrial fibrillation (AF)
- Atrial flutter
- Atrial tachycardia (AT)
- Sinus tachycardia
- Inappropriate sinus tachycardia
- Atrial premature beats

Ventricular Tachycardia

- May be asymptomatic
- Heart rate is NOT a useful guide to the arrhythmia
- More likely if
  - Previous MI / History of IHD
  - Cardiac risk factors
- Sudden onset/ offset
- Is it recurrent?
- Do they have a pacemaker or an ICD
- Family History
  - Sudden cardiac death
  - Unexplained death
  - HOCM/ Long QT syndrome / Brugada

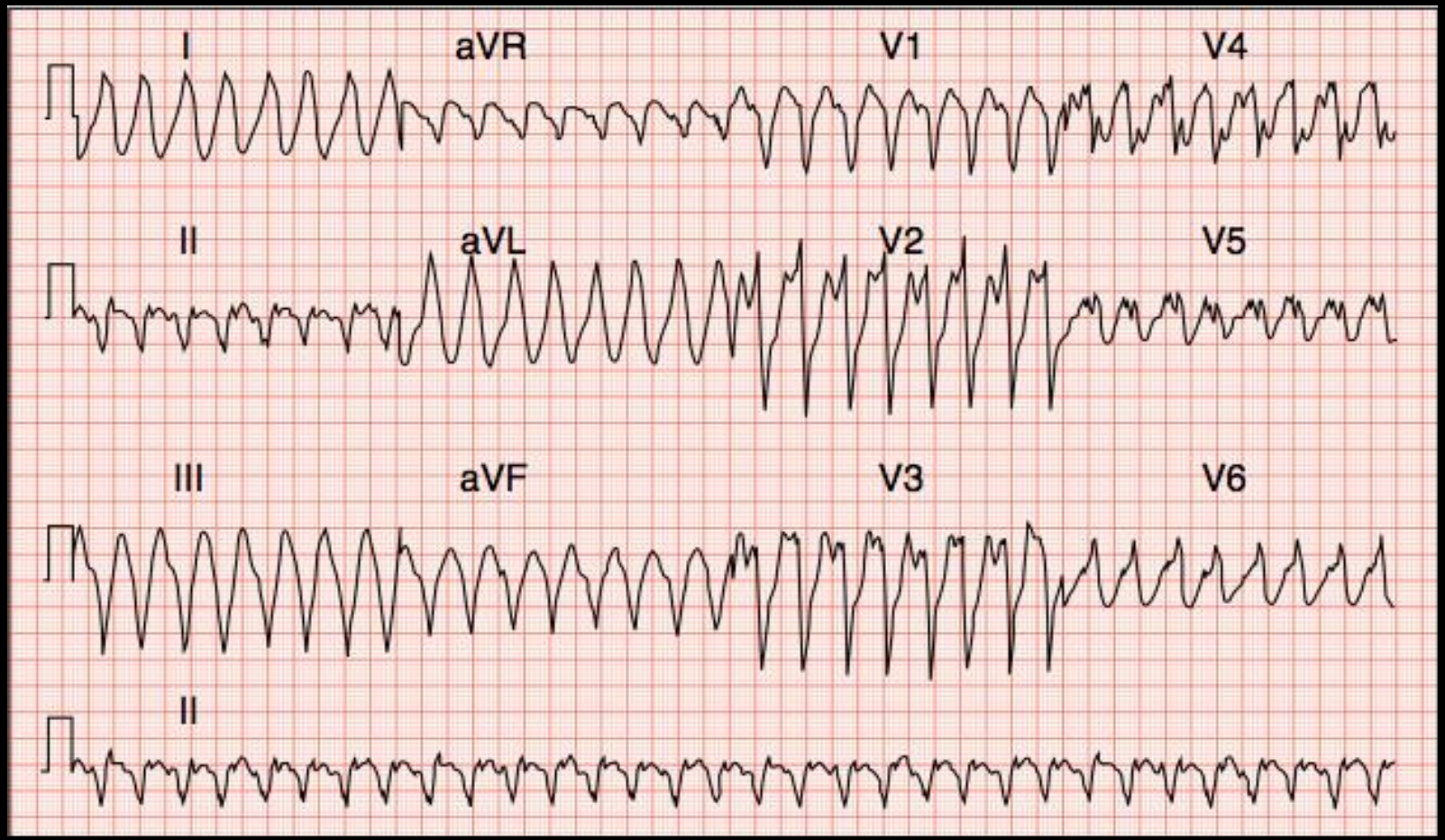
Physical Examination

- Is the patient compromised?
  - DC cardioversion if any doubt
- Assess the JVP
  - Cannon A waves ?
- Assess the praecordium
  - Pacemaker/ ICD/ Median sternotomy scar / LV Heave/ Double apical impulse?
- Ausculate
  - Variable S1; Ejection systolic murmer

