



Common ECGs in CRM

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CORE SYLLABUS

ANATOMY AND PHYSIOLOGY

Structure, including cardiac chambers, valves and great vessels (with particular reference to sites of vascular access), autonomic nervous system, blood supply, conduction system, cardiac cycle, action potential, normal physiology, cardiac failure.

CLINICAL ASSESSMENT

Basic knowledge will be expected of cardiovascular and respiratory symptoms and signs. ALS guidelines and basic X-ray interpretation.

ARRHYTHMIAS – DIAGNOSIS & ECG INTERPRETATION

ECG interpretation of common morphological abnormalities. Candidates should be aware of the features of common tachyarrhythmias such as atrial fibrillation, atrial flutter, atrial tachycardia, AVNRT, AVRT, ventricular tachycardia and ventricular fibrillation. Ventricular pre-excitation and the importance of accessory pathways should be understood. The common bradyarrhythmias including sinus node disorders and heart blocks should be recognised. Clinical evaluation of syncope and its causes and risk assessment of individuals at risk of sudden cardiac death. Evaluation of patients with atrial fibrillation, including stroke risk.

DEVICES

Although a greater depth of understanding will be expected in the specialist section, a general understanding will be expected of: indications for pacing and ICD implantation including CRT & implantable cardiac recorders, device circuitry, sensing technology, conductors and impedance, lead technology, defibrillator testing, pacemaker codes, lead and device extraction, electromagnetic interference, pacemaker syndrome, basics of timing cycle, parameter characteristics and device programming, radiation safety, device malfunction, patient follow-up, hysteresis, mode switching, rate response, ICD detection and therapy, principles underlying device implantation, end of life issues and device deactivation.

Confessions & Disclaimers

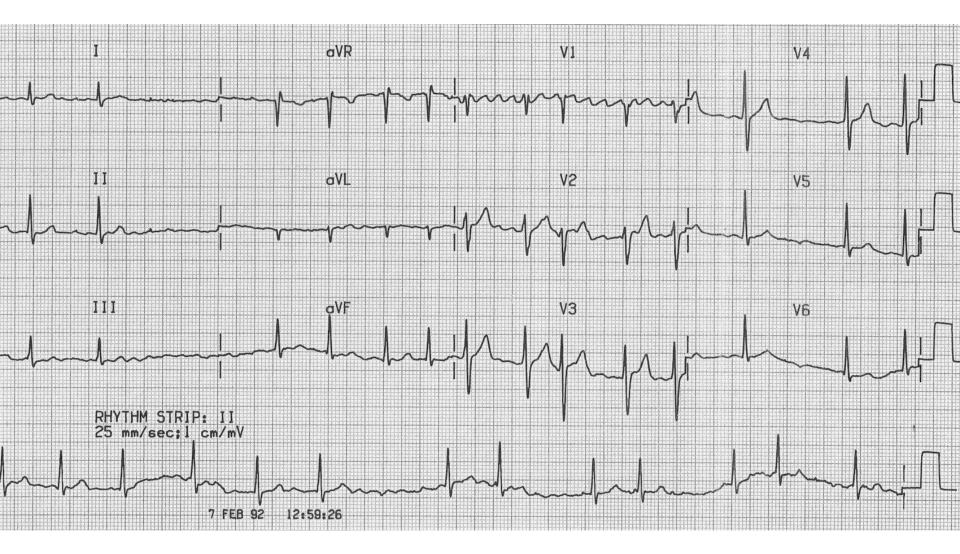
I am not an electrophysiologist!

ECGs credited to:





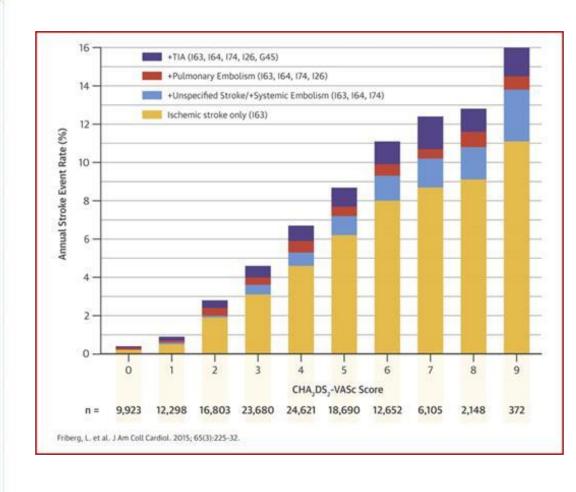
Tachycardia

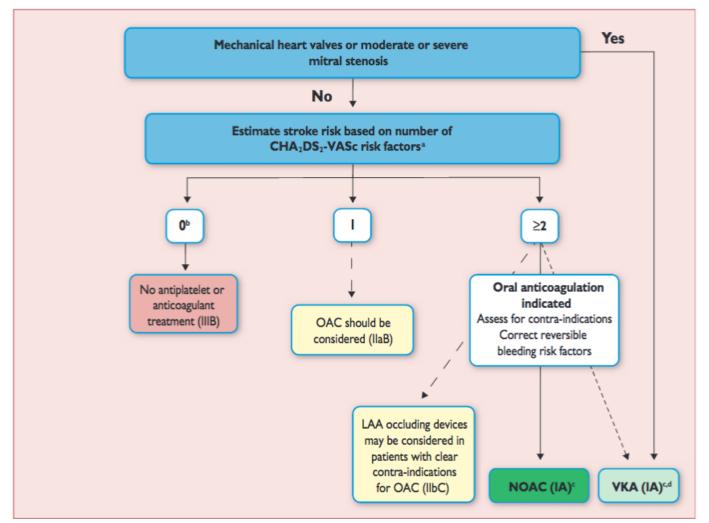


Atrial Fibrillation

Anticoagulation in AF

Risk factors	
Congestive Heart Failure	+1 point
Hypertension	+1 point
A2Age ≥75	+2 point
Diabetes	+1 point
S ₂ Stroke/TIA History	+2 point
Vascular Disease	+1 point
Age 65-74	+1 point
Sex (Female)	+1 point





AF = atrial fibrillation; LAA = left atrial appendage; NOAC = non-vitamin K antagonist oral anticoagulant; OAC = oral anticoagulation; VKA = vitamin K antagonist.

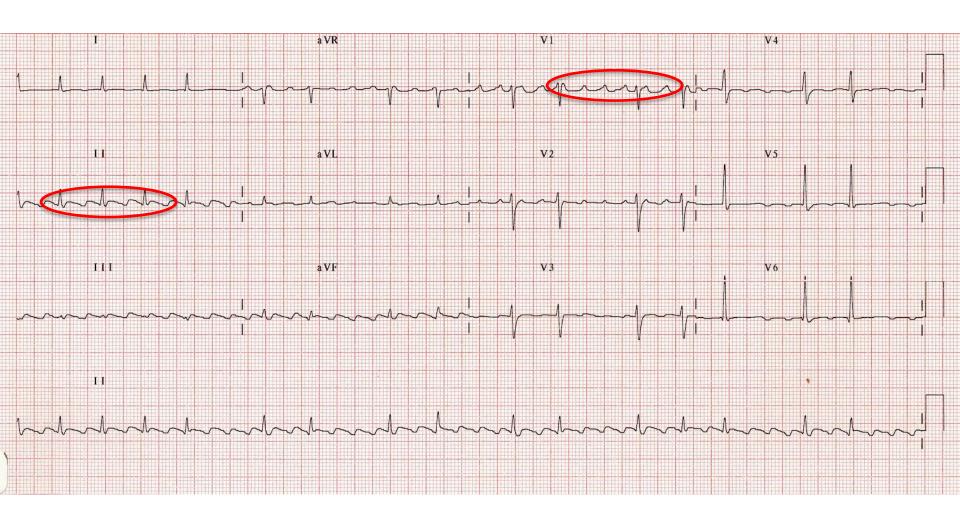
Figure 8 Stroke prevention in atrial fibrillation.

^aCongestive heart failure, Hypertension, Age ≥75 years (2 points), Diabetes, prior Sstroke/TIA/embolus (2 points), Vascular disease, age 65–74 years, female Sex.

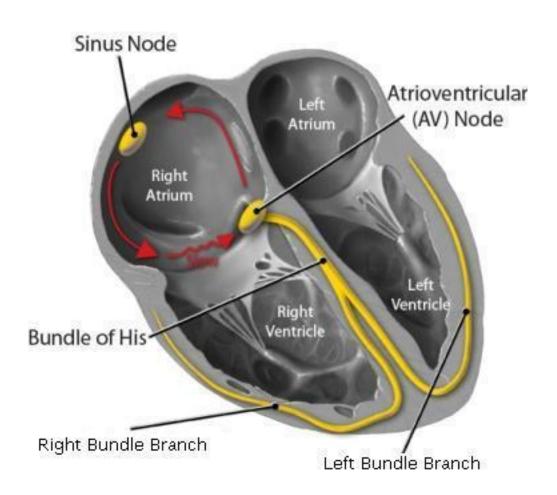
Includes women without other stroke risk factors.

