

Allied Cardiovascular Health Professional (ACHP) Symposium

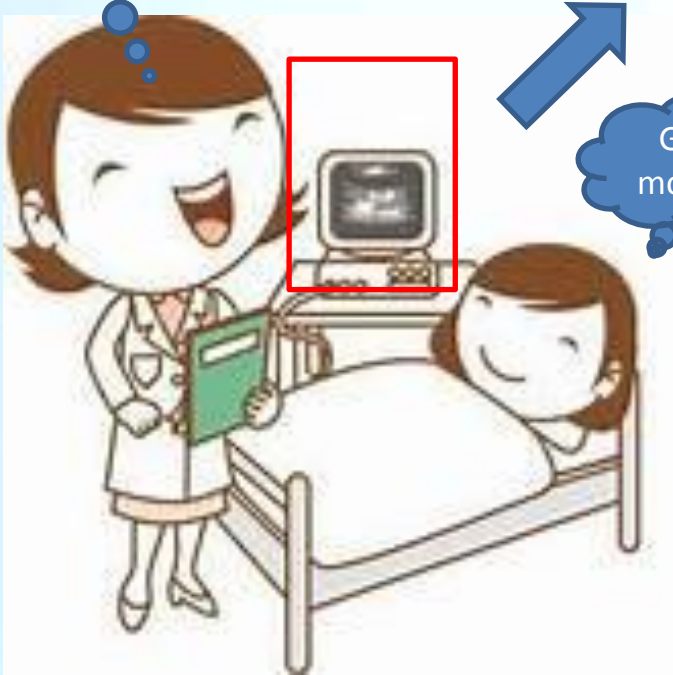
“Tips and Tricks” from Case Sharing in Cardiac Catheterization Laboratory
and Coronary Care Unit