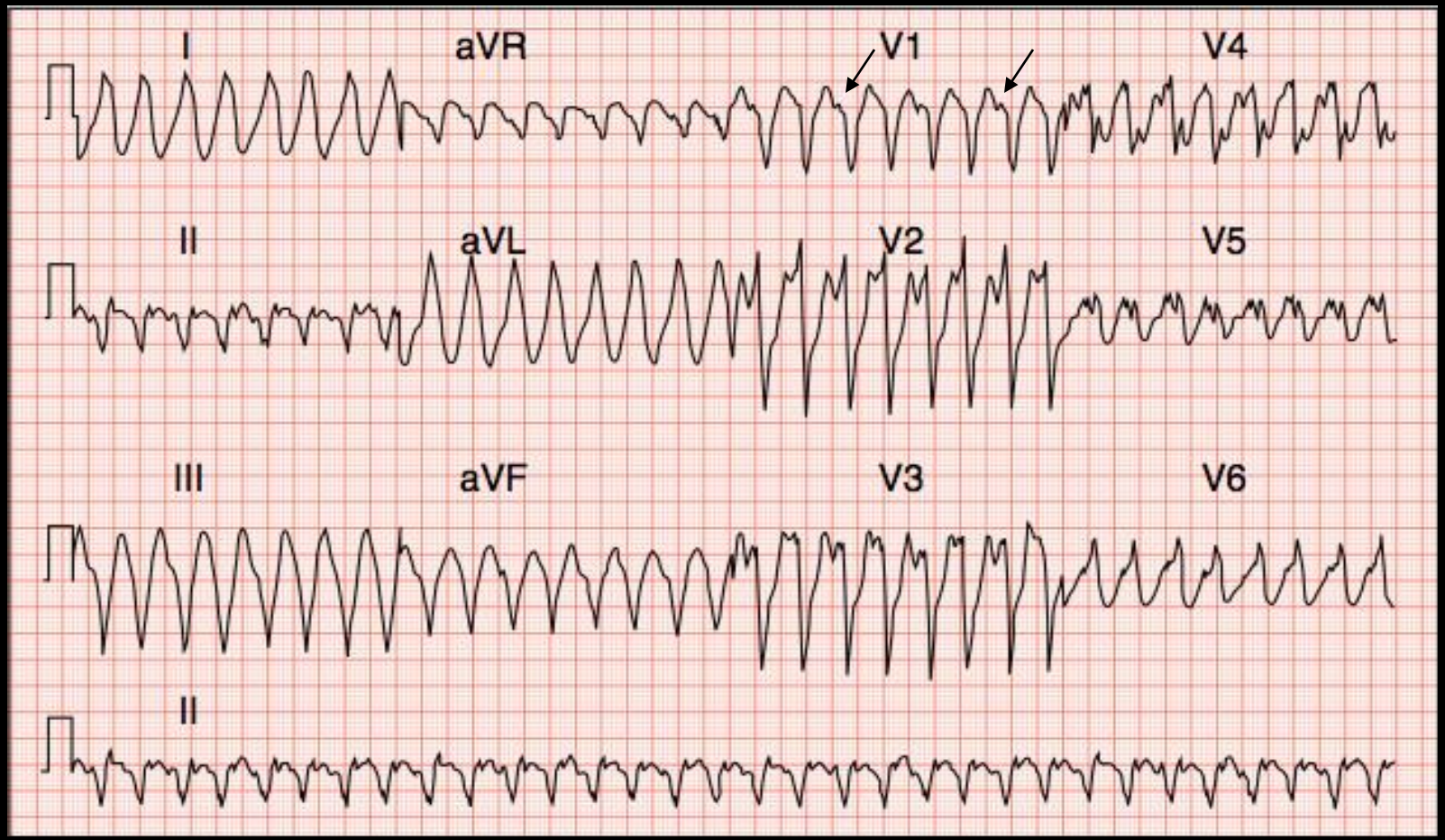
ECG Findings- VT

- Regular broad complex tachycardia (QRS > 120ms)
  - Normally RBBB >140ms
  - LBBB>160ms
- Evidence of A-V Dyssynchrony
- Fusion beats
- Capture beats
- Concordance
- If a 12 lead in sinus rhythm is available
  - ?Q waves; Delta waves; RBBB and ST Elevation