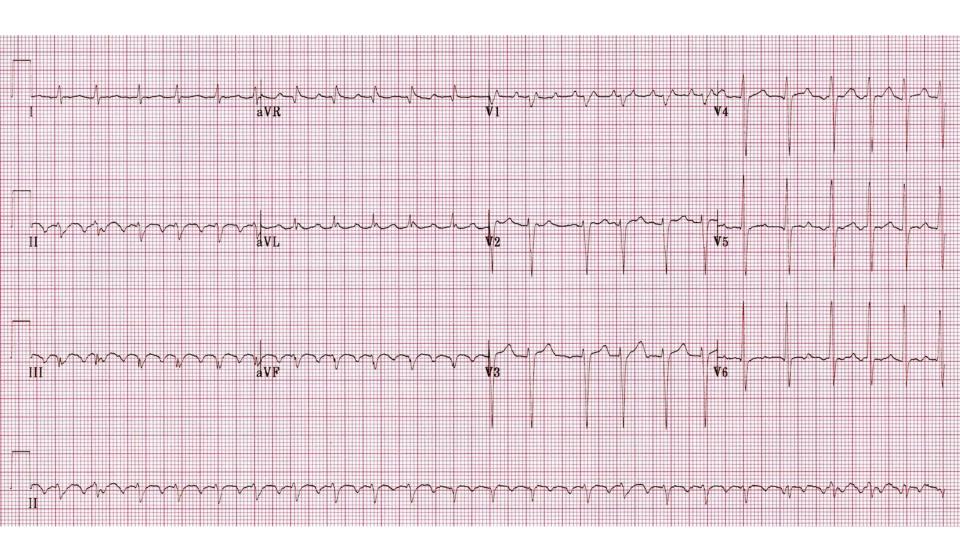
^{&#}x27;IlaB for women with only one additional stroke risk factor.

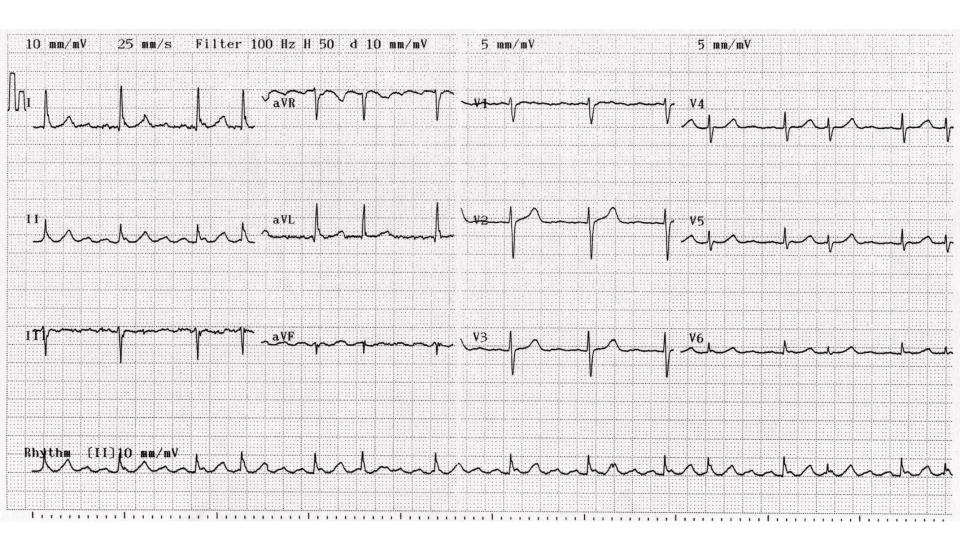
^dIB for patients with mechanical heart valves or mitral stenosis.



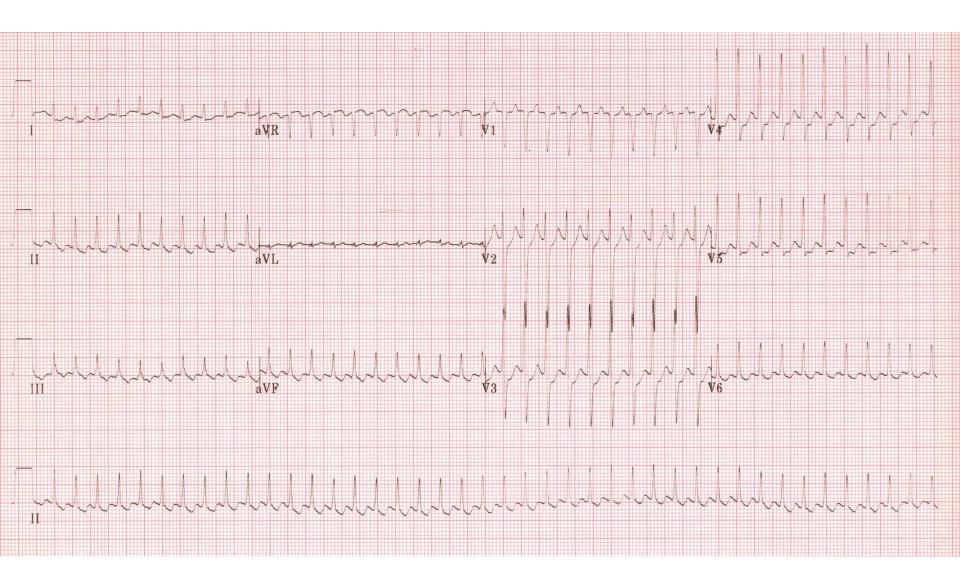
(Typical, counter-clockwise) atrial flutter



