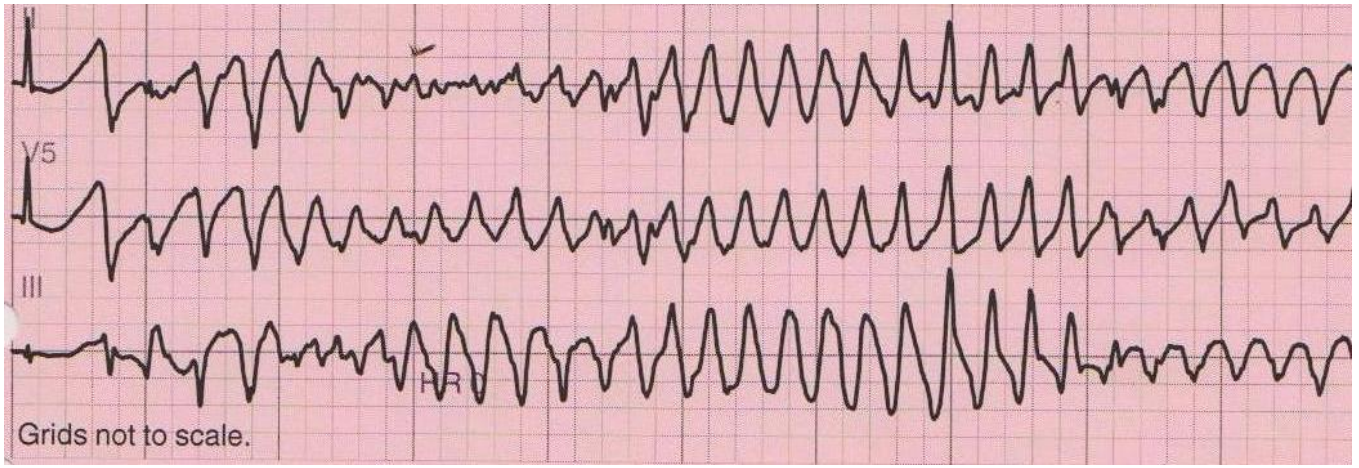
Update on Palpitations and AF

February 28th 2018



Dr Mrinal Andrew Saha

MA(Cantab) MBBS FRCP PhD

Consultant Interventional Cardiologist

GHNHSFT

Dr Mrinal Saha

Appointed 2010

Special interests:

Angioplasty, stents
Heart valve disease
Palpitations
Heart failure

Objectives

1. ECG interpretation: a systematic approach

2. Whom should I refer?
   - Red flags in the history and examination
   - Red flags in the 24 hr tape report
   - Should I refer everyone with a new diagnosis of AF?
   - Does everyone with AF need follow up?

3. Interpretation of a 24hr tape report

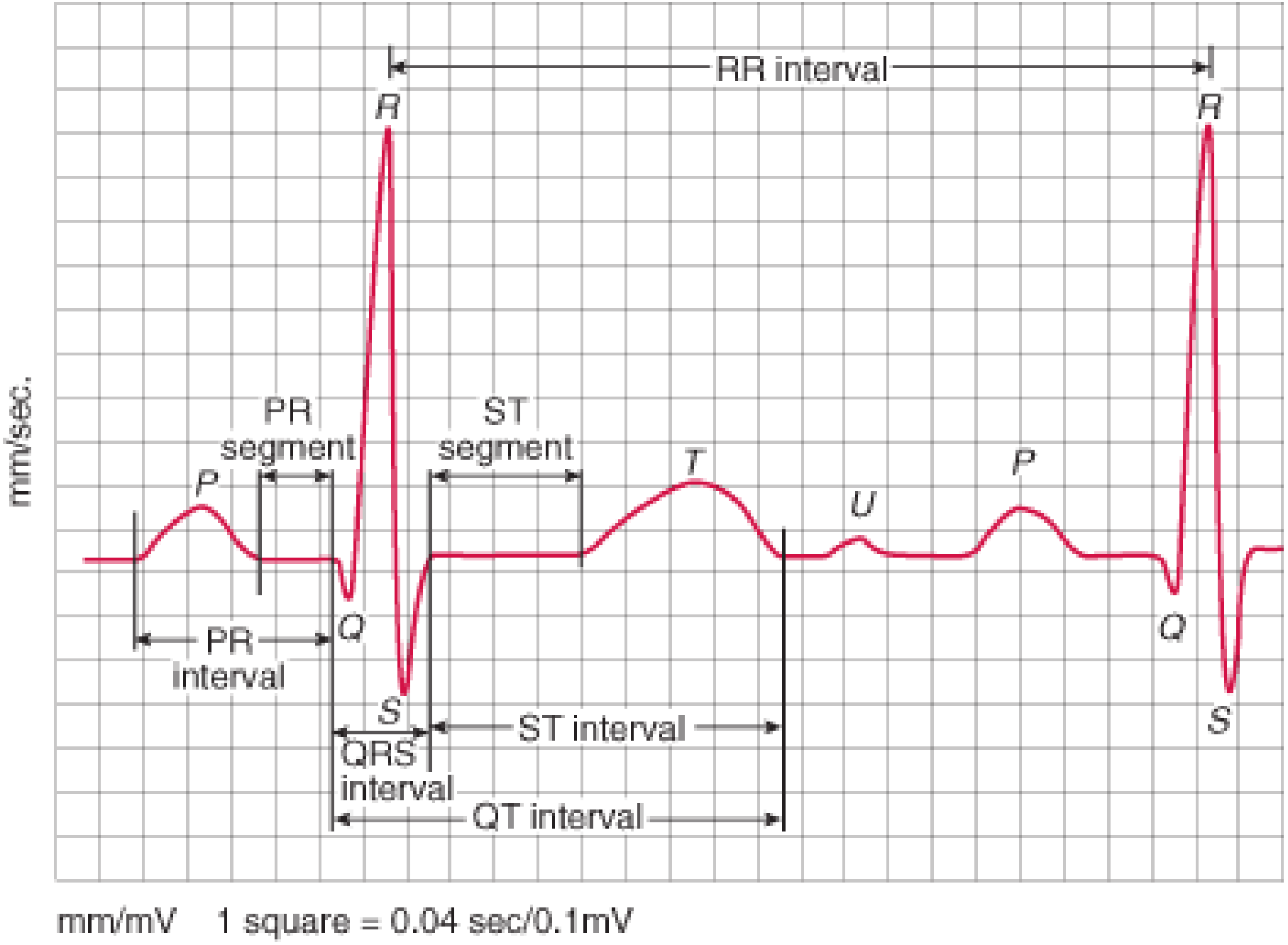
4. What to do if AF is identified in clinic

5. Treatment options for common arrhythmias

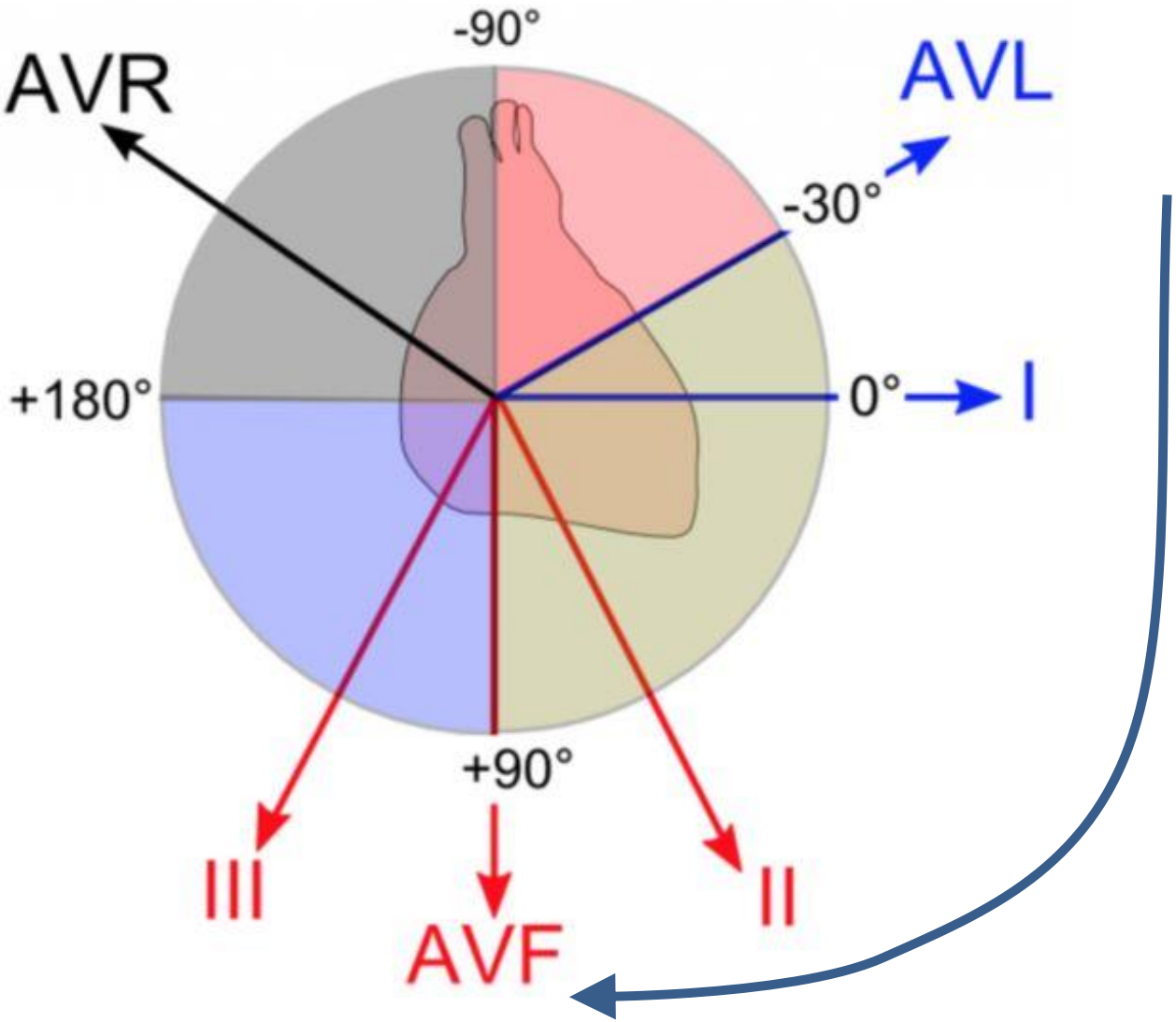
ECG analysis: a systematic approach

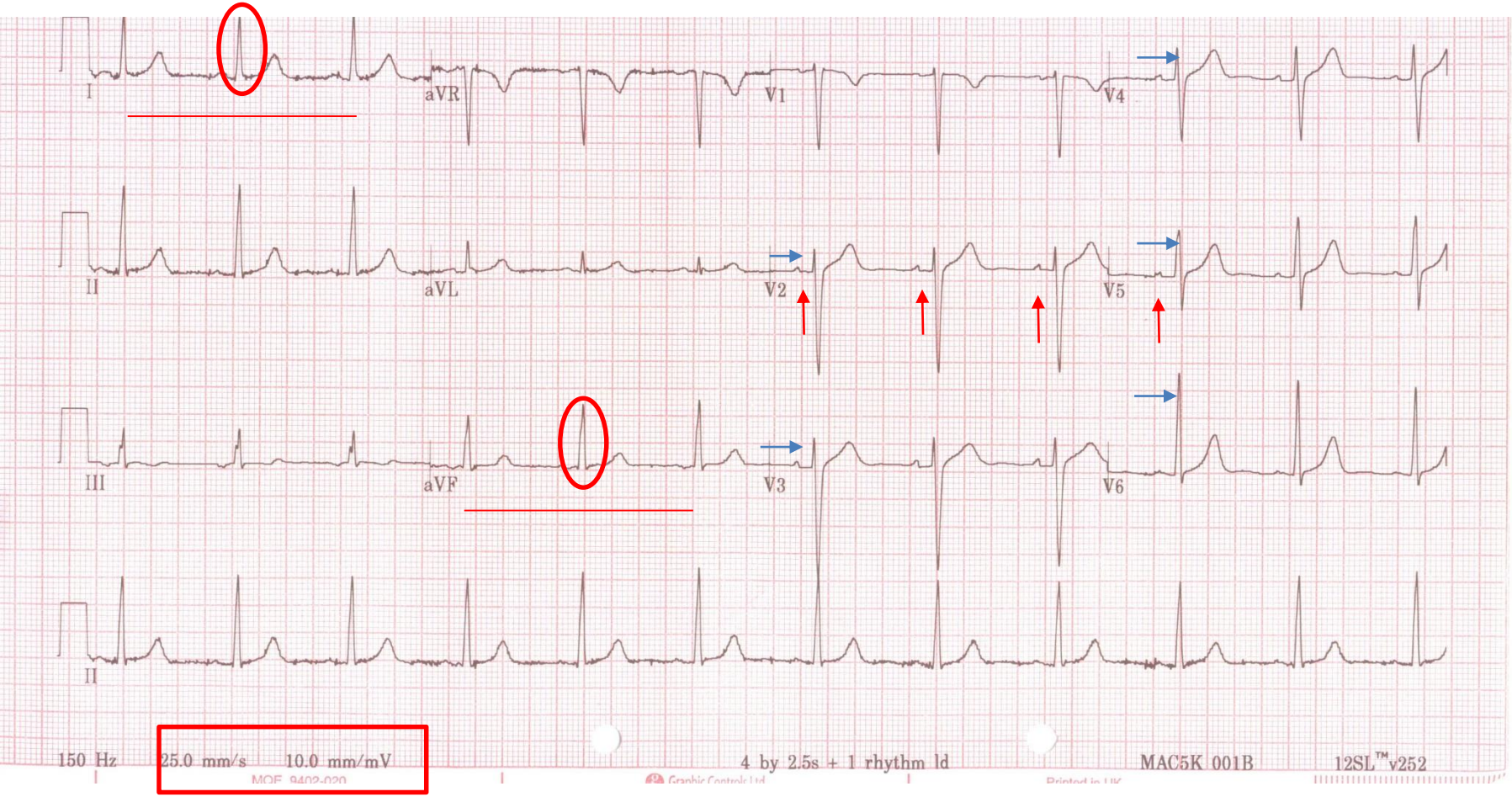
Is it regular or irregular
What is the ventricular rate
Can you see P waves
Are the P waves followed by a QRS
Is the QRS narrow or wide
What are the ST segments doing