Interesting ECG Sharing



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1st June 2019

How come VF
can still
talking.....



Good
morning

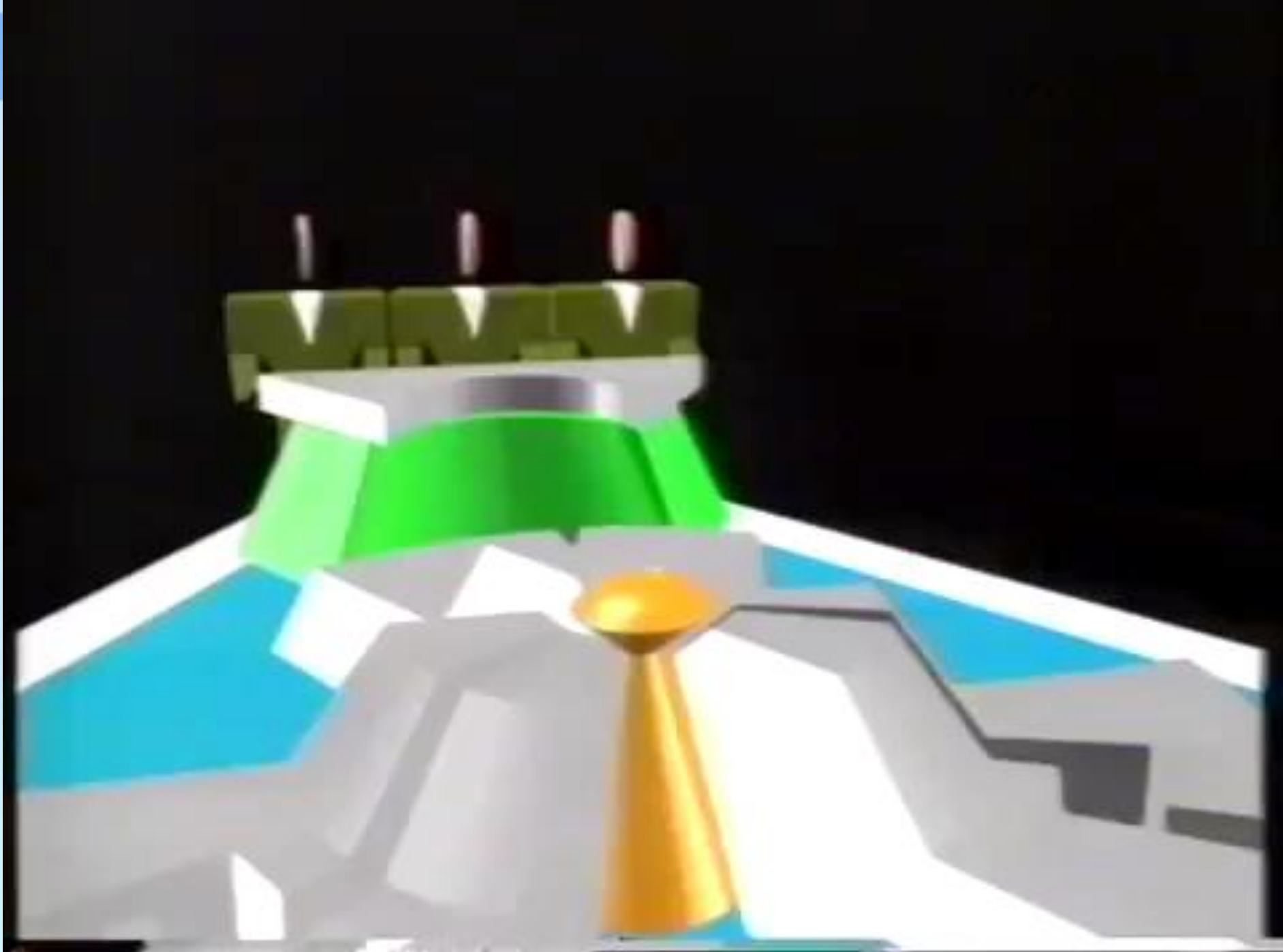
Shock!!