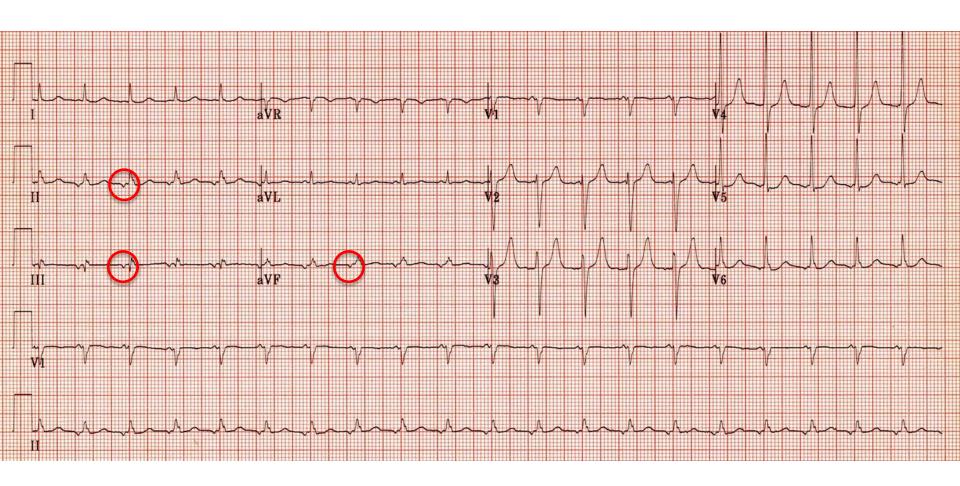




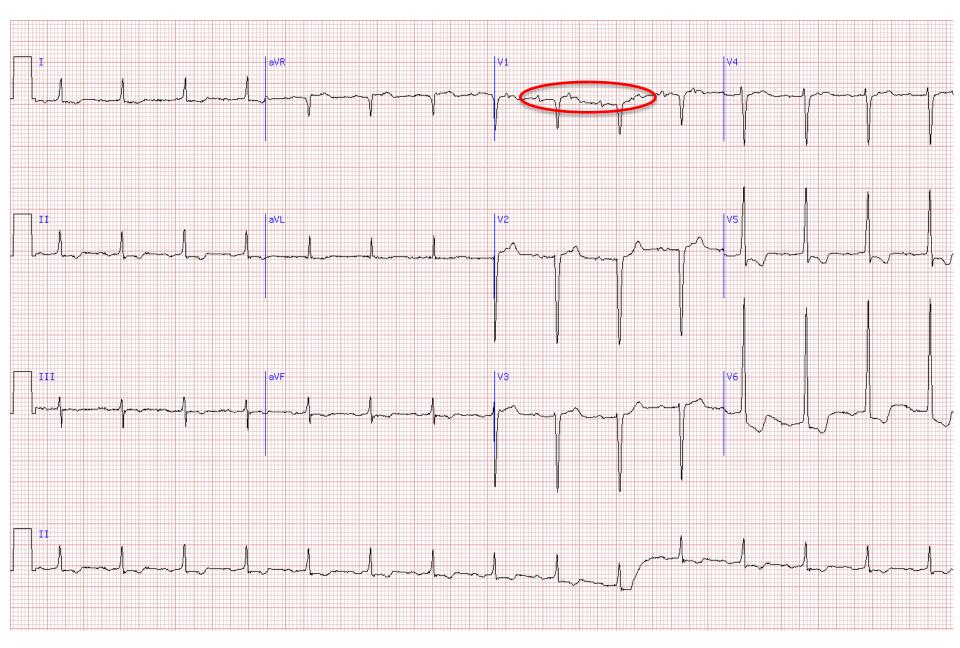
(Clockwise) atrial flutter



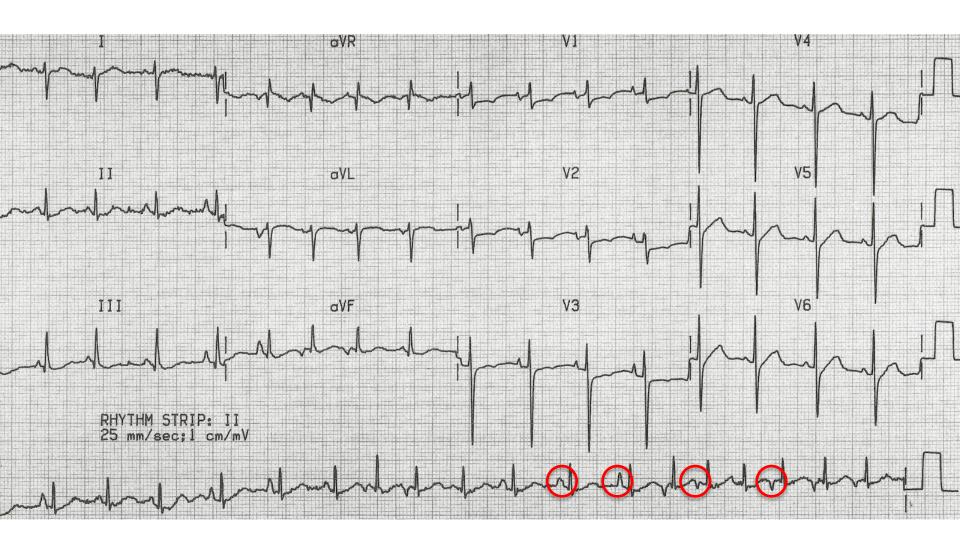
Atrial flutter with 1:1 conduction



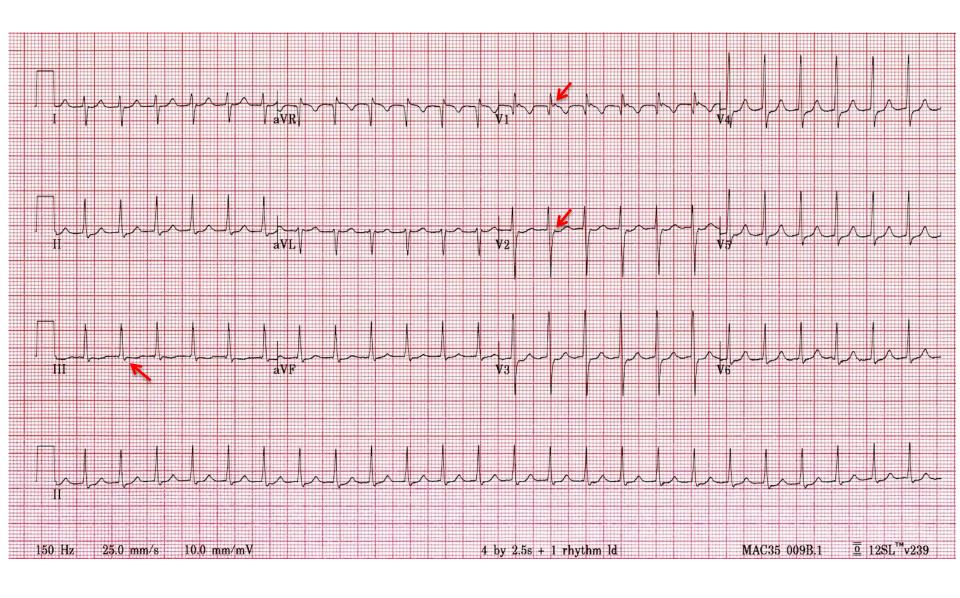
Ectopic atrial tachycardia



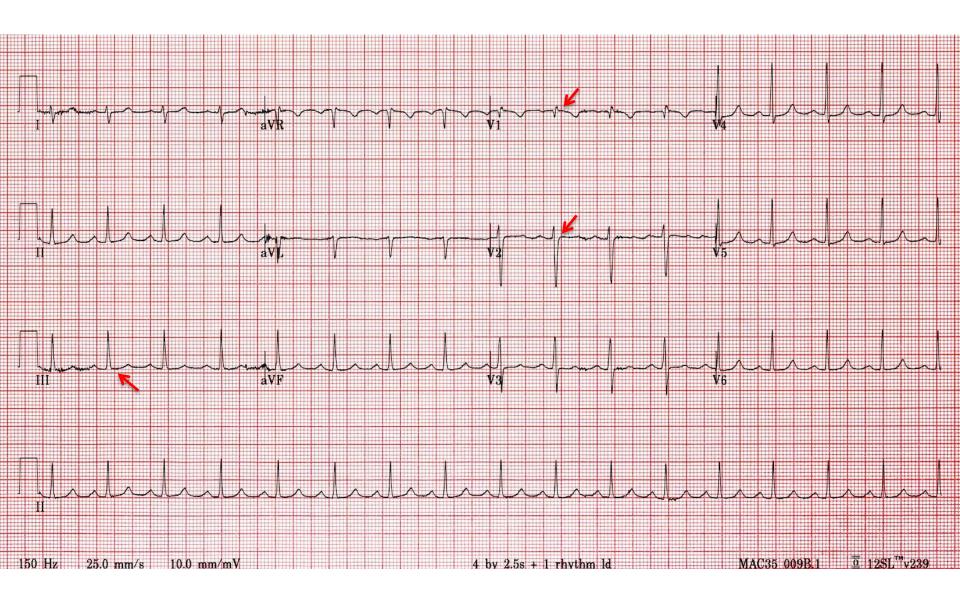
Atrial tachycardia with 2:1 conduction



Multifocal atrial tachycardia

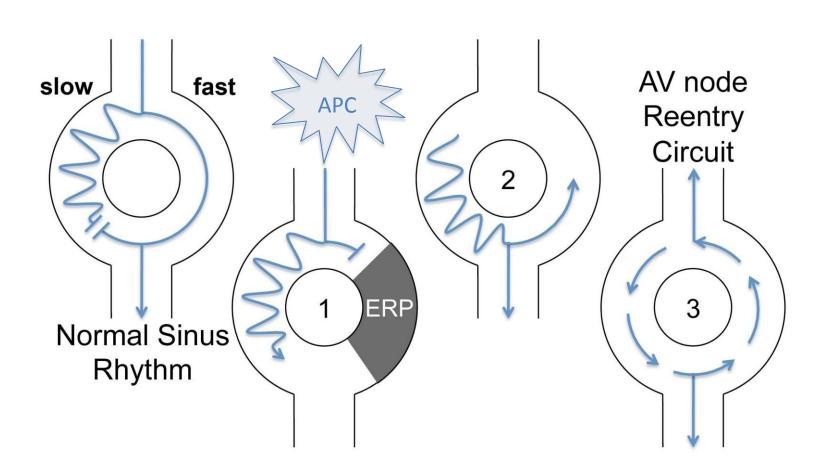


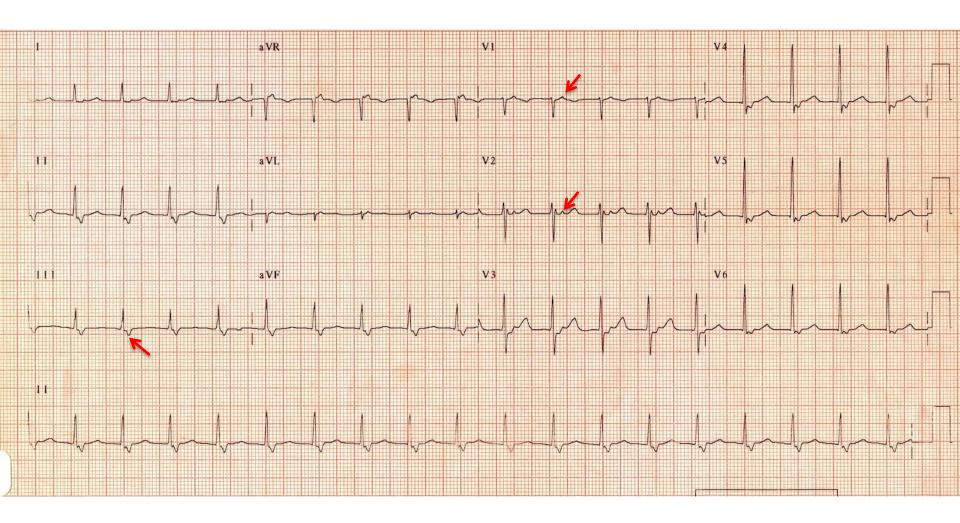
(Typical, slow-fast) AVNRT



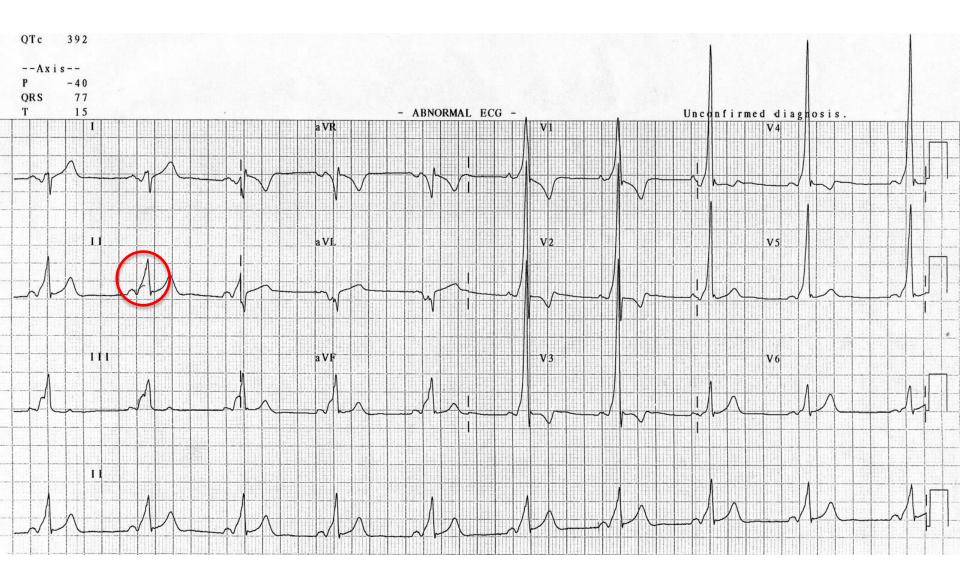