PATTERN RECOGNITION


RR interval
R
R
PR
segment
ST
segment
P
T
U
P
PR
interval
Q
Q
S
S
ST interval
QRS
interval
QT interval
mm/sec.
mm/mV
1 square = 0.04 sec/0.1mV

ECG Axis

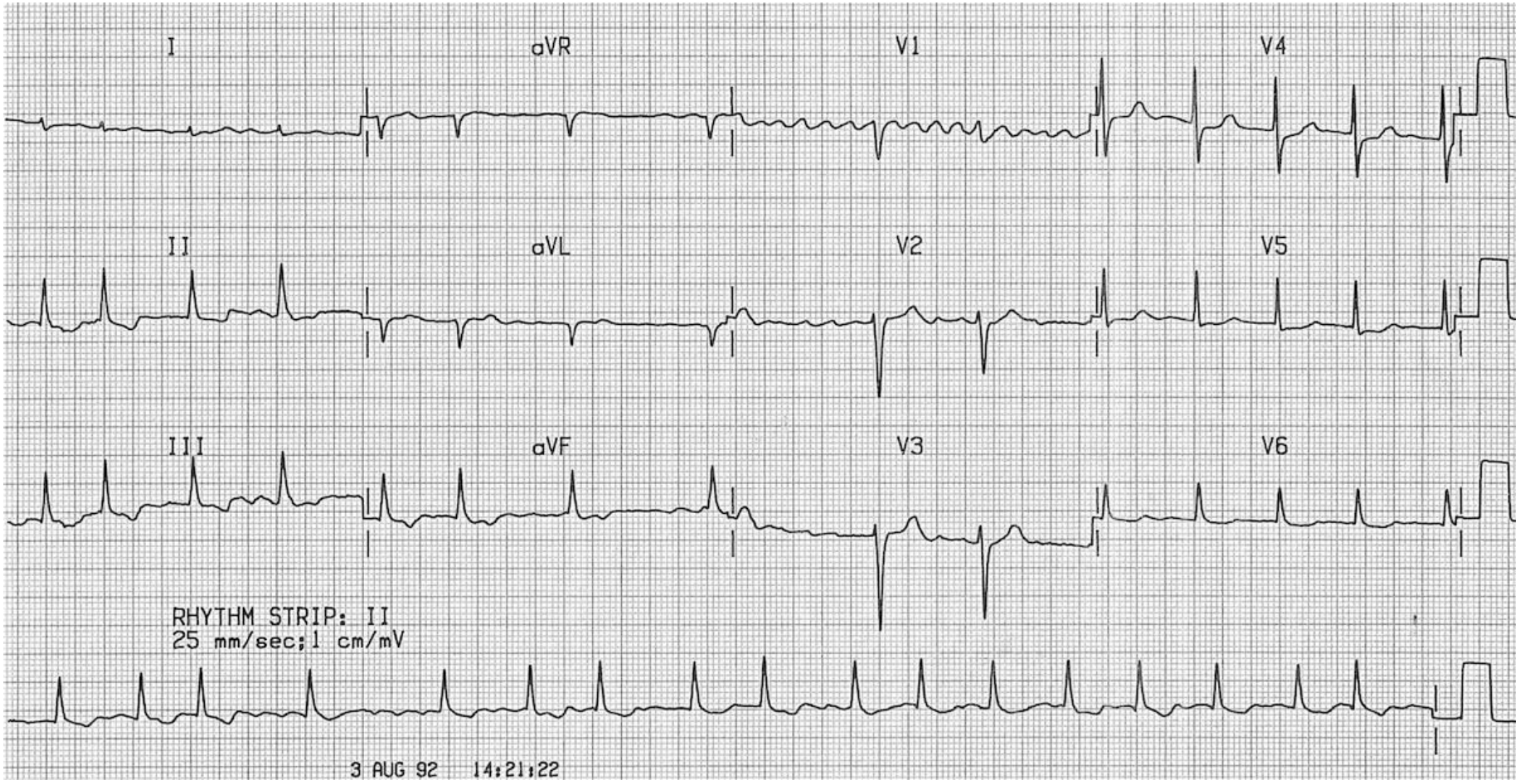




Sinus rhythm
72/min
Normal R wave progression
Normal Axis

Case 1

65 yr old woman
2 week history of palpitations
Murmur
BP 100/60
Clear chest, no oedema



Management?

Check bloods- U+E, TSH, FBC
Anticoagulate (if bleeding risk low)
Refer for echo (moderate MR)+/- other tests
Hold off beta blockade/ digoxin

Cardiovascular morbidity and mortality associated with AF

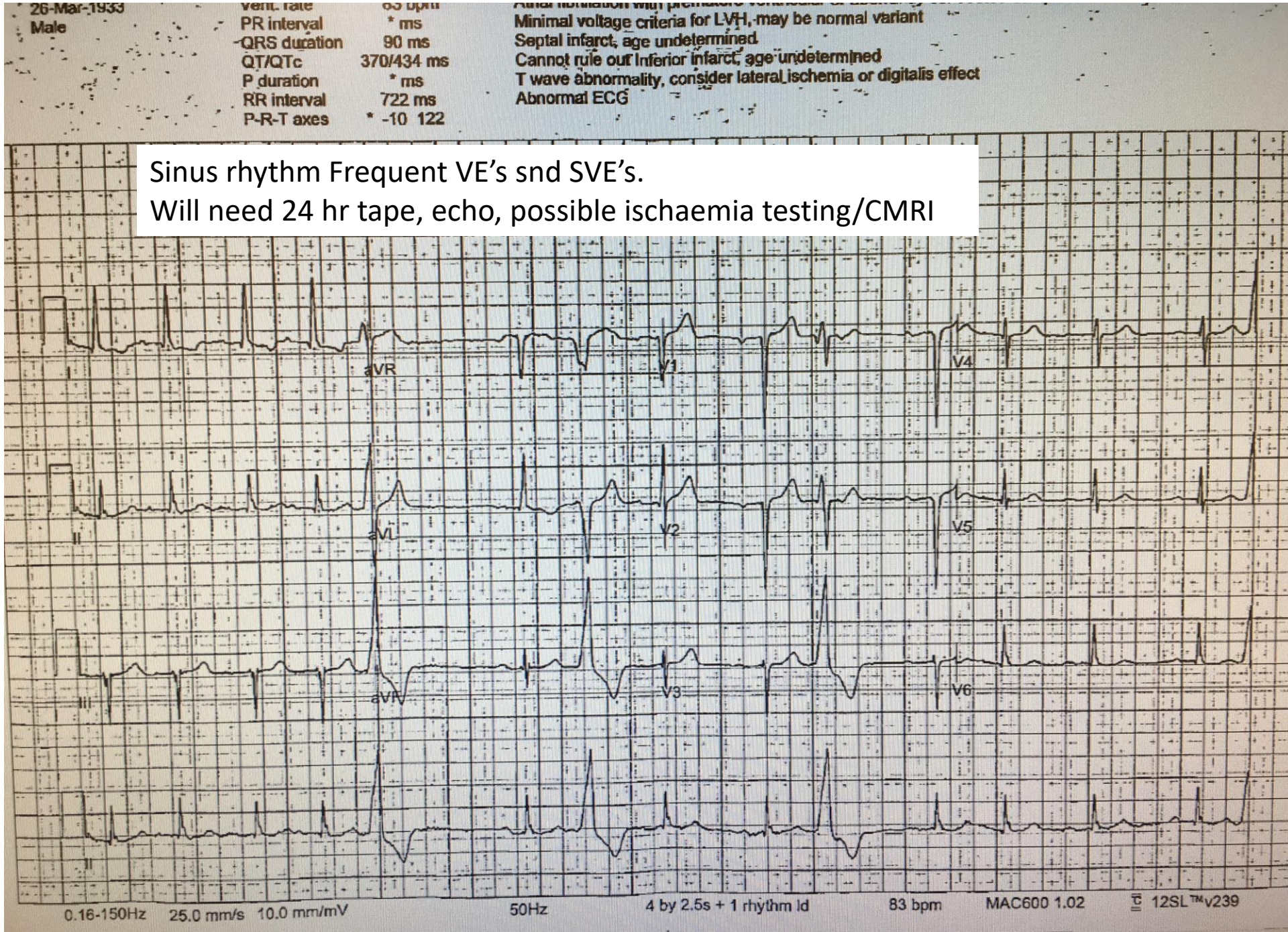
Event	Association with AF
Death	Increased mortality, especially cardiovascular mortality due to sudden death, heart failure or stroke.
Stroke	20–30% of all strokes are due to AF. A growing number of patients with stroke are diagnosed with 'silent', paroxysmal AF.
Hospitalizations	10–40% of AF patients are hospitalized every year.
Quality of life	Quality of life is impaired in AF patients independent of other cardiovascular conditions.
Left ventricular dysfunction and heart failure	Left ventricular dysfunction is found in 20–30% of all AF patients. AF causes or aggravates LV dysfunction in many AF patients, while others have completely preserved LV function despite long-standing AF.
Cognitive decline and vascular dementia	Cognitive decline and vascular dementia can develop even in anticoagulated AF patients. Brain white matter lesions are more common in AF patients than in patients without AF.

Case 2

I would be grateful for your opinion on this man's ECG. He came for a routine review with our nurses who noticed an irregular pulse. His ECG I think shows atrial fibrillation but I am not absolutely certain as I thought I could see the odd P wave. He had an ECG done earlier this year as well which appears to show sinus rhythm with some extra beats.

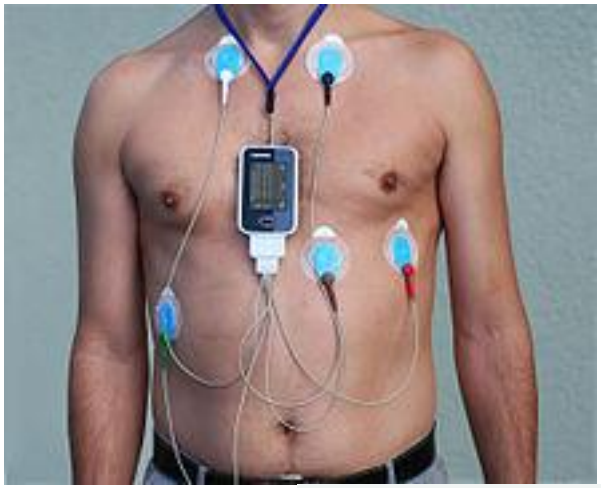
He is completely asymptomatic but he does have a history of ischaemic heart disease (he had a coronary artery bypass graft and an aortic valve replacement in 2012). He also has a diagnosis of heart failure. I have commenced him on Rivaroxaban pending your opinion; he also takes Lansoprazole 30mg OD, Ramipril 1.25mg OD, and Simvastatin 40mg at night. Thank you for your opinion.