Importance

- Sheilini & Devi (2014) found that there was significant association between the knowledge of nurses on ECG monitoring and interpretation and area of work experience and years of work experience.
- The PULSE (Practical Use of the Latest Standards for Electrocardiography) Trial is a 5-year multisite randomized clinical trial with purpose to “test the effect of implementing newly developed practice standards for ECG monitoring on nurses’ knowledge, quality of care, and patient outcomes” (Funk, PULSE proposal, 2007, p33)
- A recent finding from PULSE Trial has shown that nurses’ ECG monitoring knowledge can improve after the online ECG educational course (Funk et al, 2011)







ECG



Question? Answer

Case 1

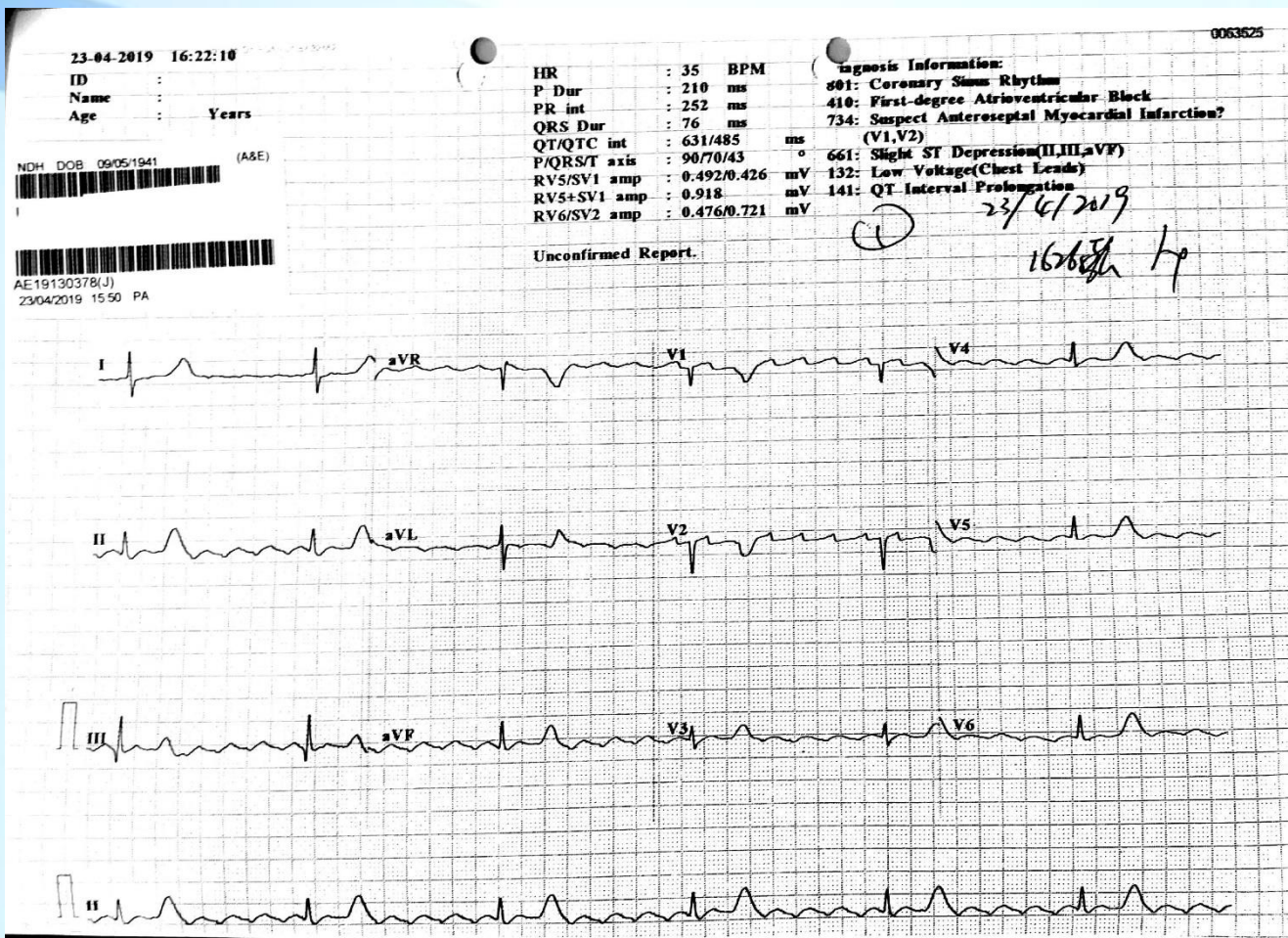
Case 1

- Madam Chan, 77-year old lady with history of hypertension, DM, Hypothyroid and atrial flutter was admitted due to dizziness while crossing border to HK. She complaint sweating, SOB, chest discomfort.
- On admission, she was fully conscious. The blood pressure was 102/37 mmHg, with a pulse rate of 41 beats /min. She was taking Aspirin, Dilitiazem, Diamicon, Metformin, Betaloc, Pantaloc, Zocor and Thyroxine. Physical examination showed no focal neurological deficit.

ECG on admission:

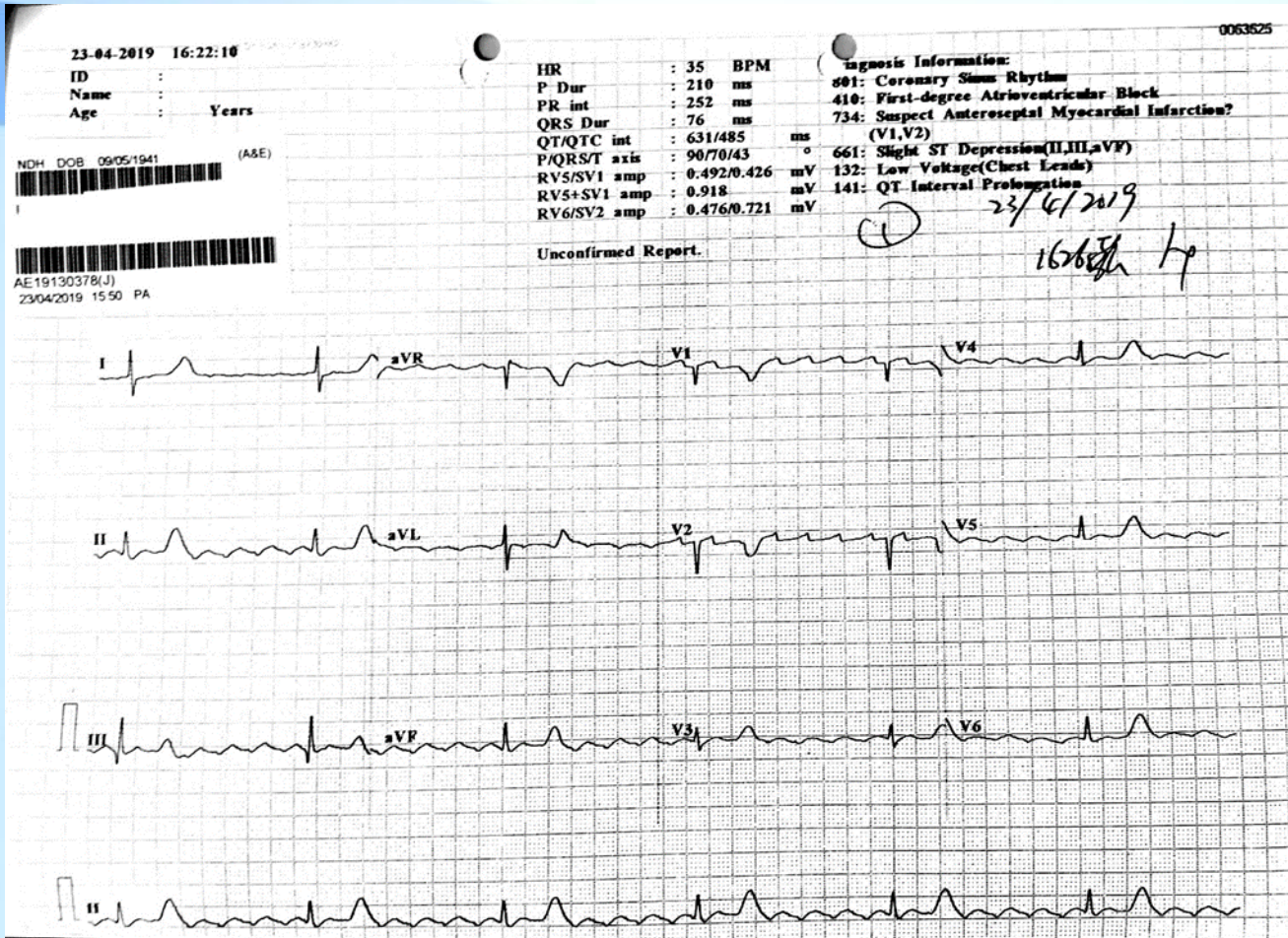


Case 1. ECG on Admission , Please comment the ECG pattern for most suitable one!



- A. Slow AF
- B. 2:1 Heart Block
- C. Atrial Flutter with complete AV block
- D. All of above