Same patient, after adenosine

AV nodal re-entry

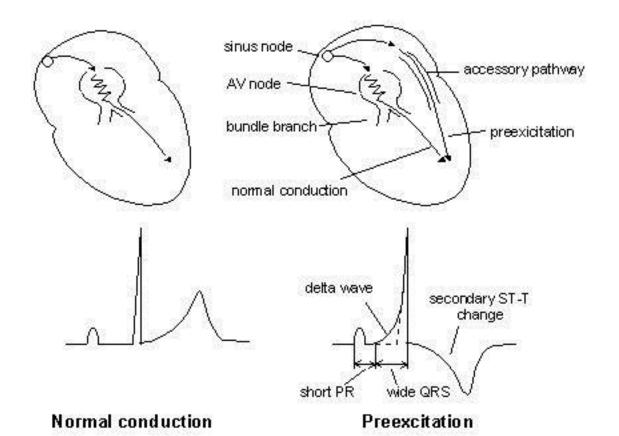




(Atypical, fast-slow) AVNRT



Pre-excitation



Accessory pathways

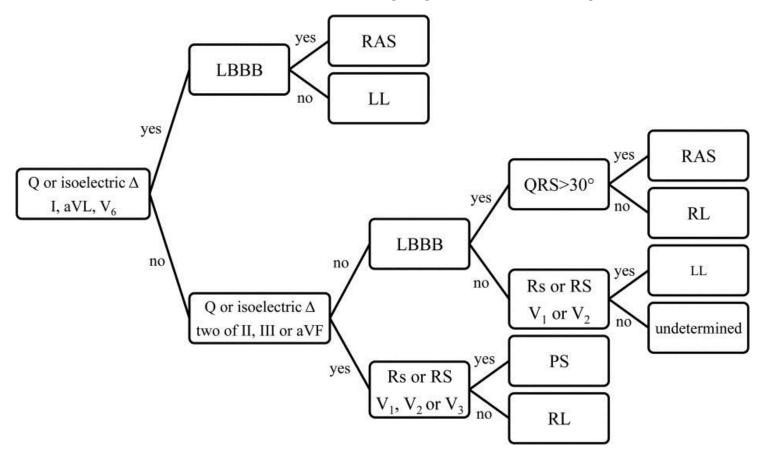
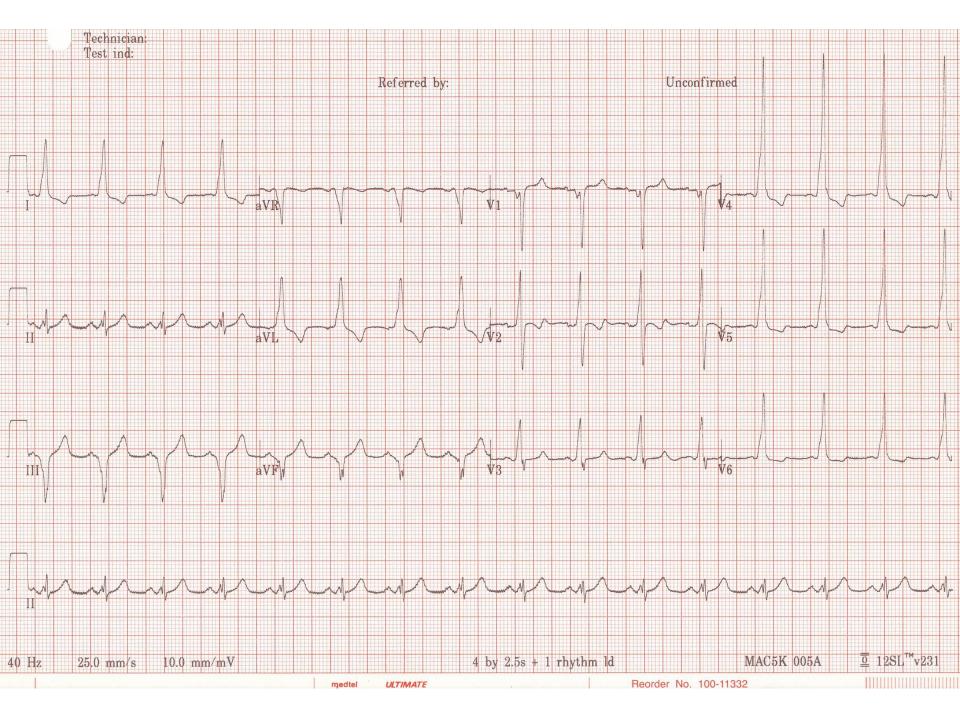


Figure 1. Algorithm summarizing steps to identify the location of an accessory pathway using the 12-lead electrocardiogram. See text for discussion.

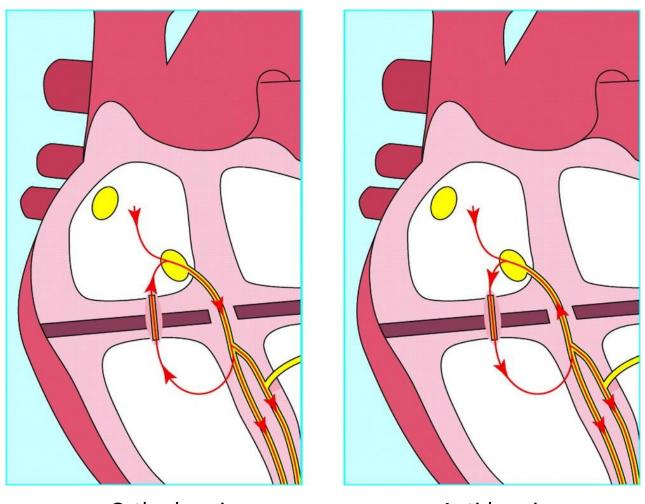
David J. Fox, George J. Klein, Allan C. Skanes, Lorne J. Gula, Raymond Yee, Andrew D. Krahn