Yours sincerely

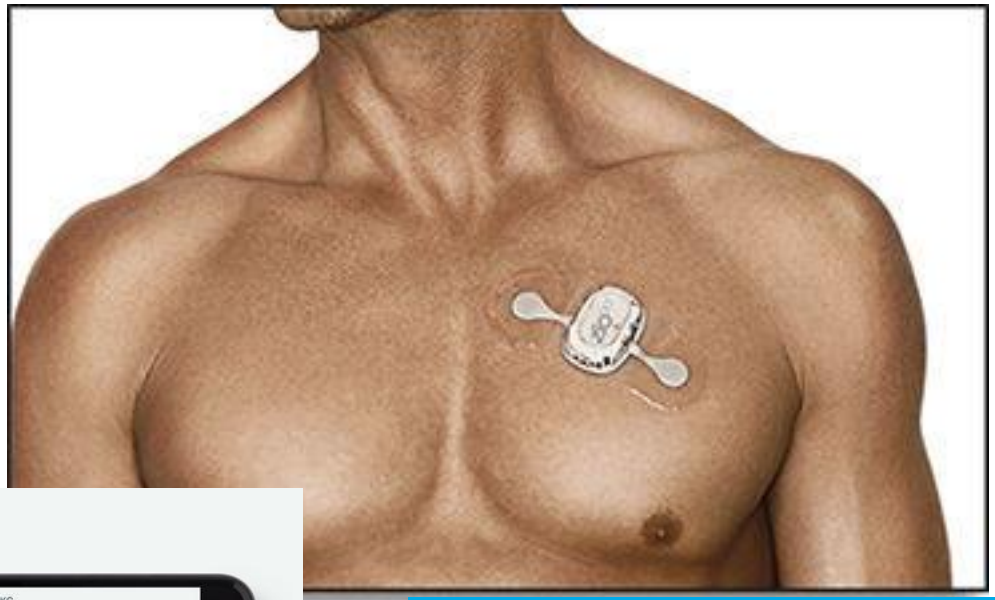

26-Mar-1933
Male
Vent. rate 63 bpm
PR interval * ms
QRS duration 90 ms
QT/QTc 370/434 ms
P duration * ms
RR interval 722 ms
P-R-T axes * -10 122
Minimal voltage criteria for LVH, may be normal variant
Septal infarct, age undetermined
Cannot rule out Inferior infarct, age undetermined
T wave abnormality, consider lateral ischemia or digitalis effect
Abnormal ECG
Sinus rhythm Frequent VE's snd SVE's.
Will need 24 hr tape, echo, possible ischaemia testing/CMRI
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
0.16-150Hz 25.0 mm/s 10.0 mm/mV 50Hz 4 by 2.5s + 1 rhythm ld 83 bpm MAC600 1.02 12SL™v239

Heart monitors

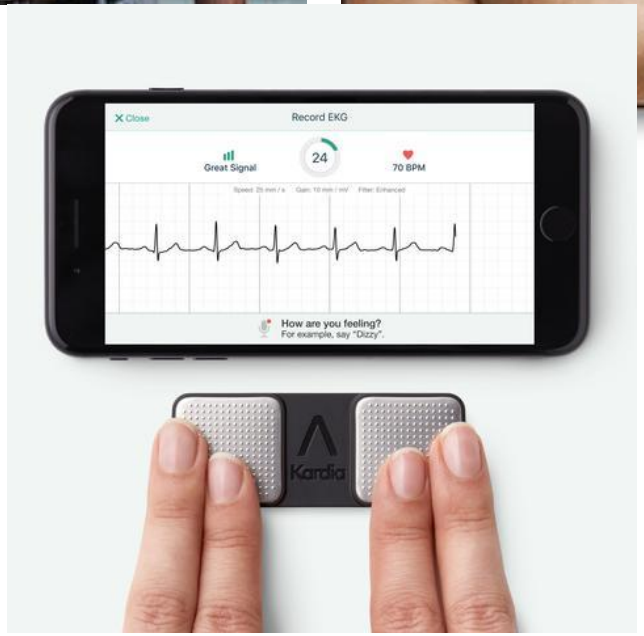
Holter



ziopatch

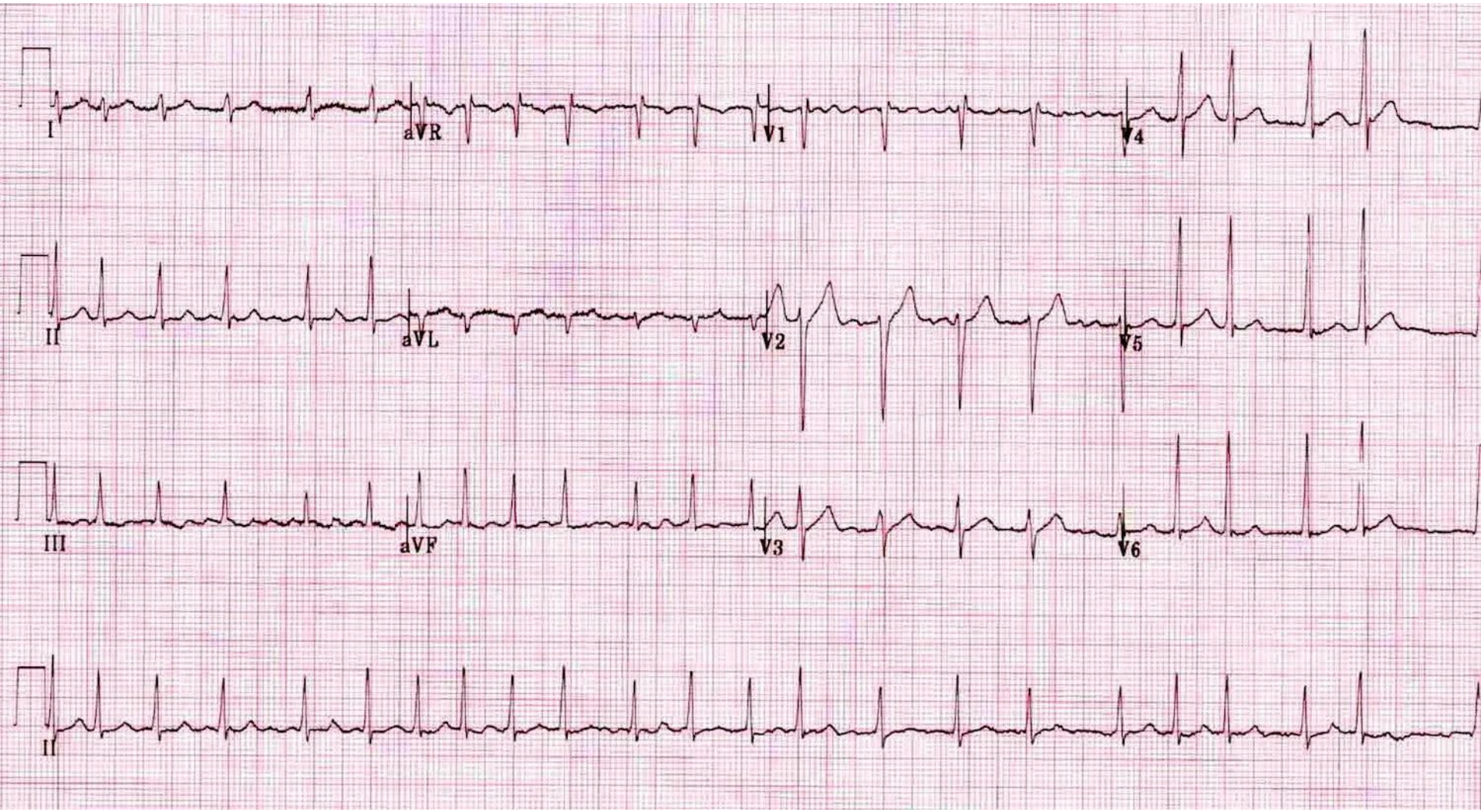


Kardia by Alivecor



Case 3

80 yr old man
Hx of CABG
Short of breath for 6 weeks
BP 90/60
Pulse 138/min irregular
Bibasal crackles, pitting oedema
sO2= 92%, rr 24/min



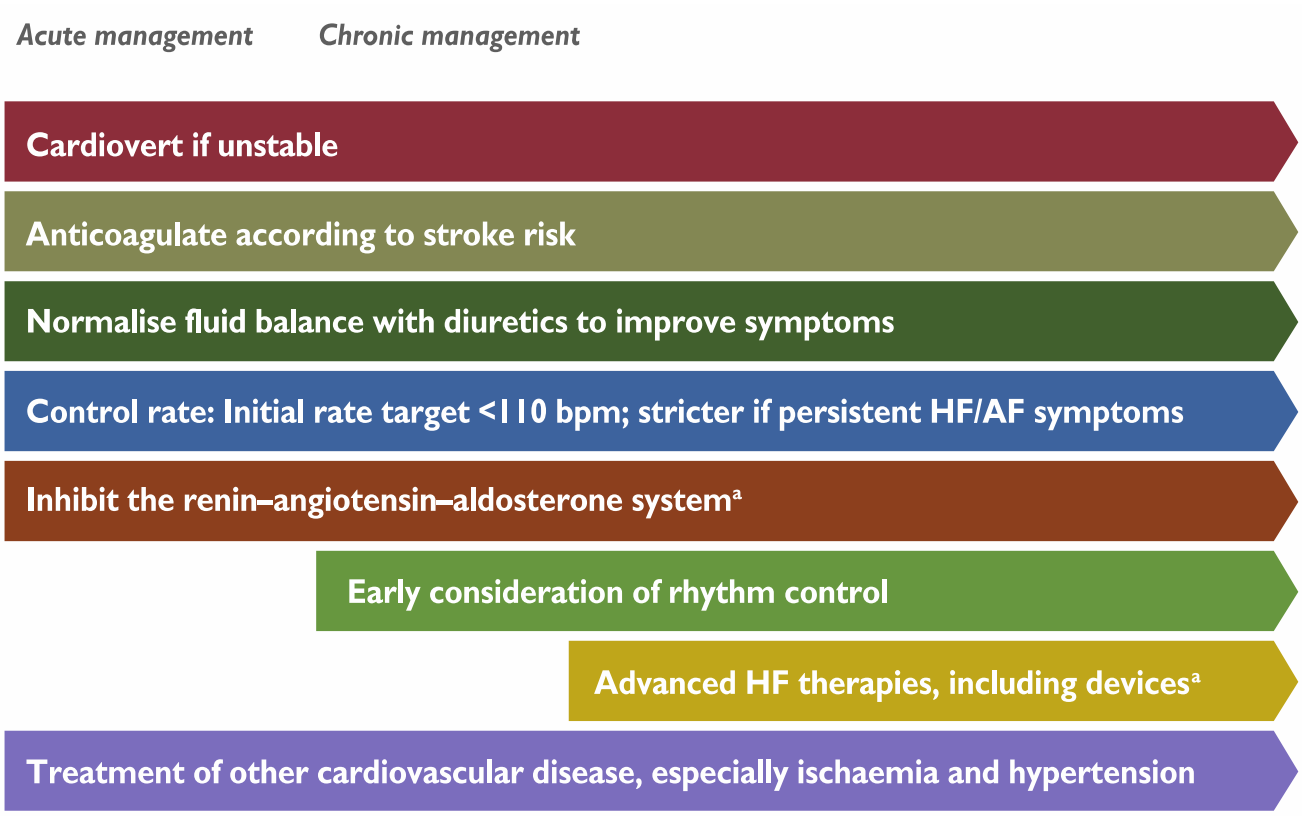
Management?

Send to A+E!
Check bloods, CXR
Cardiovert and anticoagulate
Diurese, amiodarone iv then oral
Echo, angiogram

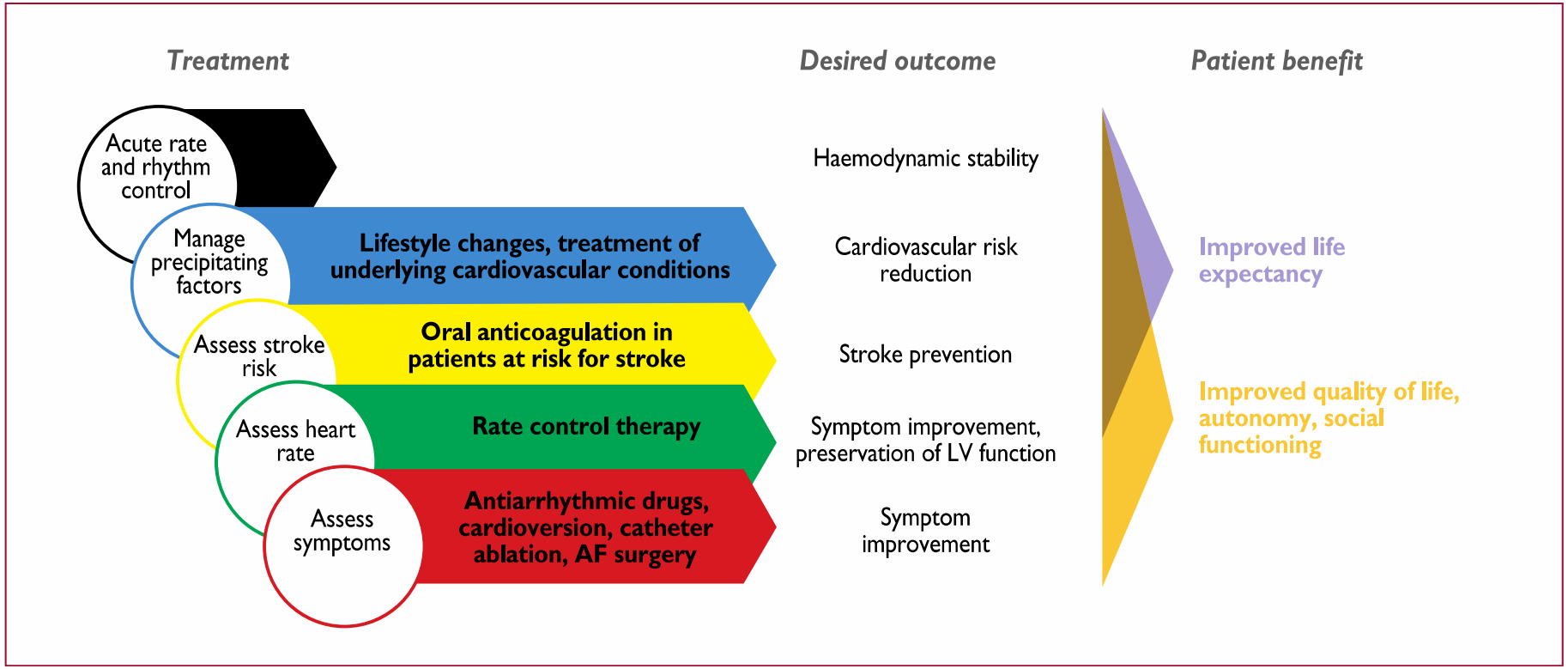
Red flags: Clinical Signs prompting urgent referral to secondary care

Haemodynamic instability
Uncontrollable rate
Symptomatic bradycardia not amenable to reduction of rate control agents
Severe angina or worsening left ventricular function
Transient ischaemic attack or stroke