Ventricular Tachycardia



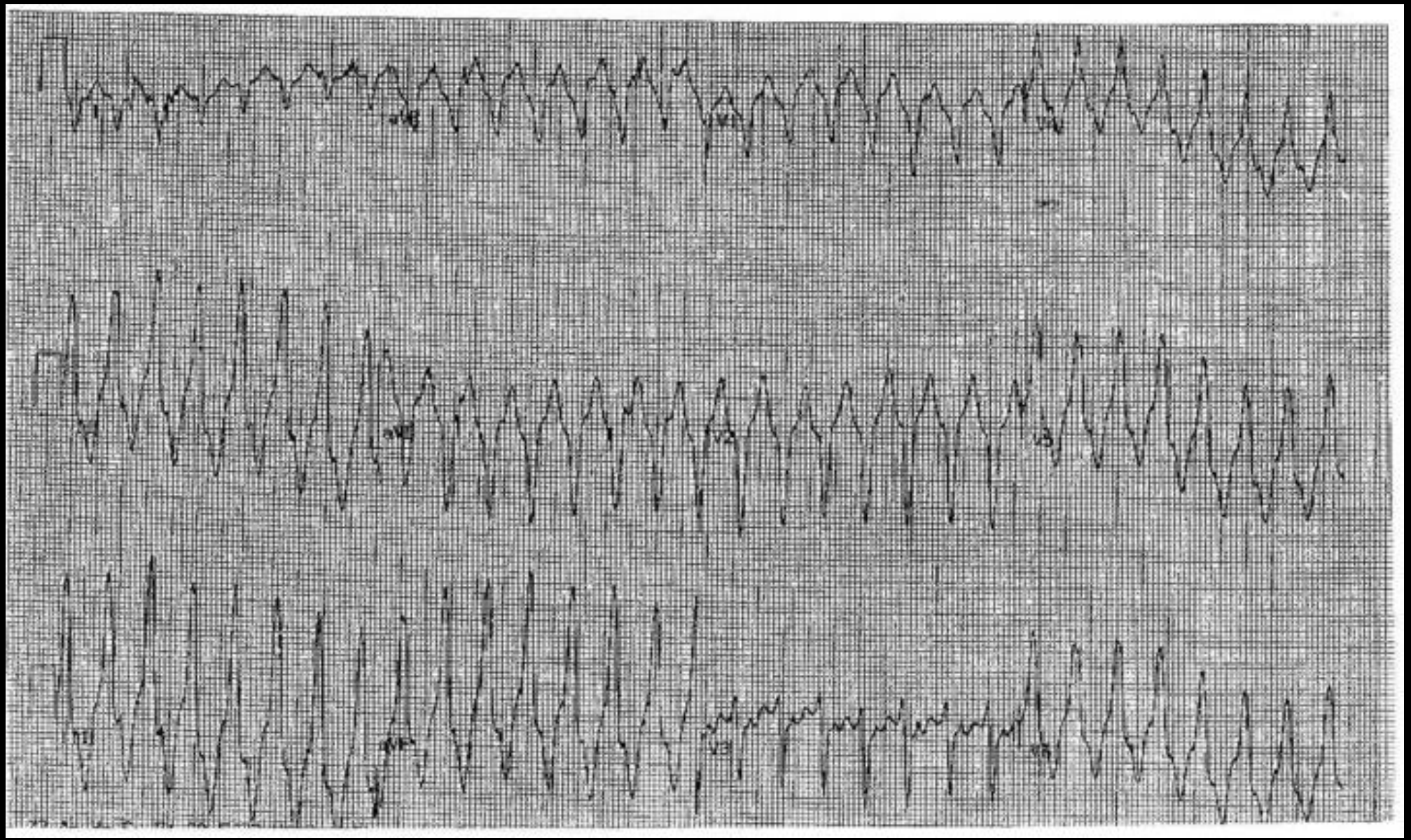
Ventricular Tachycardia



Right Ventricular Outflow Tract Tachycardia (RVOT VT)

- Young patients
- Atheletic
- Occur during exercise
- Can be terminated by vagal manouevres