How to identify the location of an accessory pathway by the 12-lead ECG

Heart Rhythm, Volume 5, Issue 12, 2008, 1763-1766

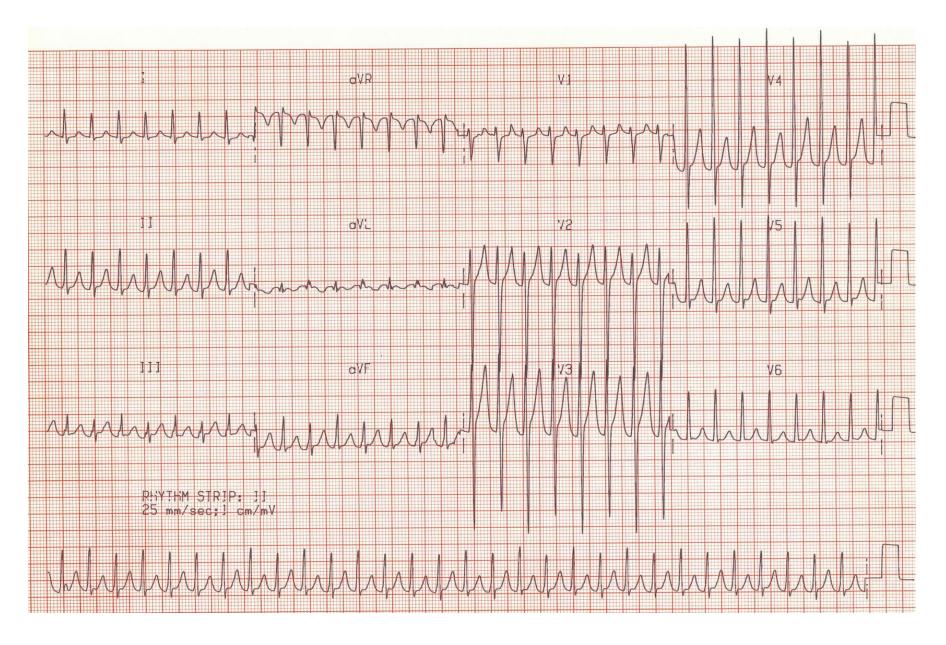


AVRT mechanisms

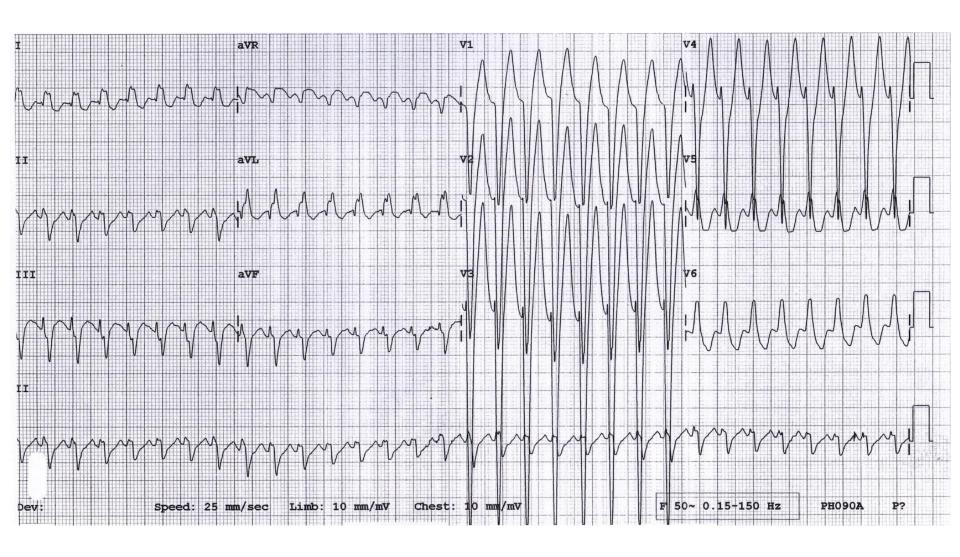


Orthodromic

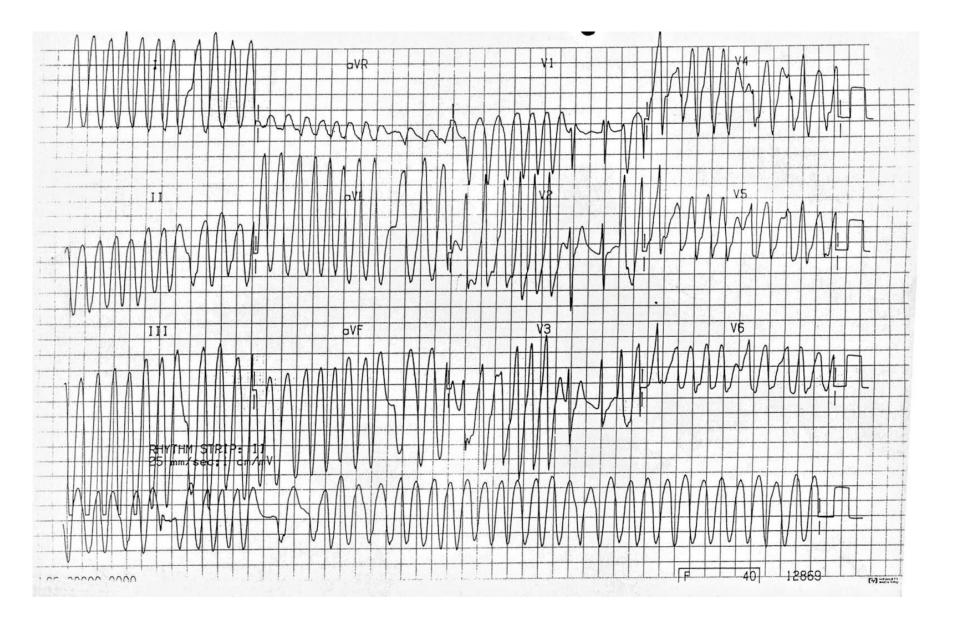
Antidromic



Orthodromic AVRT



Antidromic AVRT

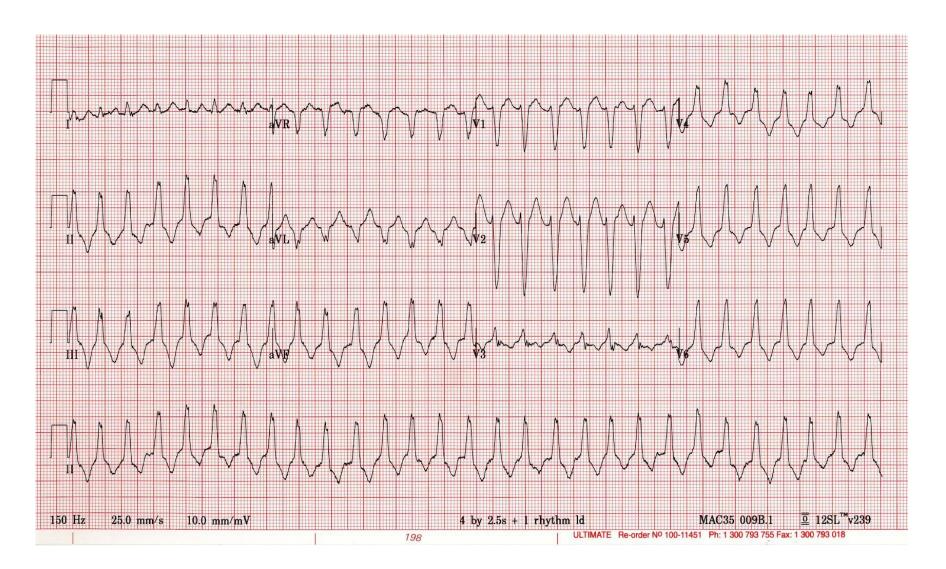


Pre-excited atrial fibrillation

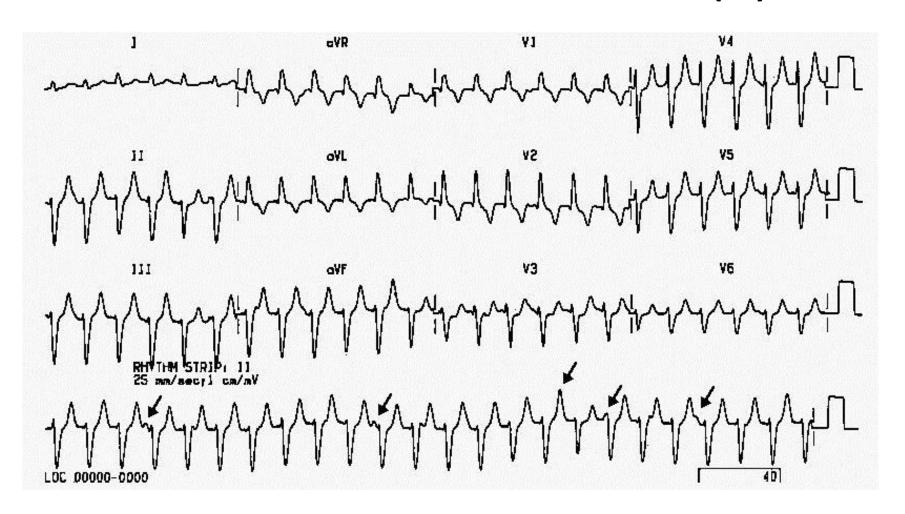
Broad complex tachycardias

VT vs SVT with aberrancy

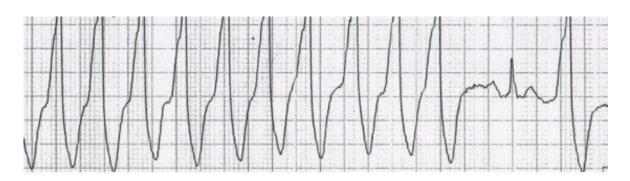
VT or SVT?



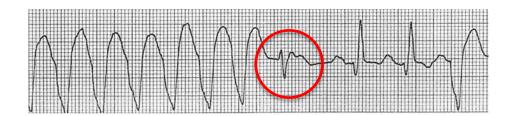
Features that favour VT (1)



Features that favour VT (2)



Capture beat

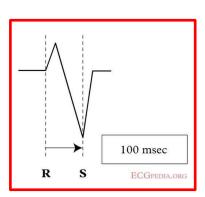


Fusion beat

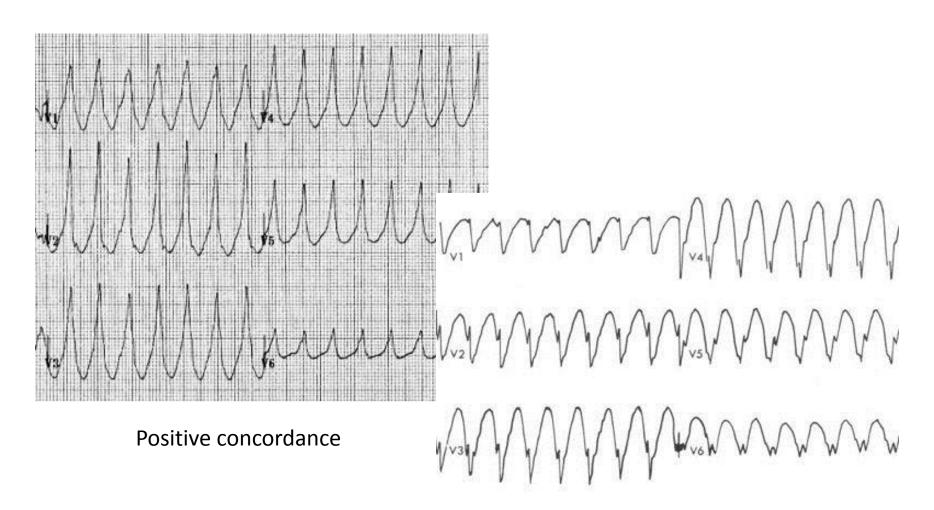


Brugada's sign

Josephson's sign

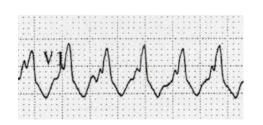


Features that favour VT (3)