Management of patients presenting acutely with AF and heart failure

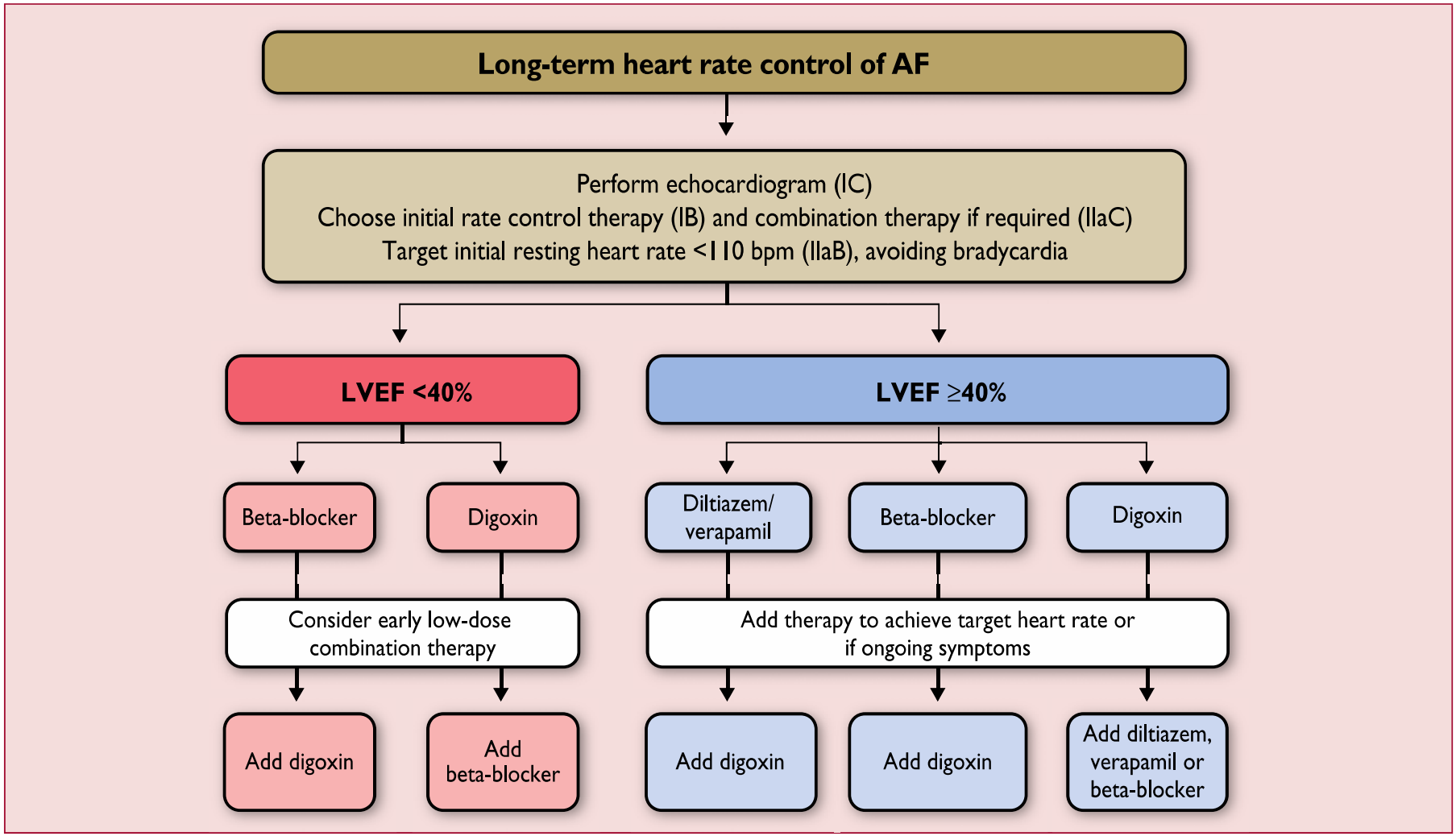


Acute and chronic management of patients with AF



AF = atrial fibrillation; LV = left ventricular.

Drugs for ventricular rate control in AF



Type of AF by Pathophysiology

AF type	Clinical presentation	Possible pathophysiology
AF secondary to structural heart disease	AF in patients with LV systolic or diastolic dysfunction, long-standing hypertension with LVH, and/or other structural heart disease. The onset of AF in these patients is a common cause of hospitalization and a predictor of poor outcome.	Increased atrial pressure and atrial structural remodelling, together with activation of the sympathetic and renin-angiotensin system.
Focal AF	Patients with repetitive atrial runs and frequent, short episodes of paroxysmal atrial fibrillation. Often highly symptomatic, younger patients with distinguishable atrial waves (coarse AF), atrial ectopy, and/or atrial tachycardia deteriorating in AF.	Localized triggers, in most cases originating from the pulmonary veins, initiate AF. AF due to one or a few re-entrant drivers is also considered to be part of this type of AF.
Polygenic AF	AF in carriers of common gene variants that have been associated with early onset AF.	Currently under study. The presence of selected gene variants may also influence treatment outcomes.
Post-operative AF	New onset of AF (usually self-terminating) after major (typically cardiac) surgery in patients who were in sinus rhythm before surgery and had no prior history of AF.	Acute factors: inflammation, atrial oxidative stress, high sympathetic tone, electrolyte changes, and volume overload, possibly interacting with a pre-existing substrate.
AF in patients with mitral stenosis or prosthetic heart valves	AF in patients with mitral stenosis, after mitral valve surgery and in some cases other valvular disease.	Left atrial pressure (stenosis) and volume (regurgitation) load are the main drivers of atrial enlargement and structural atrial remodelling in these patients.
AF in athletes	Usually paroxysmal, related to duration and intensity of training.	Increased vagal tone and atrial volume.
Monogenic AF	AF in patients with inherited cardiomyopathies, including channelopathies.	The arrhythmogenic mechanisms responsible for sudden death are likely to contribute to the occurrence of AF in these patients.

8.4 Structured follow-up

Most AF patients need regular follow-up to ensure continued optimal management. Follow-up may be undertaken in primary care, by specially trained nurses, by cardiologists, or by AF specialists.[325,330] A specialist should co-ordinate care and follow-up. Follow-up should ensure implementation of the management plan, continued engagement of the patient, and therapy adaptation where needed.

Goal-based follow up of AF patients

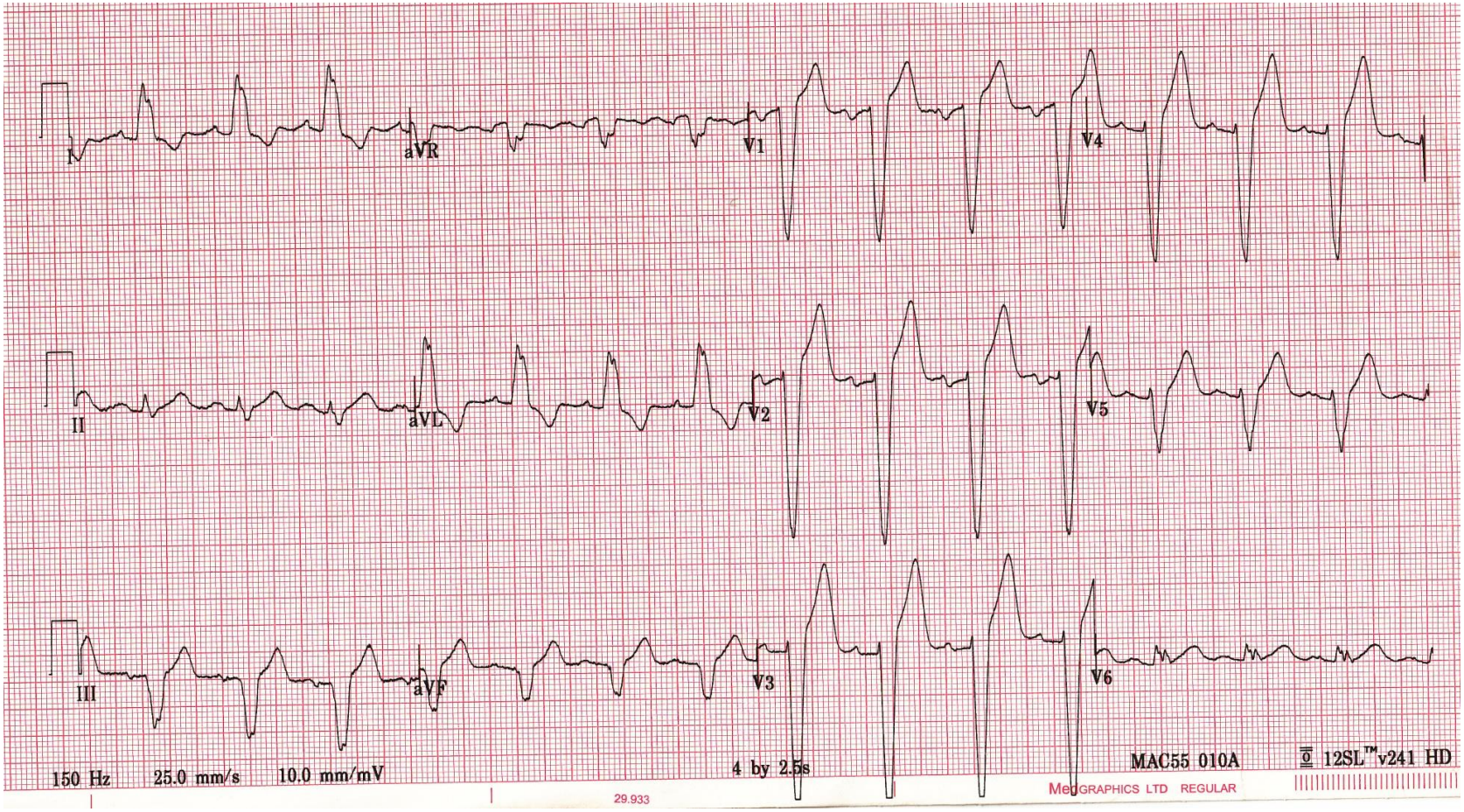
Category	Intervention	Follow-up aspects	Performance indicator (examples)
Prognostic	Comorbidity control (relevant examples given)	Obesity Arterial hypertension Heart failure Coronary artery disease Diabetes Valvular heart disease	Weight loss Blood pressure control Heart failure therapy and hospitalizations Statin and antiplatelet therapy; revascularization Glycaemic control Valve repair or replacement
Prognostic	Anticoagulation	Indication (risk profile; timing, e.g. post-cardioversion). Adherence (NOAC or VKA) and INR (if VKA). NOAC dosing (co-medications; age; weight; renal function).	Stroke Bleeding Mortality
Mainly symptomatic Partly prognostic	Rate control	Symptoms Average resting heart rate <110 bpm	Modified EHRA score Heart failure status LV function Exercise capacity Hospitalization Therapy complications
Symptomatic at present	Rhythm control	Symptoms vs. side effects Exclusion of pro-arrhythmia (PR; QRS; QTc interval)	
Relevant for implementation of therapy and adherence	Patient education and self-care capabilities	Knowledge (about disease; about treatment; about management goals) Capabilities (what to do if…)	Adherence to therapy Directed evaluation, preferably based on systematic checklists
Relevant for chronic care management	Caregiver involvement	Who? (spouse; GP; home nurse; pharmacist) Clearly spelling out participation roles Knowledge and capabilities	Directed evaluation of task performance (e.g. via patient card) Dispensed medication Log of follow-up visits

bpm = beats per minute; mEHRA symptoms scale = modified European Heart Rhythm Association symptoms scale; GP = general practitioner; INR = international normalized ratio; LV = left ventricular; NOAC = non-vitamin K antagonist oral anticoagulant; VKA = vitamin K antagonist.

Case 4

70 yr old gentleman
Short of breath for 3 months, palpitations, dizzy spells
Systolic murmur
BP 150/100
Pulse 84 regular
Clear chest

LBBB



QRS>120ms

QS or rS in V1, “M” shape QRS in V6

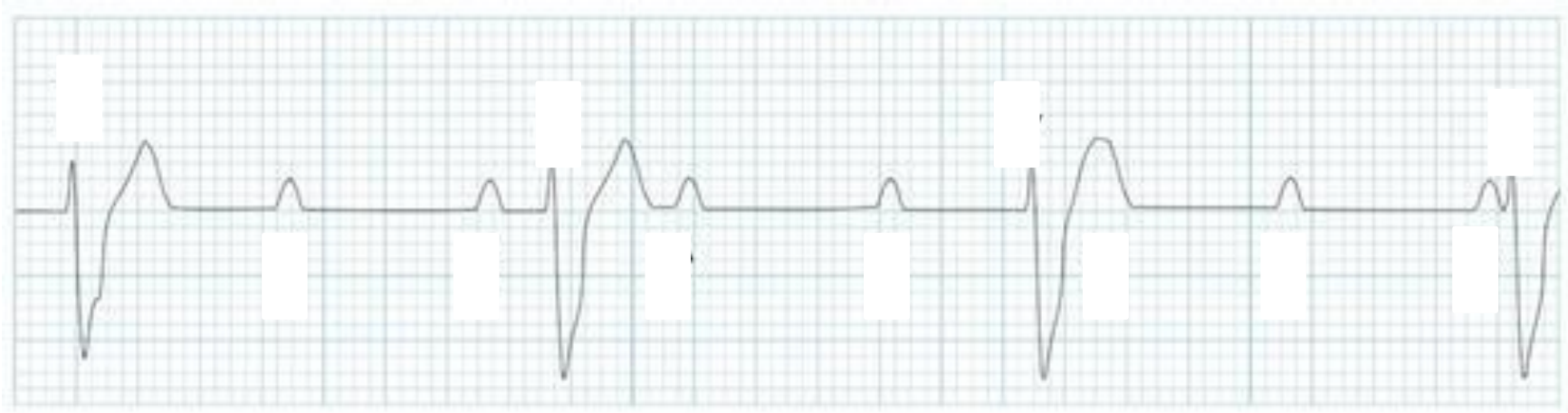
Always has left axis deviation (positive deflection in I, negative in aVF)