- ECG Findings
  - LBBB morphology in V1
  - Inferior axis



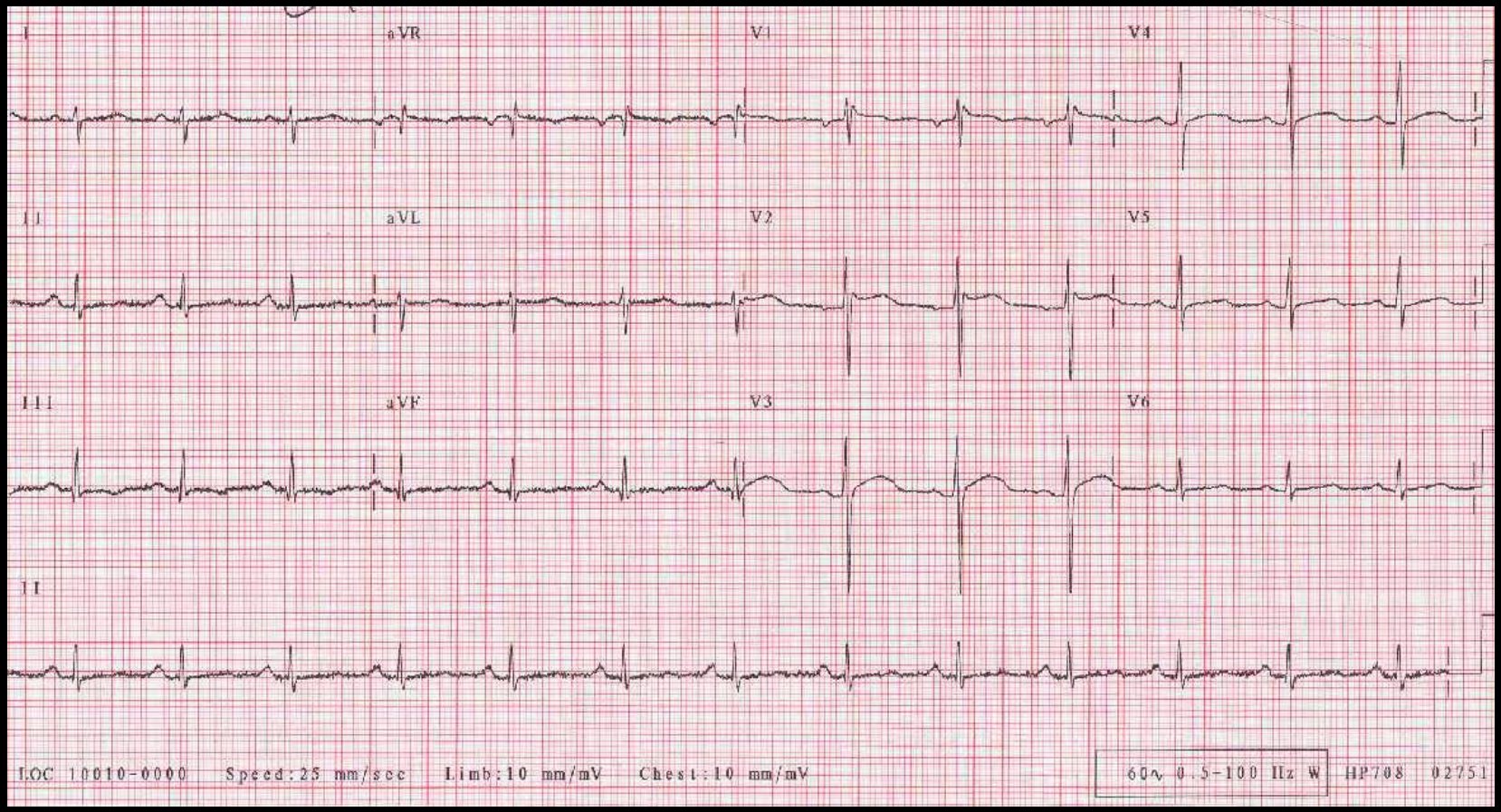
Ventricular Fibrillation

- No cardiac output
  - DC Cardioversion
- Normally cause is evident
  - Myocardial ischaemia
  - Cardiomyopathy- DCM/ HCM/ HOCM
  - Torsade de pointes and causes of long QT syndrome
  - Brugada syndrome
  - Commotio Cordis

Brugada Syndrome

- Due to a mutation in a sodium channel (SCN5A)
- 1st presentation may be failed sudden cardiac death
- Family history
- ECG
  - Right bundle branch block
  - ST elevation in the anterior precordial chest leads (V1-3)
- No evidence of structural heart disease

Brugada Syndrome



Summary

- The arrhythmia must be seen in the context of the patient
  - Not just the ECG
- The state of the patient will depend on the heart rate and underlying heart disease *not* the arrhythmia per se
- The age of the patient, and associated disease can guide the provisional differential diagnosis before seeing the ECG
- Examine for signs of AV dissociation