Negative concordance

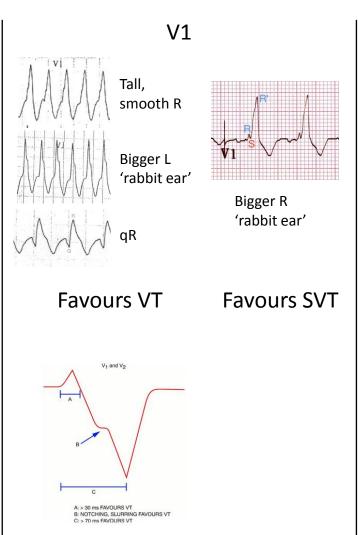
Features that favour VT (4)

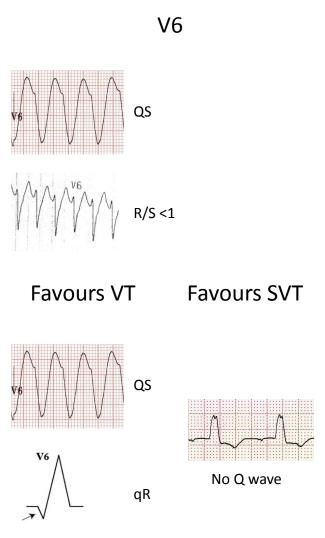


RBBB-like morphology

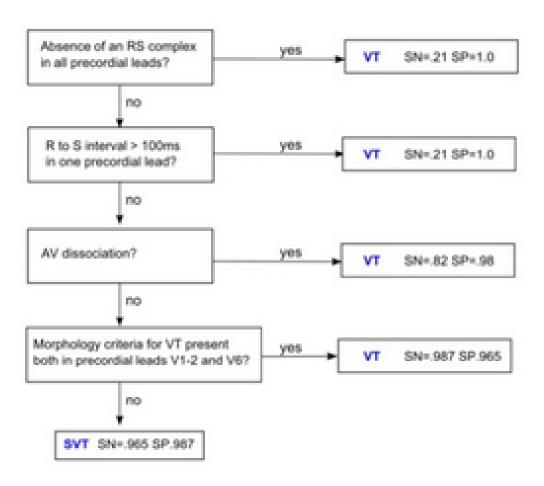


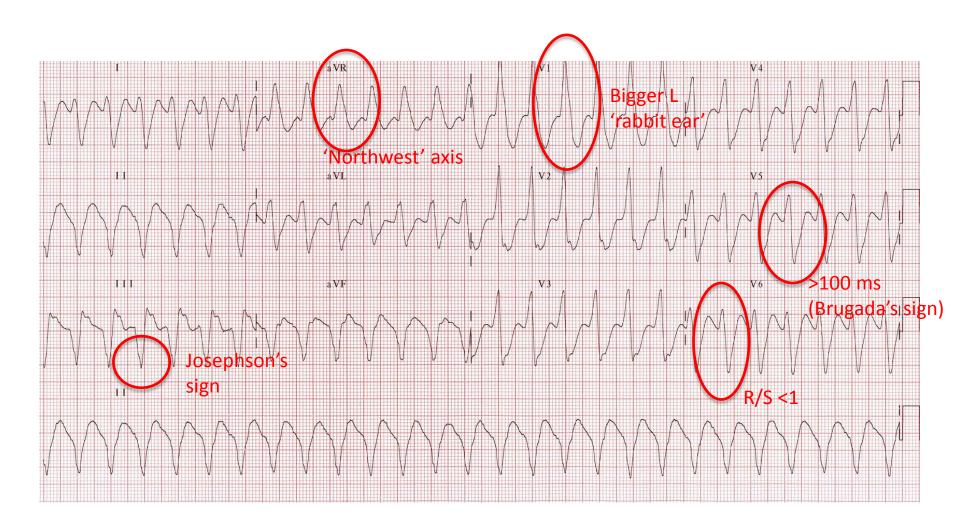
LBBB-like morphology

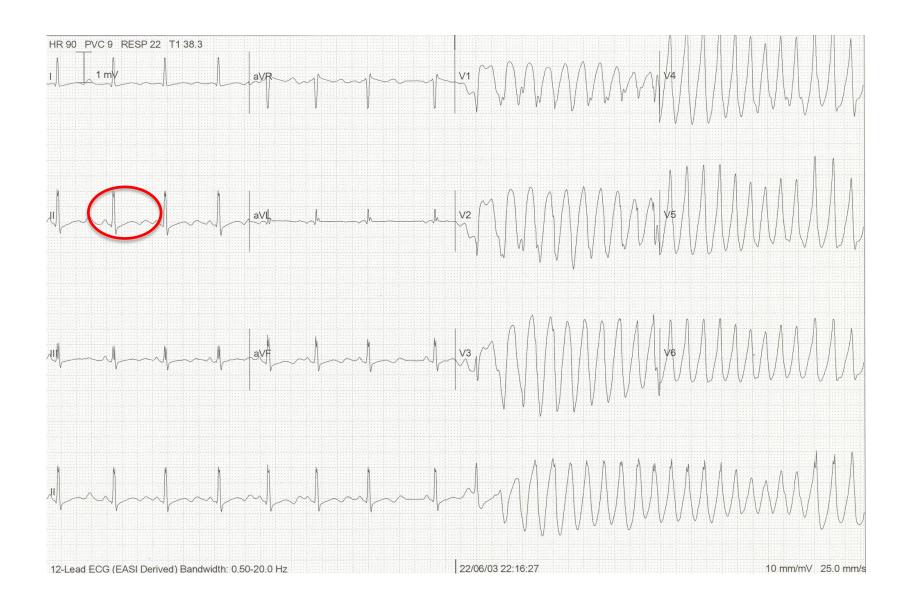




Brugada Algorithm

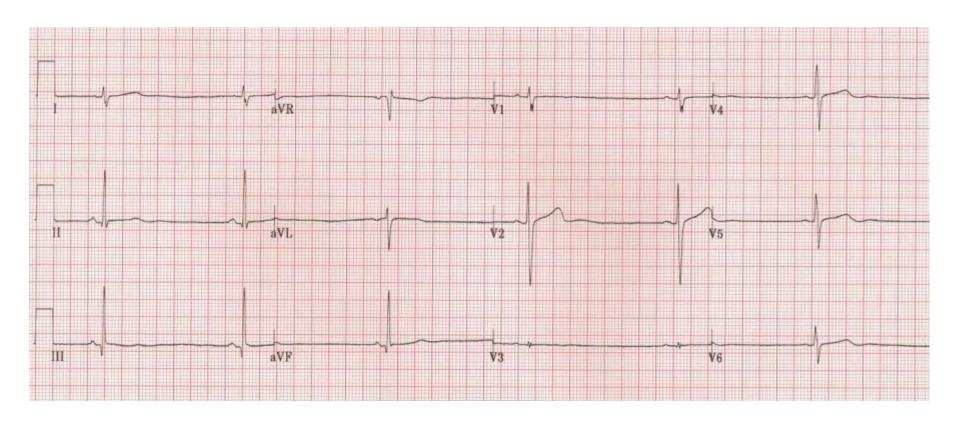






Polymorphic VT (Torsades de Pointes)