Always pathological

Common associations: hypertension, aortic stenosis, ischaemic heart disease, a tendency for further heart block

24 hr tape

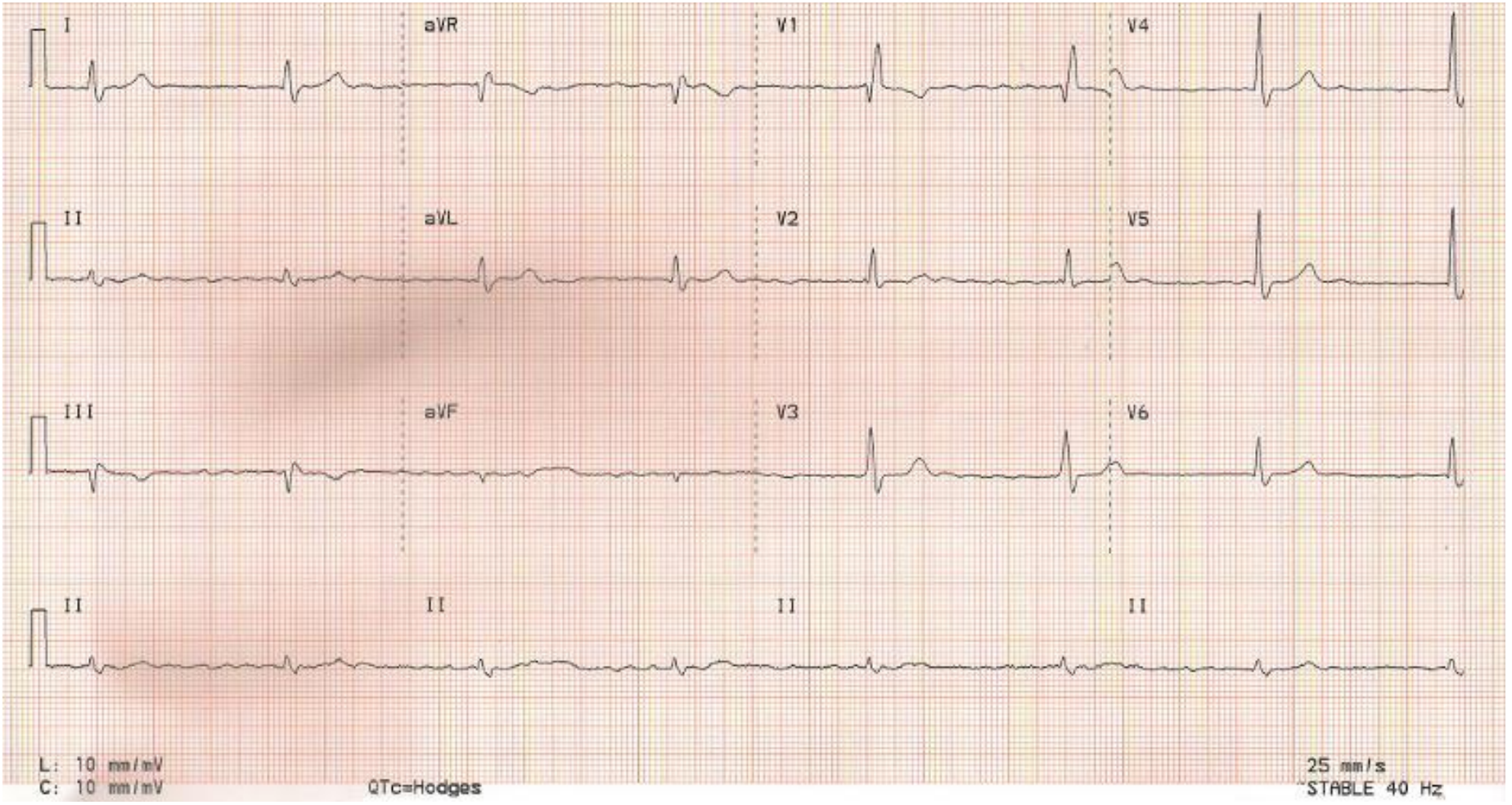
"Sinus Rhythm. Mean rate 60/min. No AF. Occasional VEs and SVE's. Infrequent bigeminy. Episodes of bradycardia with HR 36/min, some AV dissociation."



Complete heart block
Will need urgent referral for PPM

Case 5

90 yr old lady
Short of breath for 3 weeks,
some dizzy episodes
BP 150/100
Clear chest



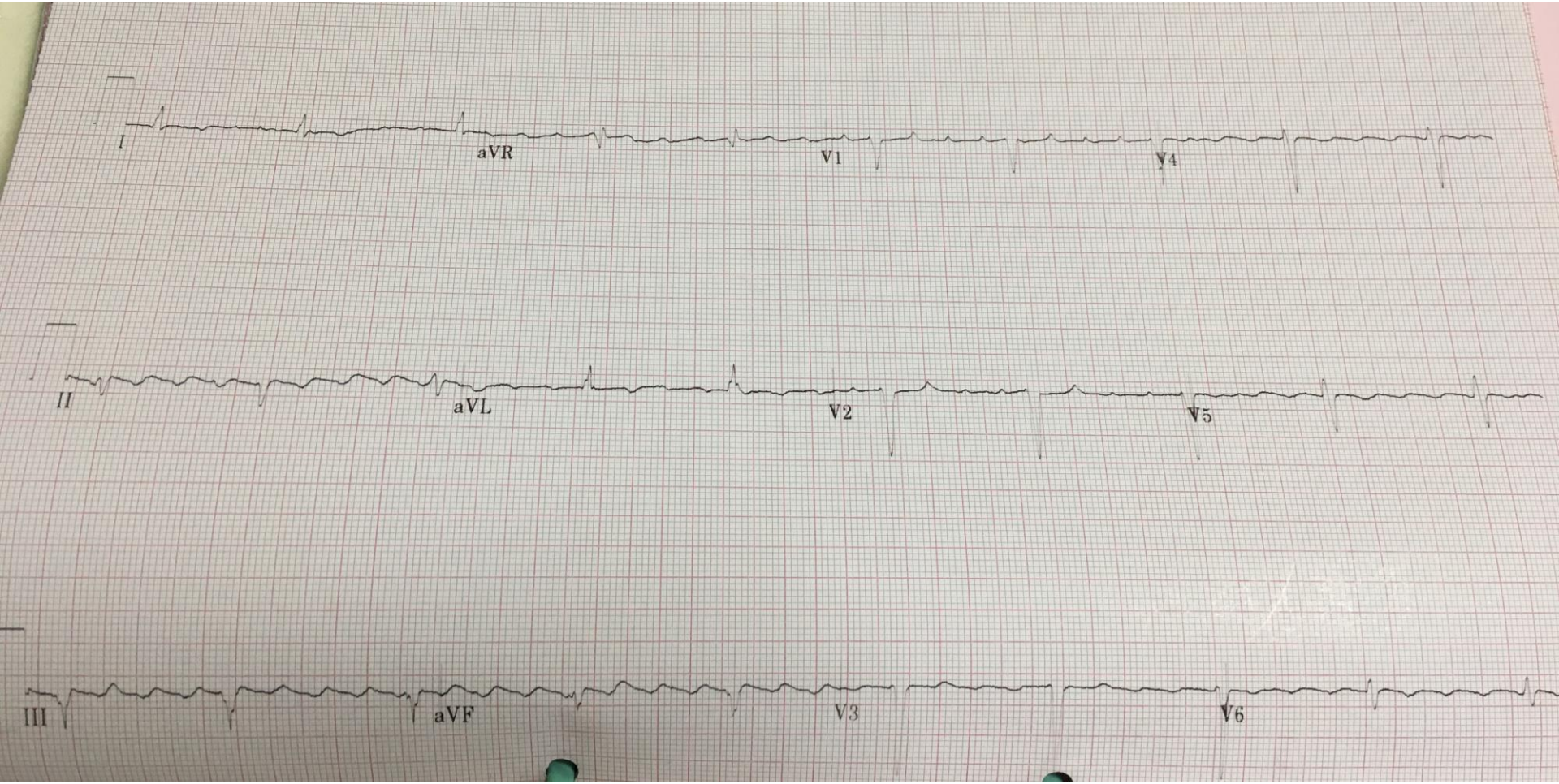
Bradycardic (48/min)
Regular
No P waves
Irregular baseline: AF with CHB

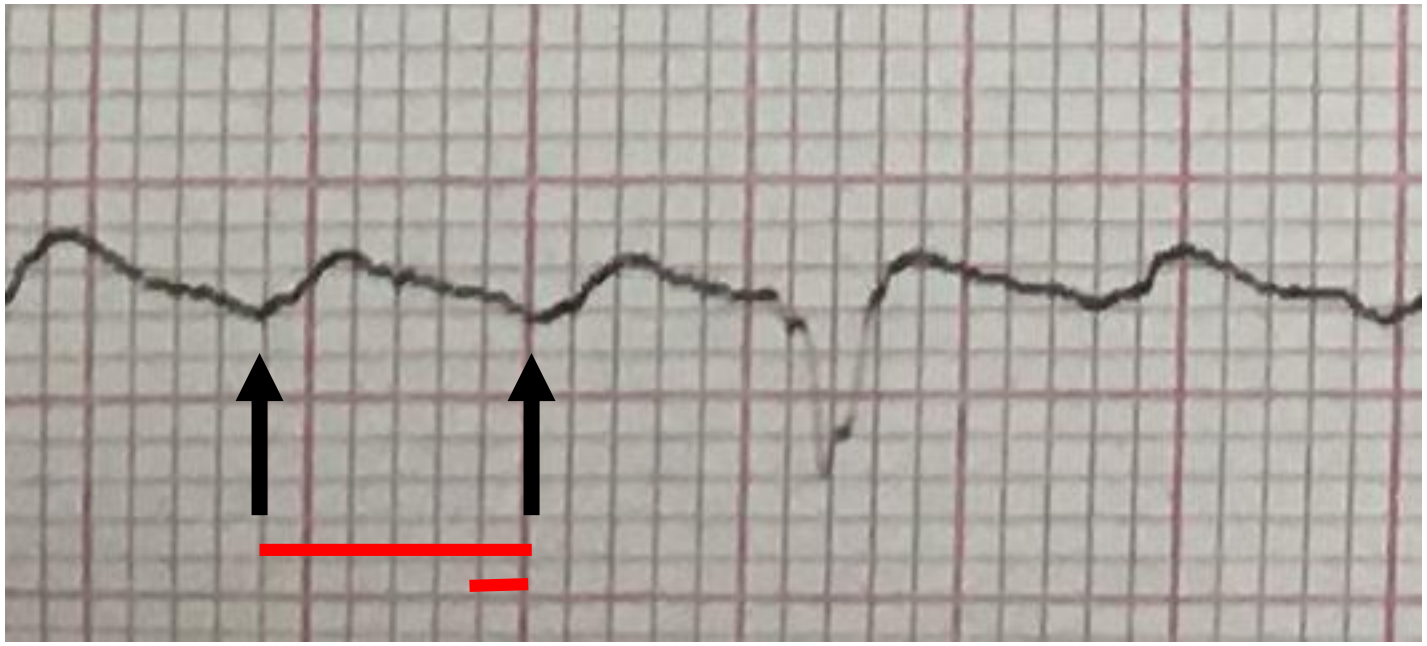
- Arrange admission to cardiology
- Needs Permanent pacemaker
- No mandate to anticoagulate in primary care- may complicate temporary wire/PPM insertion

Case 6

85 yr old gentleman
Short of breath for 6 weeks,
some dizzy episodes
BP 140/80
Pulse 44 regular
Bibasal crackles, pitting oedema
Frusemide given, HFS echo
requested

Atrial Flutter with 5:1 conduction
Anticoagulate; diurese, refer for echo and urgent assessment; consider admission to secondary care given low ventricular rate