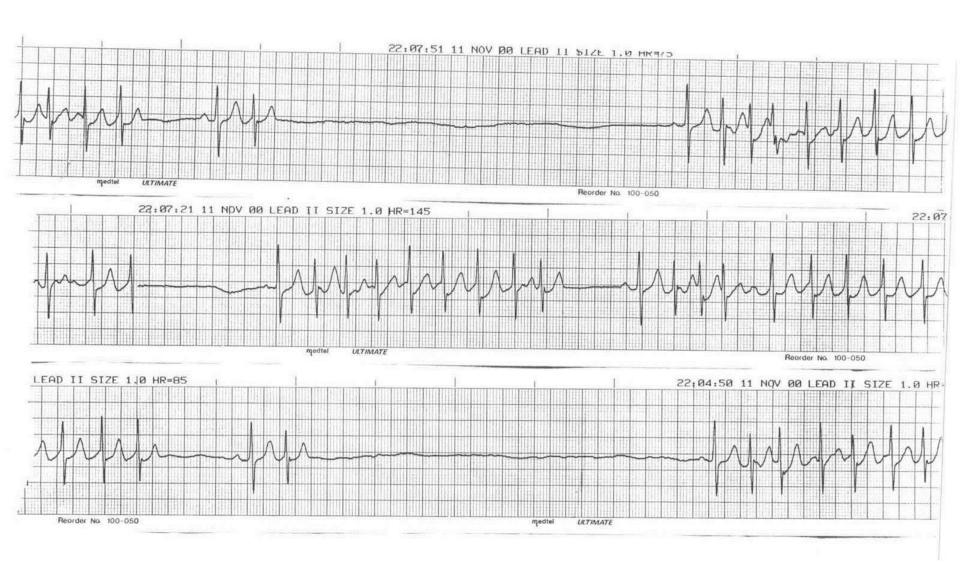
Bradycardia



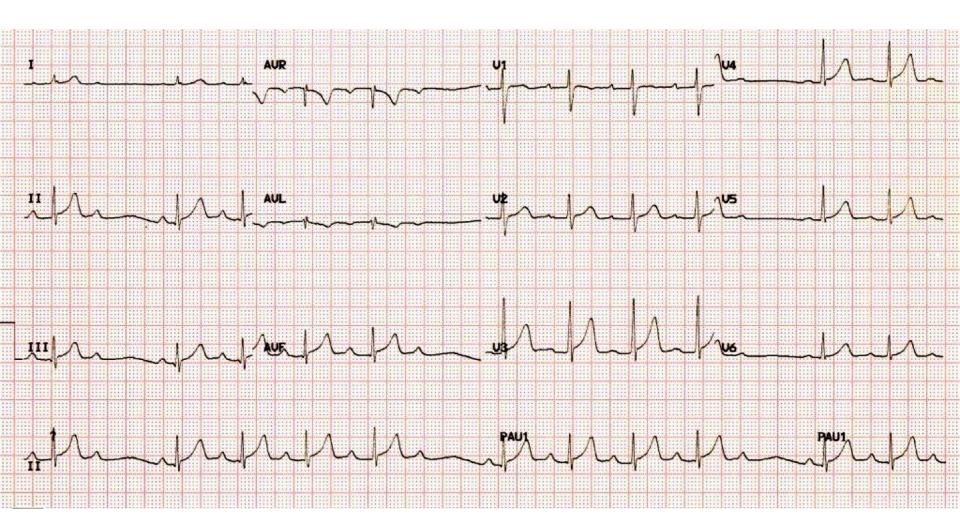
Sinus bradycardia



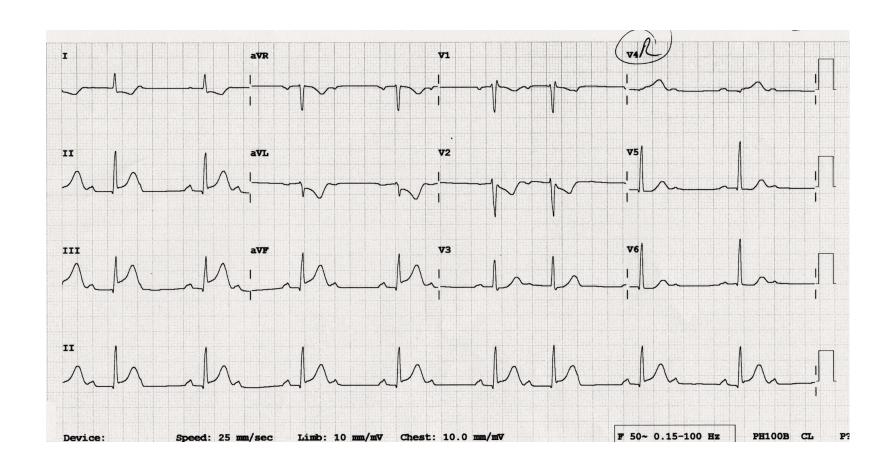
Sinus arrest



Tachy-brady syndrome



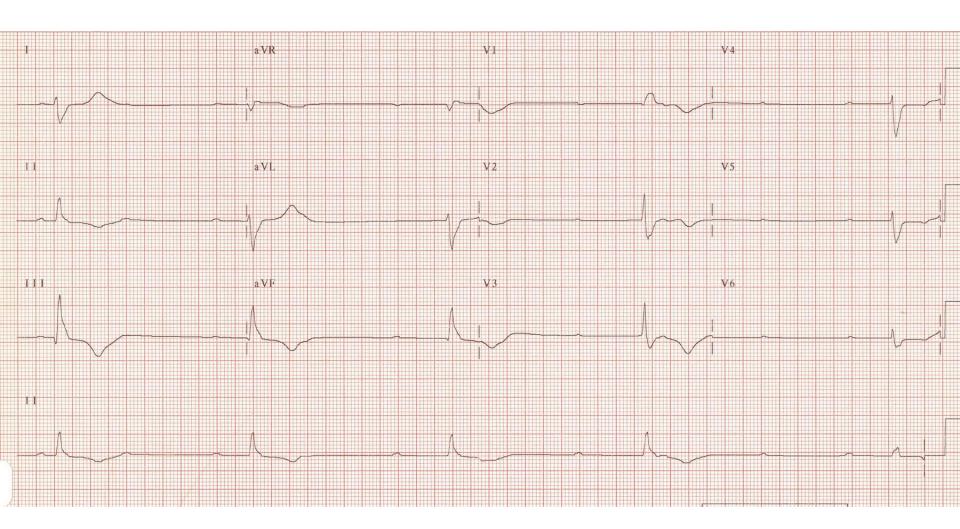
Mobitz type I 2nd degree AV block (Wenckebach)



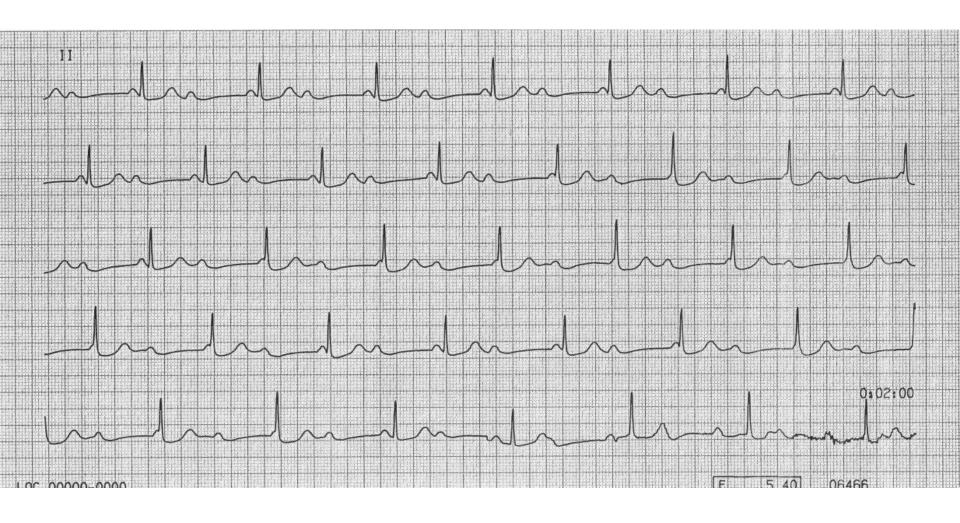
2:1 AV block and Wenckebach (in setting of acute inferior/RV MI)