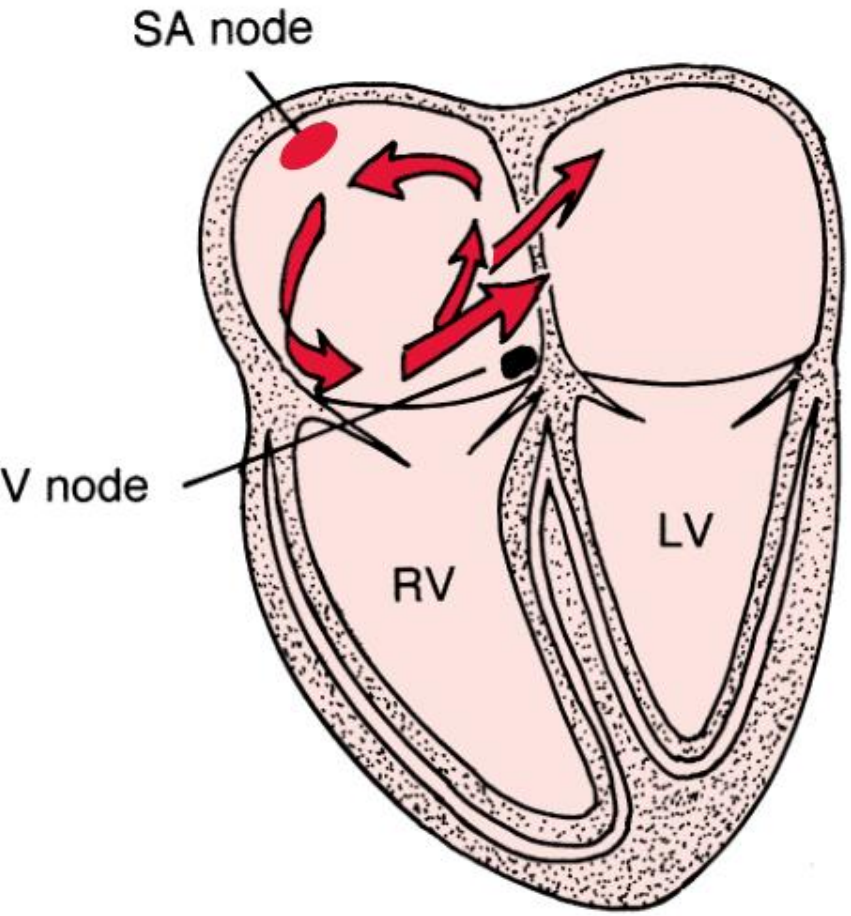


1 small square = 40ms

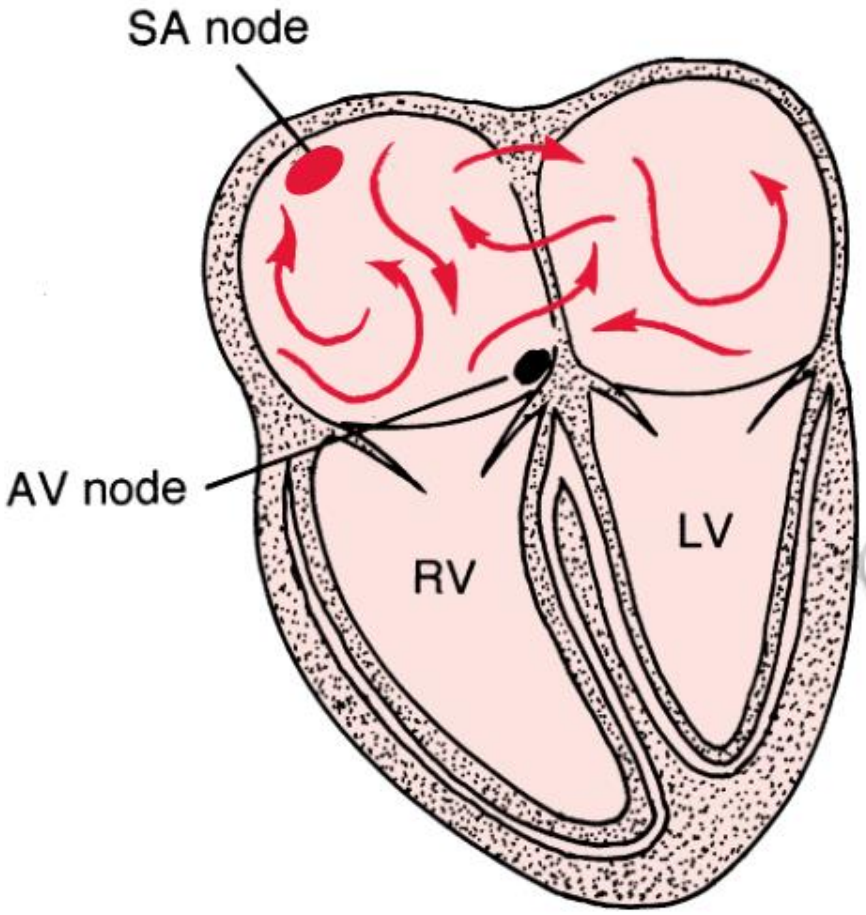
6 small square = 240ms

Flutter rate = $\frac{60{,}000}{240}$ = 250/min

ATRIAL FLUTTER



ATRIAL FIBRILLATION

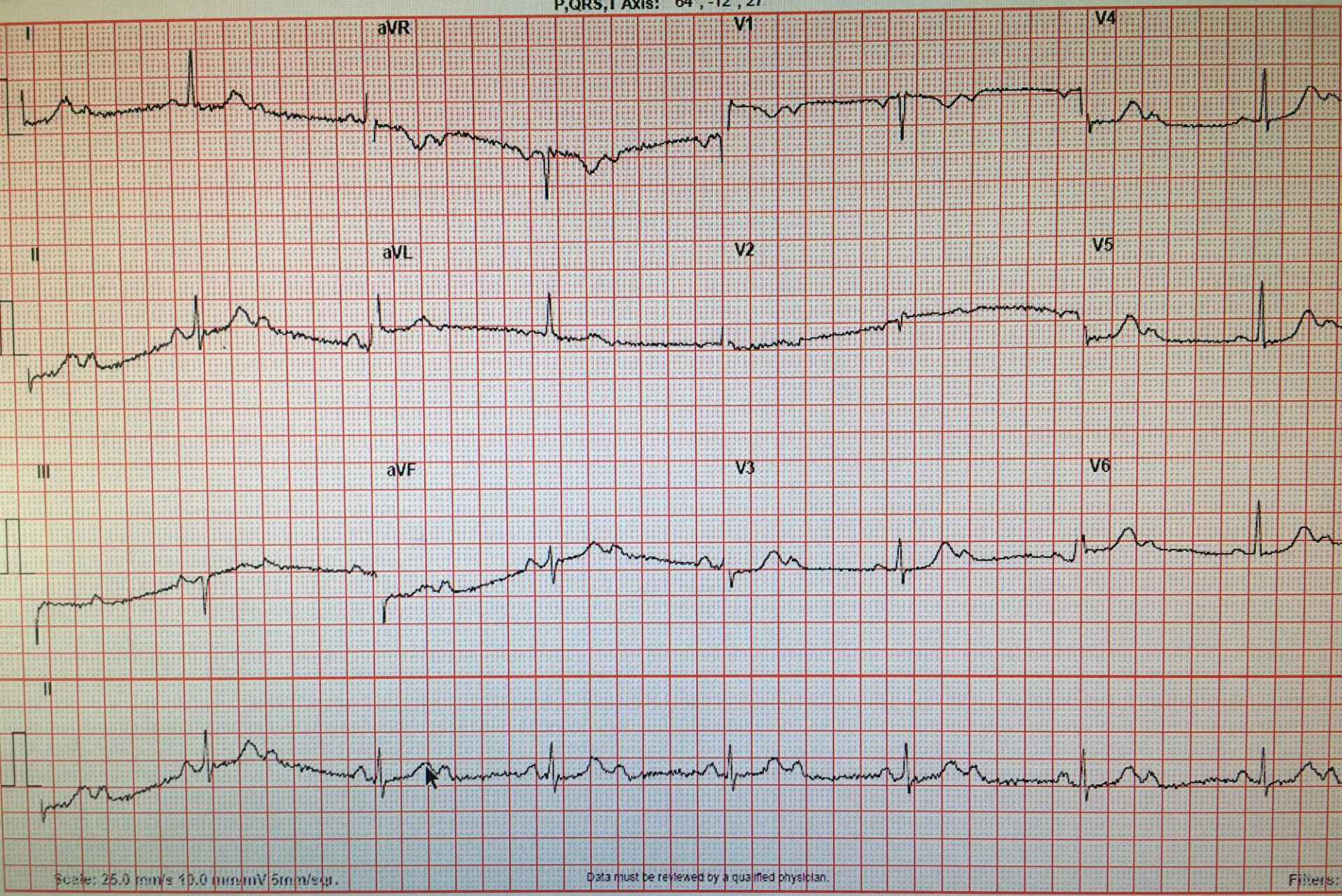


Case 8

70 yr old lady
Episodes of “head rush”
BP 150/100
Pulse 48 regular
Clear chest, soft murmur
On antidepressants only

ht: 0 ft 0 in
ht: 0 lbs

QTc Interval: 405 ms
P,QRS,T Axis: 64°, -12°, 27°

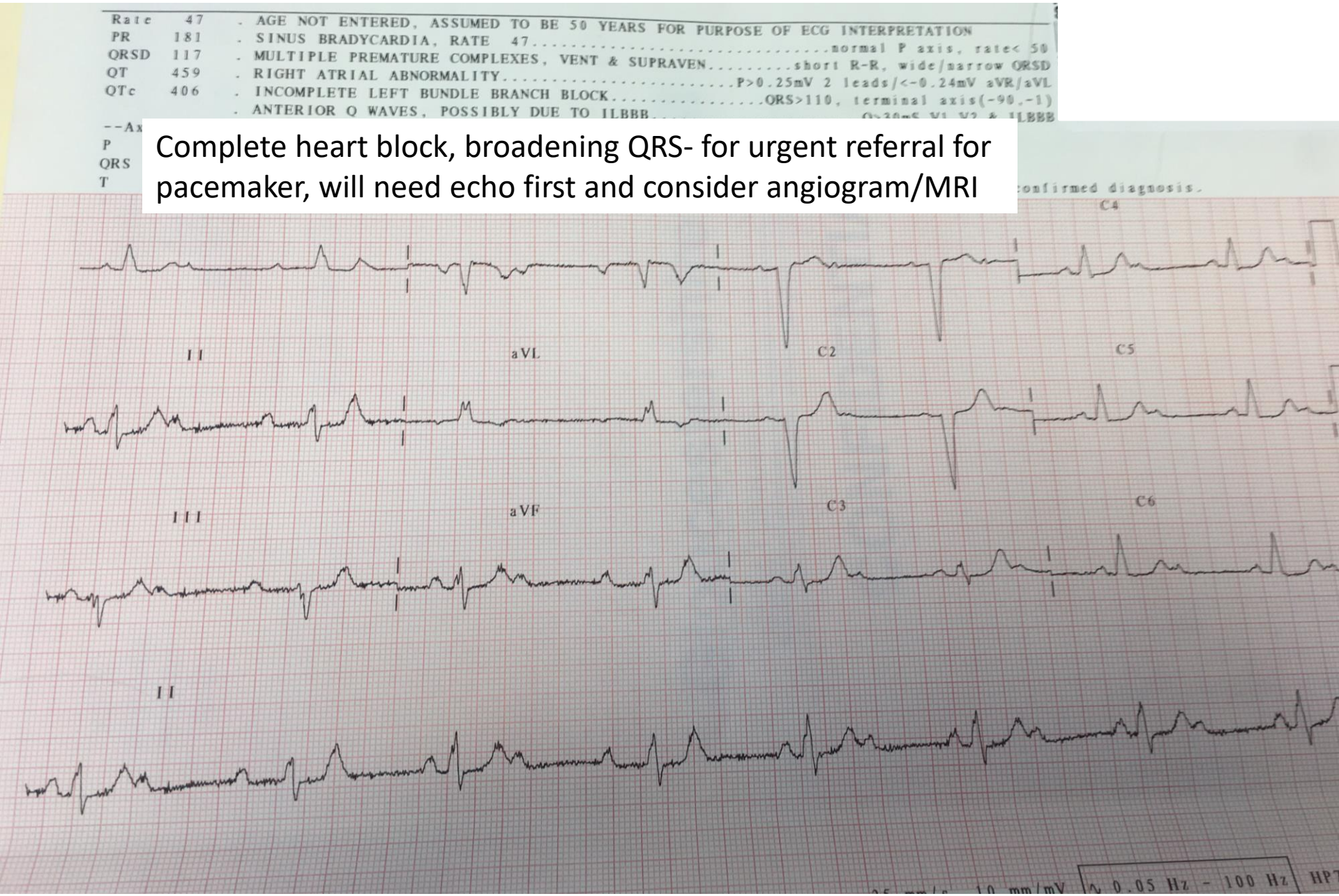


Scale: 25.0 mm/s 10.0 mm/mV 5m m/s/µ.

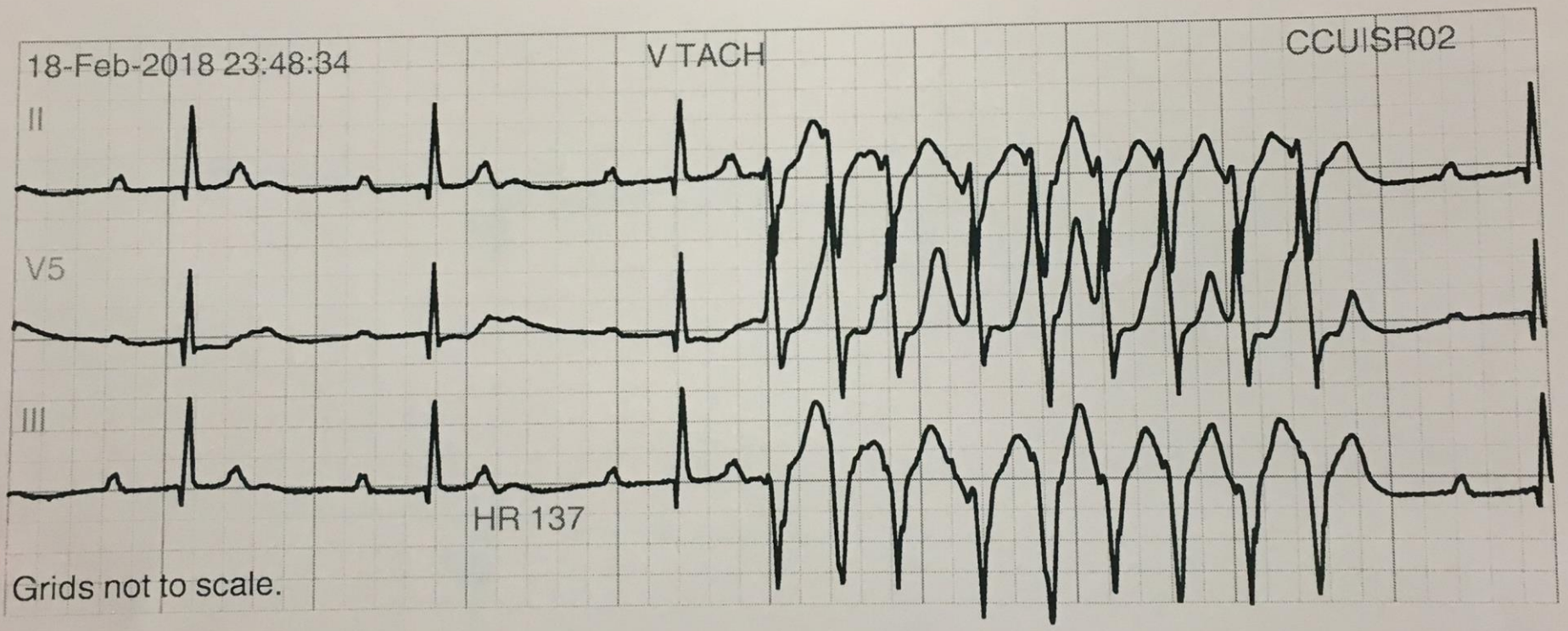
Data must be reviewed by a qualified physician.

Filters:

CardioView



Complete heart block, broadening QRS- for urgent referral for pacemaker, will need echo first and consider angiogram/MRI


18-Feb-2018 23:48:34
V TACH
CCUISR02
II
V5
III
HR 137
Grids not to scale.

Case 9

47 yr old man
Smoker, hypertensive
Atypical chest pain (left shoulder ache at rest)
Pulse 60/min irregular
BP 140/80
Clear chest, no murmur
24 hr tape arranged by GP
Cardiology referral, not RACPC as atypical pains

Dropped Beats	: 0	QRS Total	: 126292
Pauses	: 0	Paced Beats	: 0

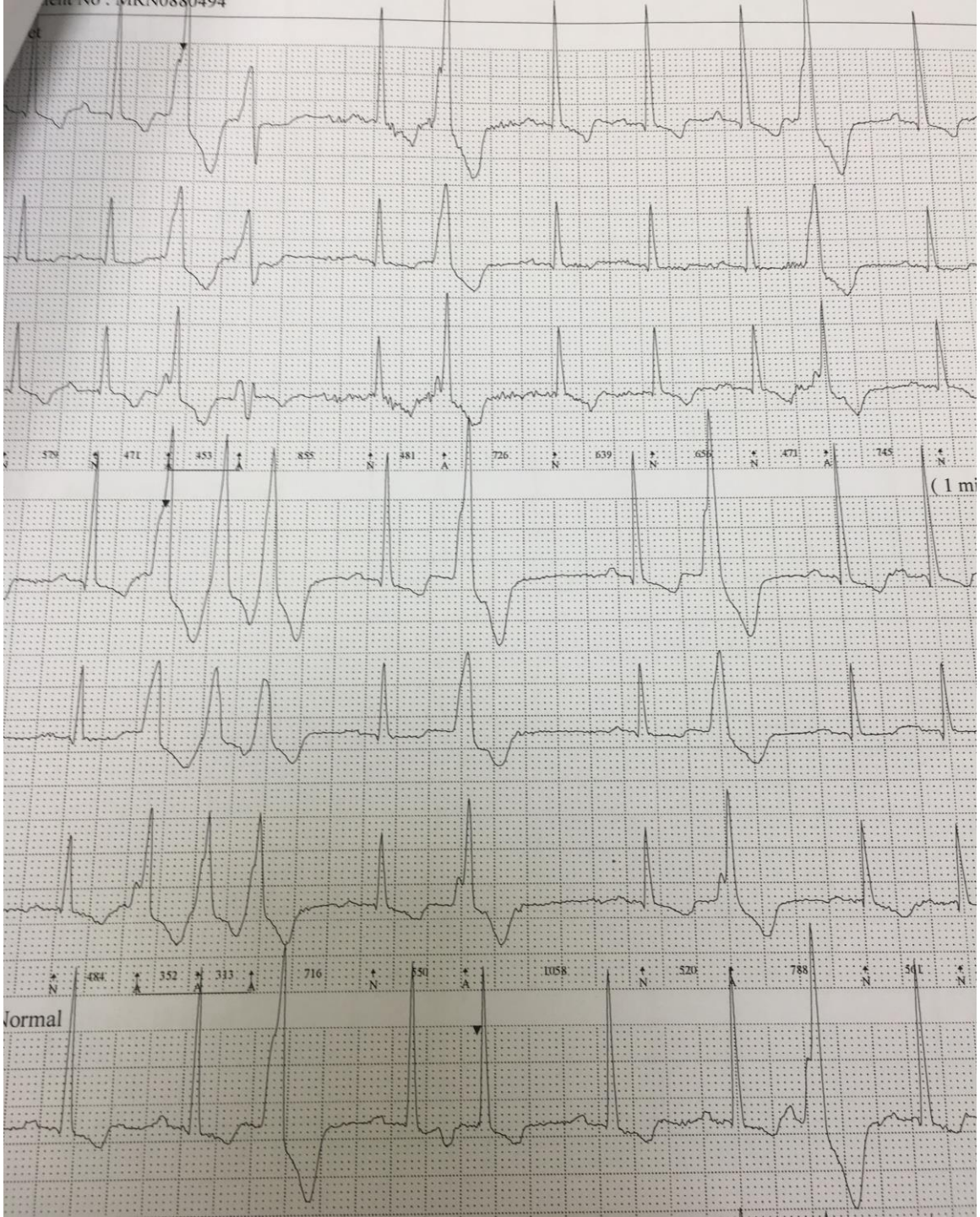
Comments :

THIS IS TECHNICIANS REPORT ONLY
THE REFERRING PHYSICIAN MUST CONFIRM THE ACCURACY OF THESE COMMENTS BEFORE MAKING CLINICAL DECISIONS.

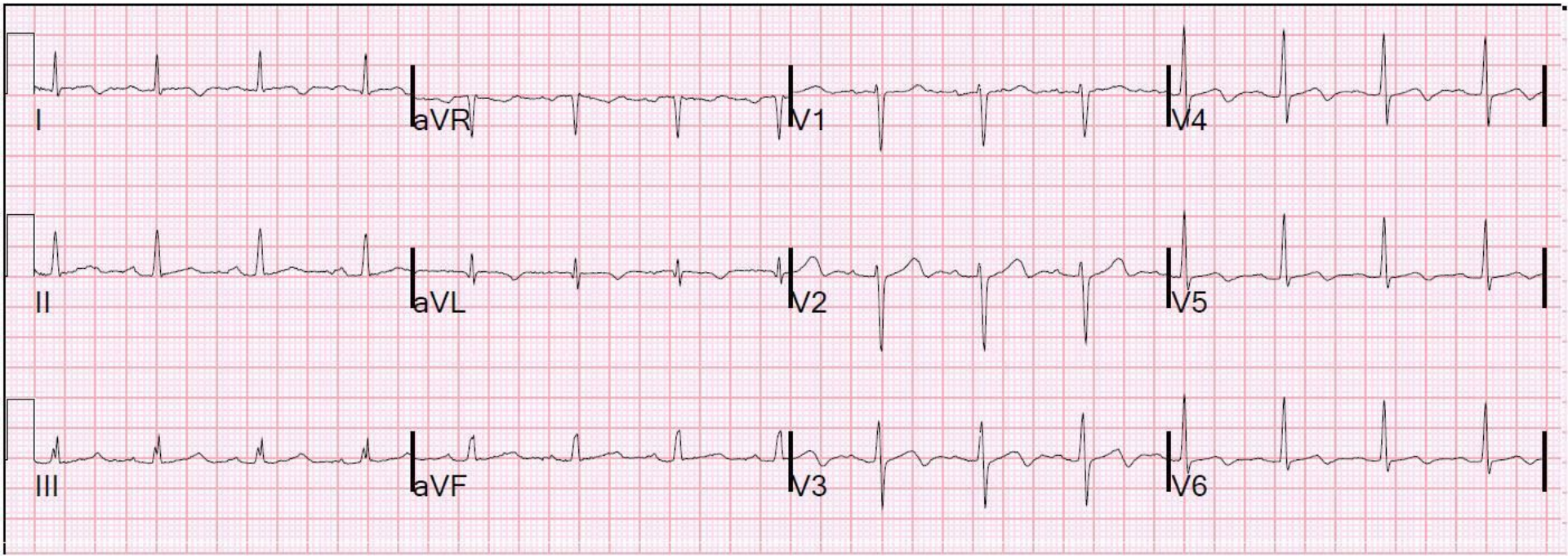
SINUS RHYTHM WITH BORDERLINE 1ST DEGREE HEART BLOCK AND BIPHASIC/INVERTED T WAVES.
RATES OF 67BPM - 115BPM.

INFREQUENT VE'S SEEN IN ISOLATION WITH OCCASIONAL COUPLETS, 1 X TRIPLET AND 1 X SALVO, OCCASIONAL EPISODES OF V BIGEMINY/TRIGEMINY.
INFREQUENT PAC'S SEEN IN ISOLATION.

NO PATIENT ACTIVATED/DIARY EVENTS NOTED.


N 570
A 471
A 453
N 855
A 481
N 726
N 639
N 658
A 473
A 745
N
(1 mi
N 484
A 352
A 313
N 716
A 550
N 1058
A 520
N 788
N 501
Normal

Biphasic T waves across chest leads: with chest pain and risk factors, this is an LAD syndrome (Wellen's syndrome).
Typically results from critical LAD disease.
Ventricular triplets result from ischaemia.
High VE burden (>10% of total) may be a clue to underlying ischaemia.
Consider urgent admission to cardiology.



Zio XT

Zio XT Final Report for

iRhythm Technologies
www.zioreports.com

iRhythm

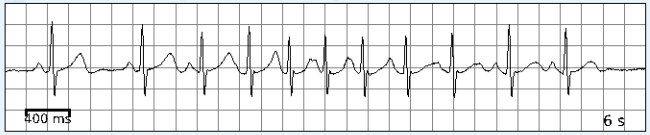
Enrollment Period
13 days 15 hours
09/02/18, 13:48 to
23/02/18, 05:07

Analysis Time
10 days 2 hours
(after artifact removed)

Supraventricular Tachycardia (4 beats or more)

Episodes
3
HR Range
102-179 bpm
Avg
126 bpm

▼ Fastest SVT (HR Range 112-179 bpm, Avg 145 bpm)



Ventricular Tachycardia (4 beats or more)

None found

Pauses (3 secs or longer)

None found

AV Block (2nd° Mobitz II, 3rd°)

None found

Atrial Fibrillation

None found

Heart Rate

Overall	**Max**	**179 bpm**	00:20, 11/02
	Min	**47 bpm**	05:24, 20/02
	Avg	**72 bpm**	
Sinus	**Max**	**127 bpm**	11:22, 12/02
	Min	**47 bpm**	05:24, 20/02
	Avg	**72 bpm**	

Patient Events

Triggered Events: 9
Findings within ± 45 sec of Triggers:
Sinus Rhythm

Diary Entries: 7
Findings within ± 45 sec of Entries:
Sinus Rhythm, Supraventricular Ectopic beat(s)

Ectopics

	Rare <1%	Occasional 1% to <5%	Frequent 5%+
Supraventricular Ectopy (SVE/PACs)			
Isolated	**Rare**	**<1.0%**	
Couplet	**Rare**	**<1.0%**	
Triplet	**Rare**	**<1.0%**	
Ventricular Ectopy (VE/PVCs)			
Isolated	**Rare**	**<1.0%**	
Couplet	**0**		
Triplet	**0**		

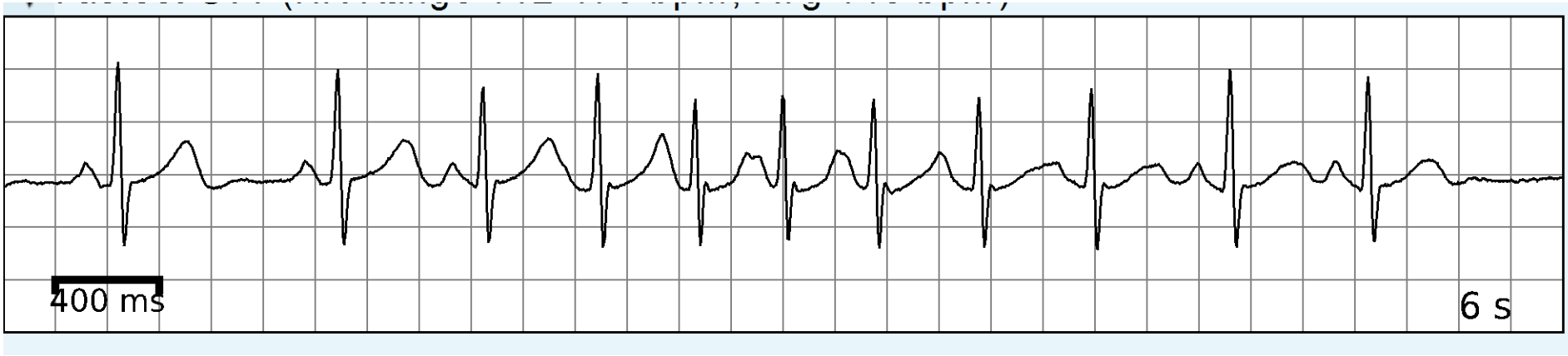
Longest Ventricular Bigeminy Episode **0 s**
Longest Ventricular Trigeminy Episode **0 s**