Mobitz type II 2nd degree AV block

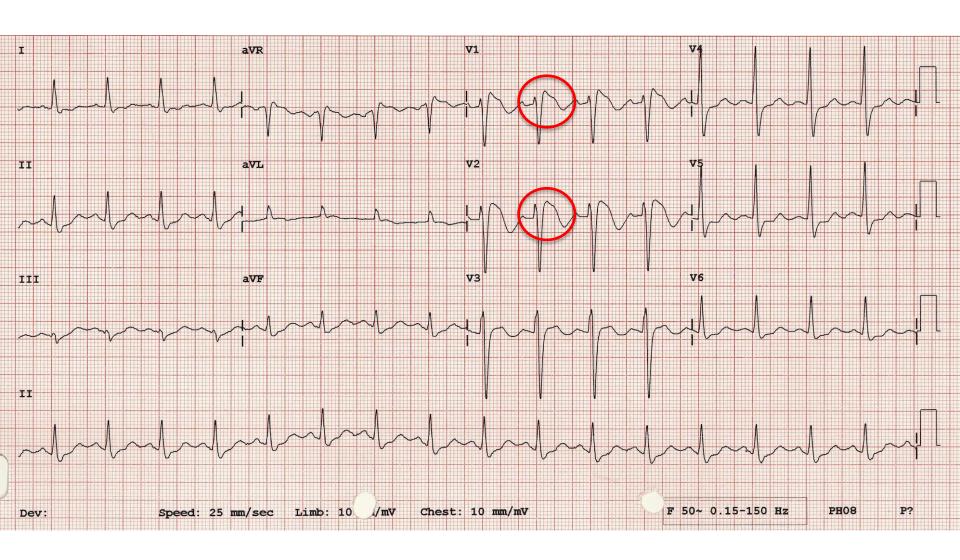


3rd degree AV block



3rd degree AV block (isorhythmic AV dissociation

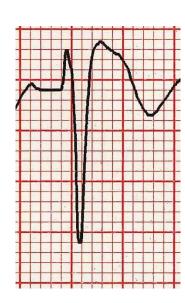
Other ECG syndromes



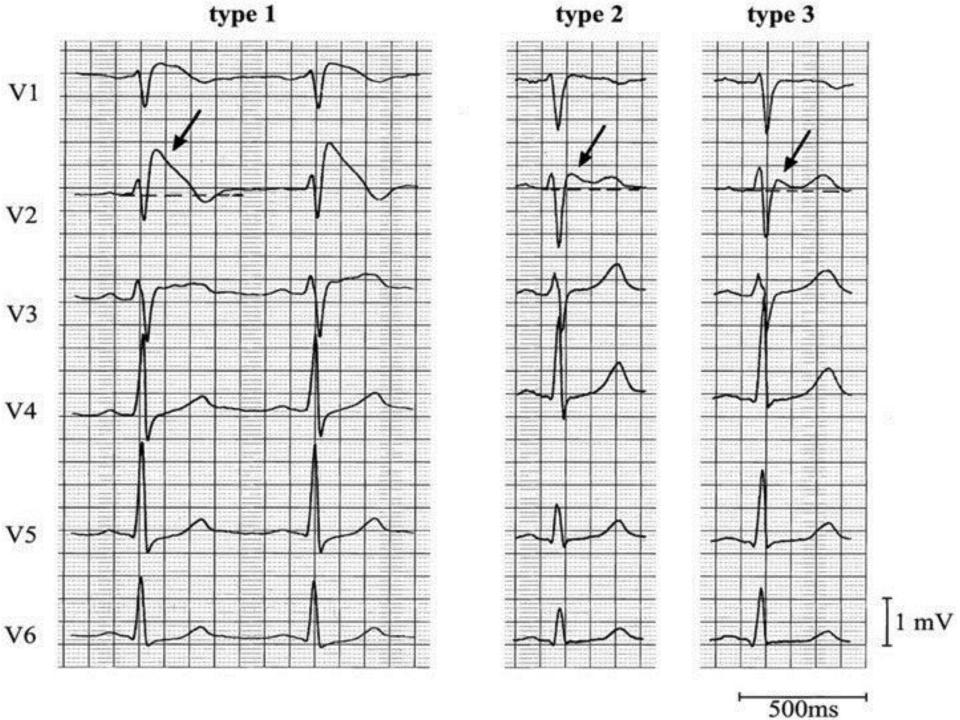
Type 1 Brugada pattern

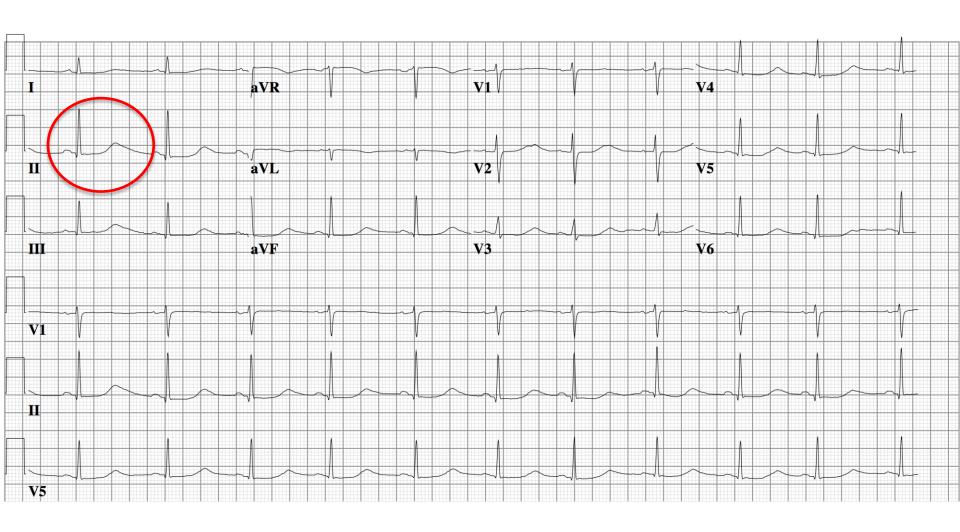
Brugada syndrome

- Mutation in cardiac sodium channel gene
- >60 known mutations, >50% sporadic
- Type 1 pattern:
- Can be unmasked by:
 - Fever
 - Ischaemia
 - Drugs (esp. Class 1C)
 - Electrolyte disturbance (K+)
- ICD if symptomatic



- V1-V3
- >2mm coved ST elevation
- T wave inv.

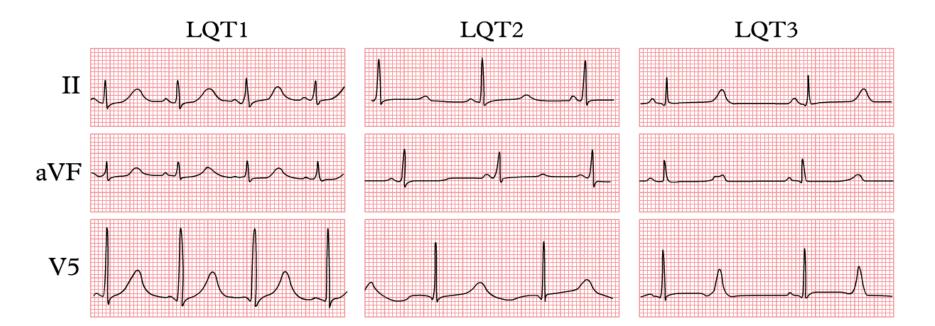


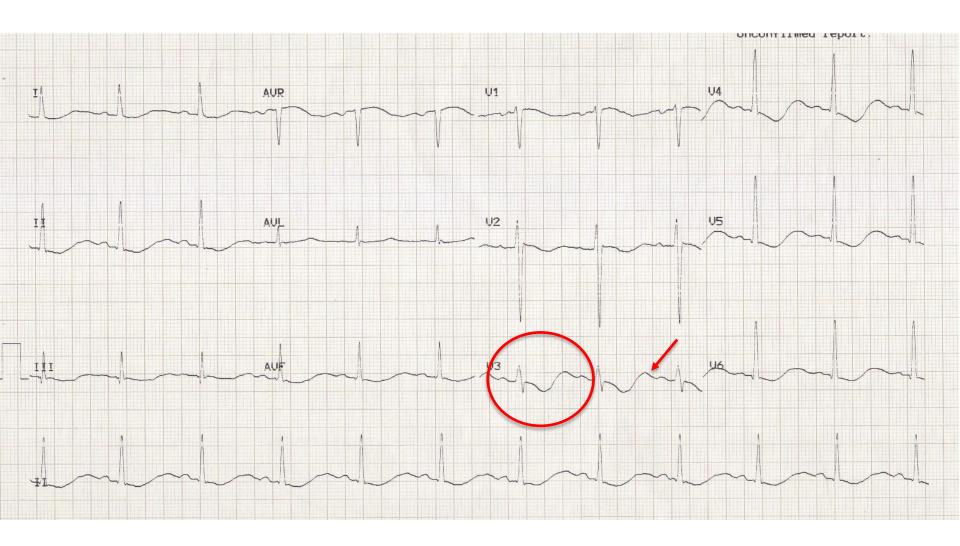


(Congenital) long QT syndrome

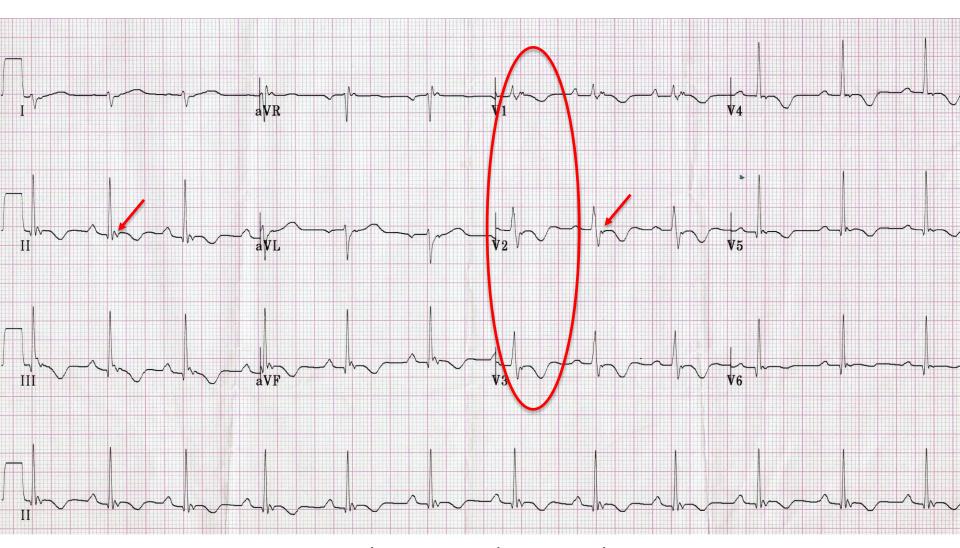
Congenital long QT syndrome

- Many subtypes
- LQT1/LQT2/LQT3 account for >97%
- Various patterns of inheritance
- QTc >440 msec (male), >460 msec (female)
- Risk of TdP greatest when QTc >500 msec
- Beta-blockers ± ICD





Acquired long QT (e.g. drugs, hypokalaemia)



Arrythmogenic right ventricular cardiomyopathy (ARVC)

ARVC

- Inherited myocardial disease
- Mainly autosomal dominant
- Fibro-fatty infiltration of RV myocardium
- Can involve LV
- Diagnosis complex: task force criteria
- ICD if high risk



KEEP CALM AND GOOD LUCK WITH THE EXAM!