Preliminary Findings

Patient had a min HR of 47 bpm, max HR of 179 bpm, and avg HR of 72 bpm. Predominant underlying rhythm was Sinus Rhythm. 3 Supraventricular Tachycardia runs occurred, the run with the fastest interval lasting 9 beats with a max rate of 179 bpm (avg 145 bpm); the run with the fastest interval was also the longest. Isolated SVEs were rare (<1.0%), SVE Couplets were rare (<1.0%), and SVE Triplets were rare (<1.0%). Isolated VEs were rare (<1.0%), and no VE Couplets or VE

Final Interpretation

Atrial ectopics

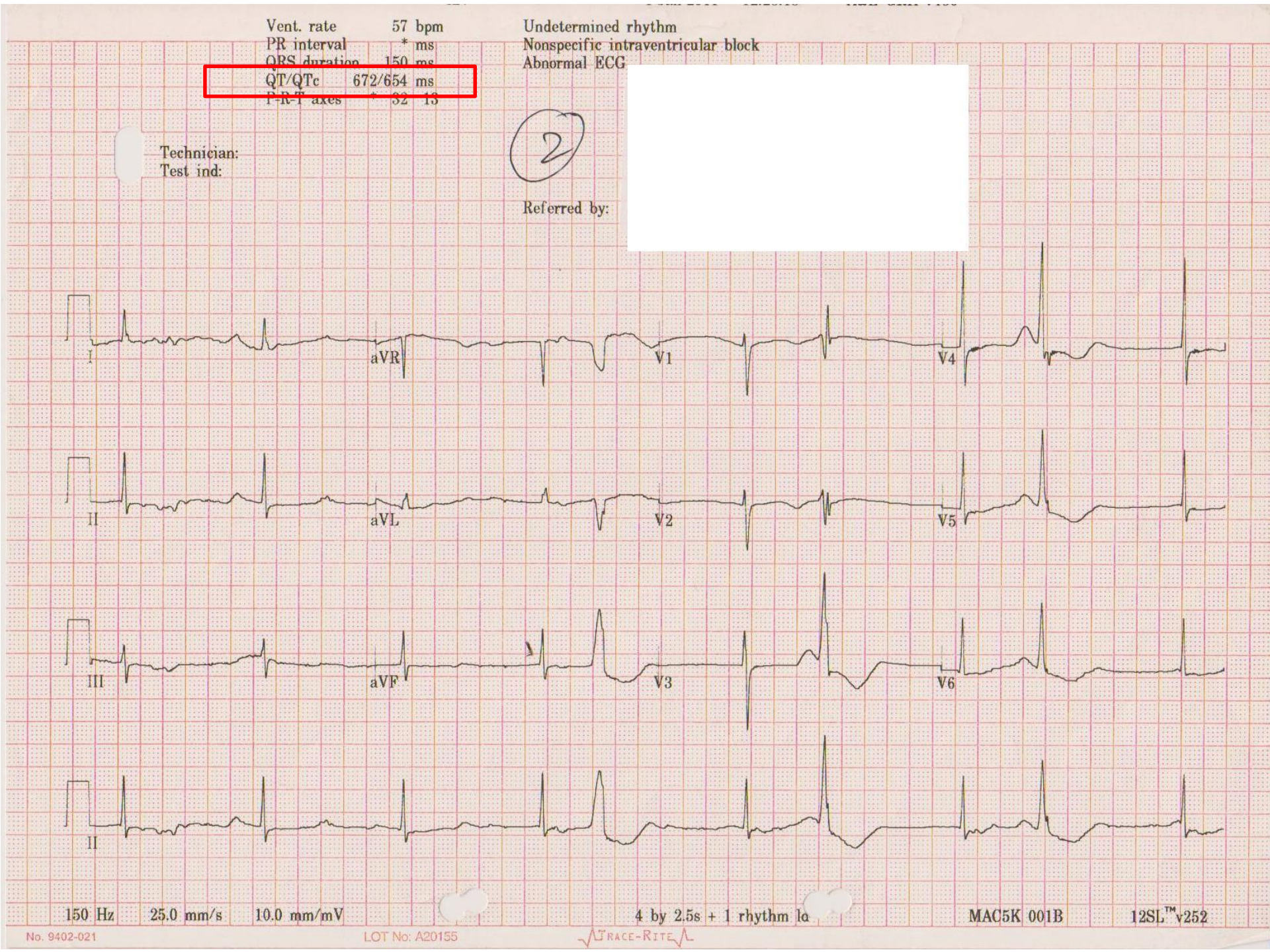


- Arise from ectopic tissue in atria
- Abnormal p wave followed by normal QRS; p wave may be hidden in preceding T wave giving “peaked” appearance
- Atrial ectopics can make SA node depolarise leading to a pause before the next sinus beat arrives
- Usually do not require treatment, but pt should have 12 lead ECG to rule out WPW, and echo to rule out atrial enlargement
- Beta blockers effective and safe

Any cell in the heart is capable of becoming the “pacemaker” if it depolarises first- including ventricular cells (leading to ventricular ectopics)

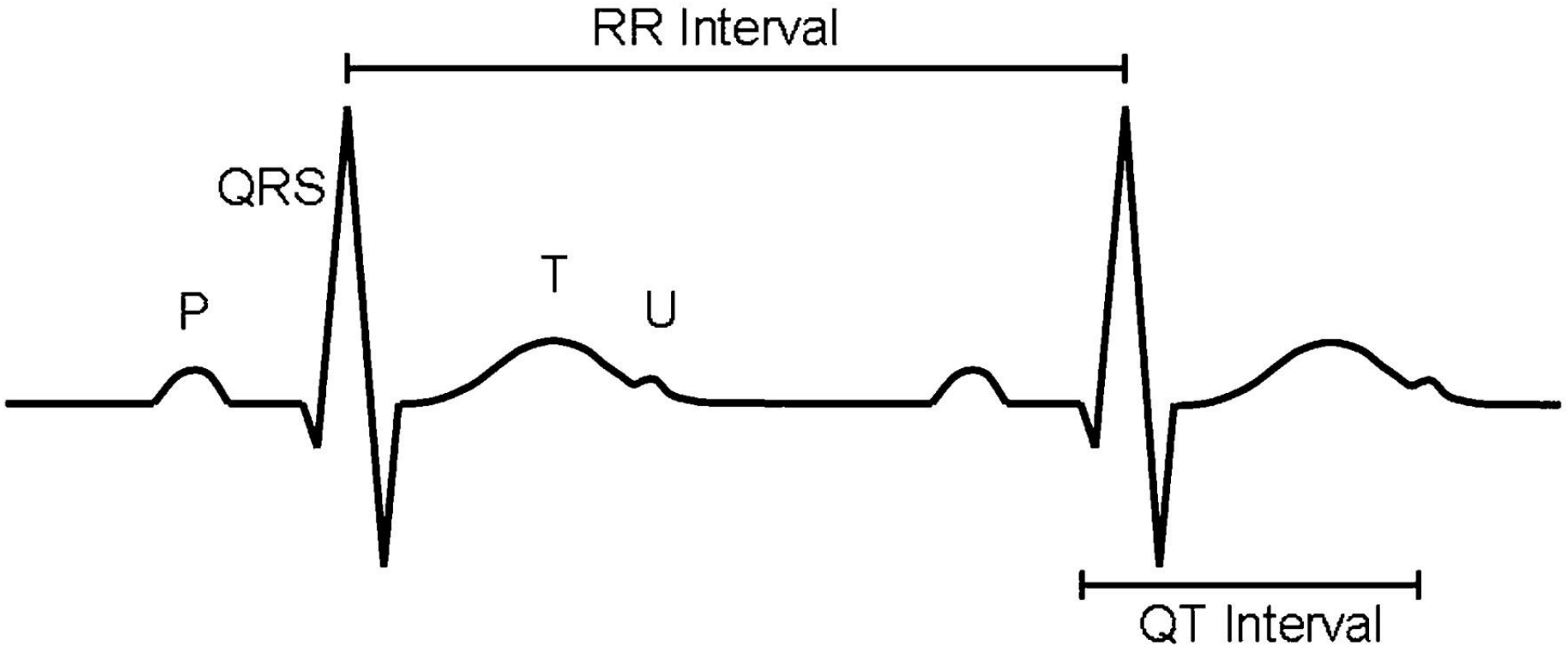
Case 10

75 yr old lady
Episodes of syncope and palpitations
Nonspecifically unwell over last few weeks
BP 140/90
Pulse 42/min irregular
Clear chest, no murmur
On no medications


Vent. rate 57 bpm
PR interval * ms
QRS duration 150 ms
QT/QTc 672/654 ms
P-R-T axes * 32 13
Undetermined rhythm
Nonspecific intraventricular block
Abnormal ECG
2
Technician:
Test ind:
Referred by:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
150 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 1 rhythm la
MAC5K 001B
12SL™v252
No. 9402-021
LOT No: A20155
TRACE-RITE

Bazett's Formula

$$QTc = \frac{QT}{\sqrt{RR}}$$

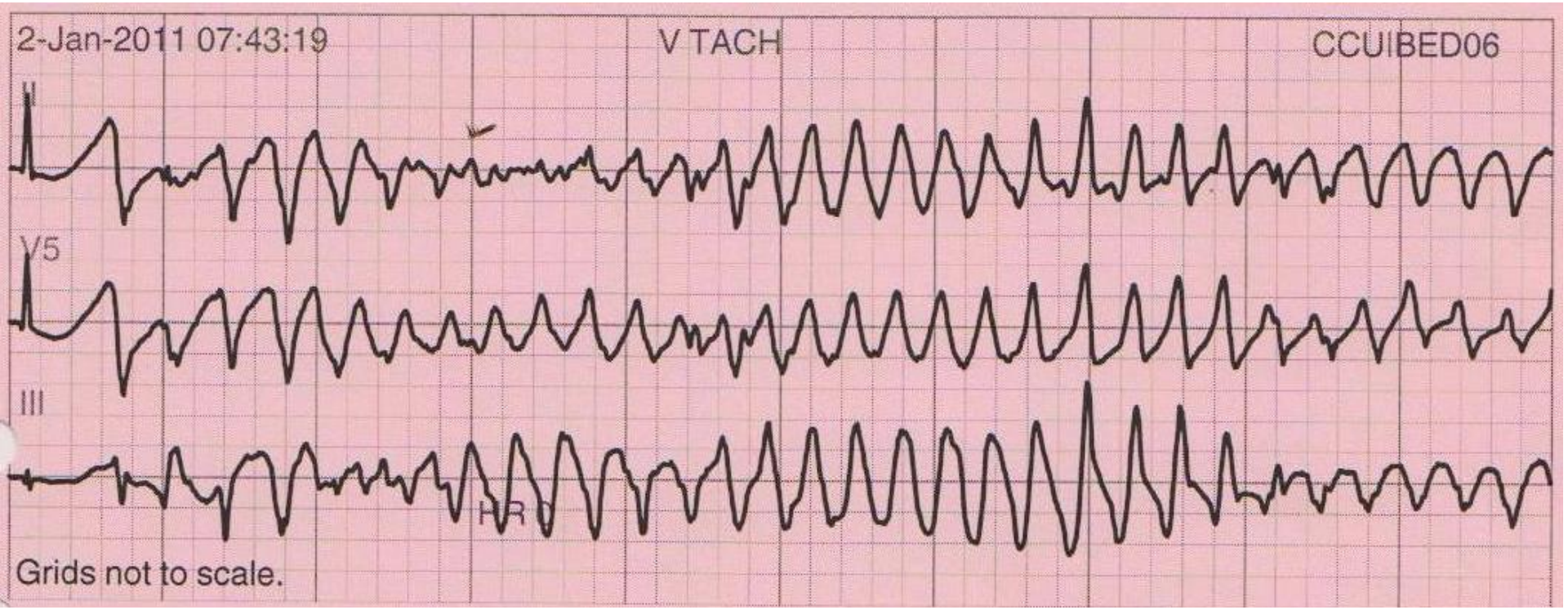


Severe primary hypothyroidism presenting with torsades de pointes

[Sri Raveen Kandan](),
[Mrinal Saha]()

BMJ Case reports 2012

Long QTC promotes Torsades. Commonest causes are drugs (longqt.org)- macrolides, amiodarone, sotalol, flecainide, citalopram. Check K, Mg, Ca, TSH.



Summary (1)

- Cardiac monitors are useful for syncope as well as palpitations
- The type/duration of monitor should be determined by the frequency of symptoms
- The rhythm recording report (12 lead ECG or Holter)doesn't always convey what the ECG traces show- it is worth scrutinising them
- Atrial ectopy is common and most don't need treating- unless symptomatic or high burden
- Runs of "SVT " are commonly just atrial tachycardias and not re-entry rhythms- and may be managed without tablets, or beta blockers if symptomatic
- Cardiac monitors can be useful for determining if AF rate control is adequate
- High VE burden (>10%) may reflect ischaemia or structural heart disease, consider referral- especially if associated with LV dysfunction
- Any symptomatic bradycardia ought to be considered for pacing

Summary (2)

- Review of case-based learning examples of patients with AF presenting in different ways
- Red flag cases highlighted
- Treatment options explored
- Palpitations are common, but associated co-morbidities are the clue to decide on referral
- ECG pattern recognition is crucial; if in doubt, please refer!

Thank you for listening

- NHS secretary: 0300 422 8286/ valmarks@nhs.net

- PP referrals: 07786069932/ drsahasecretary@gmail.com

drmrinalsaha.com