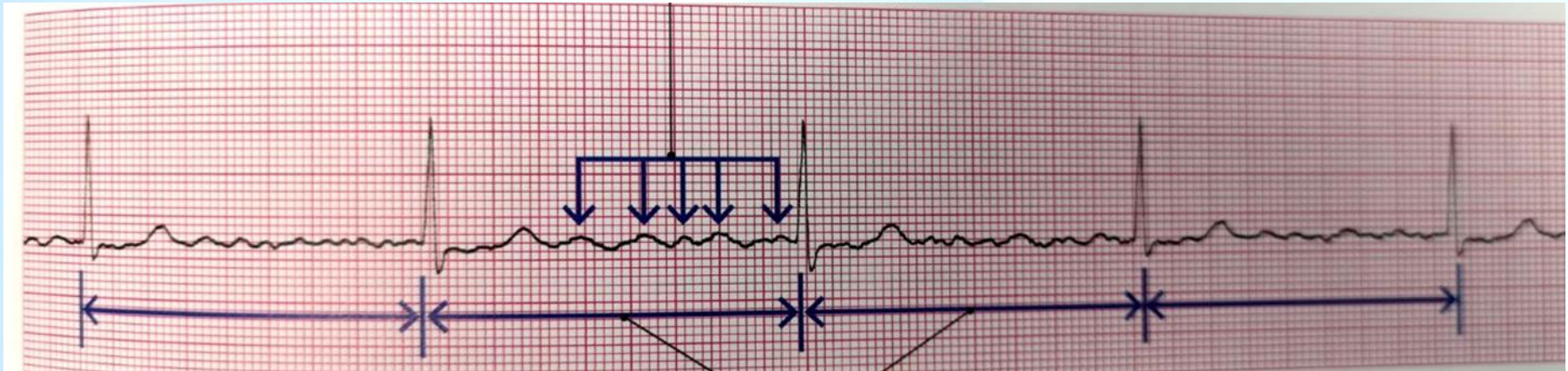


Answer : C

- A. Slow AF
- B. 2:1 Heart Block
- C. Atrial flutter with complete AV block
- D. All of above

Diagnosis:

- Atrial Flutter with complete AV block
- Fibrillary wave of atrial flutter, regular ventricular rhythm



Management:

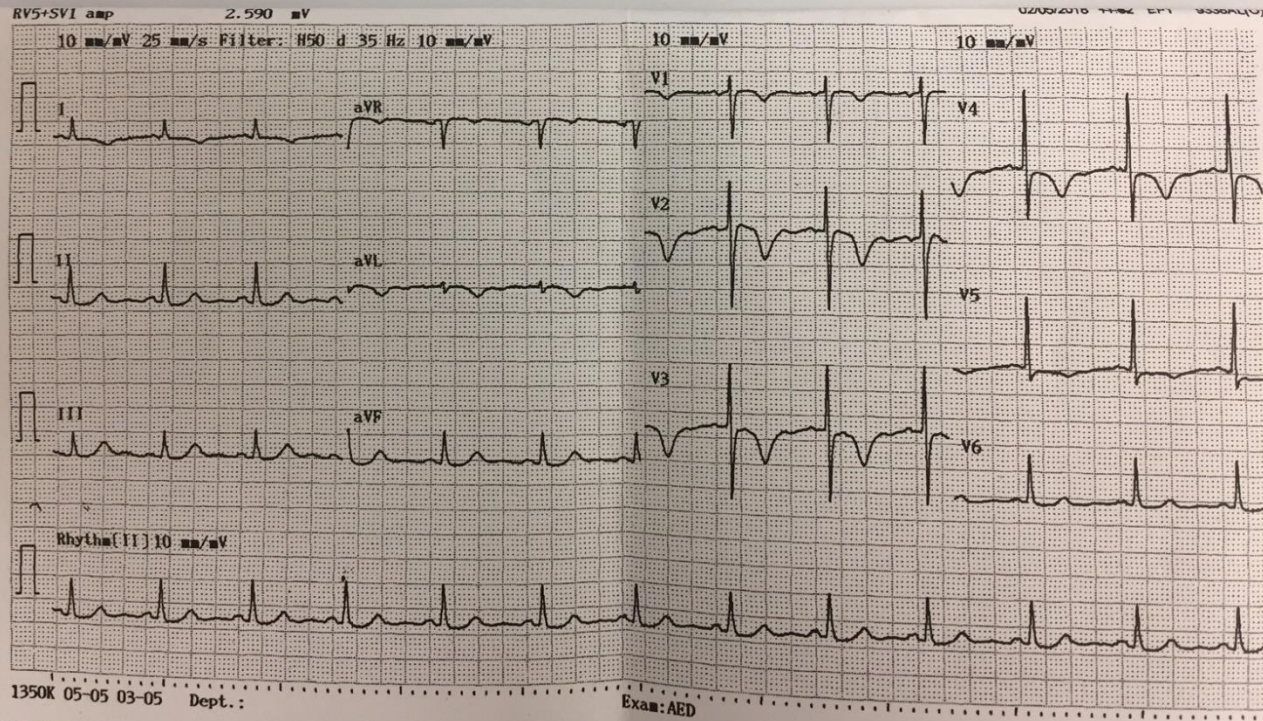
- Need continuously monitored
- Evaluate for reversible causes such as myocardial ischemia, hypothyroidism, hyperkalemia, and drugs that depress conduction.
- Consider permanent pacemaker if no reversible causes can be identified.



Case 2

Case 2:

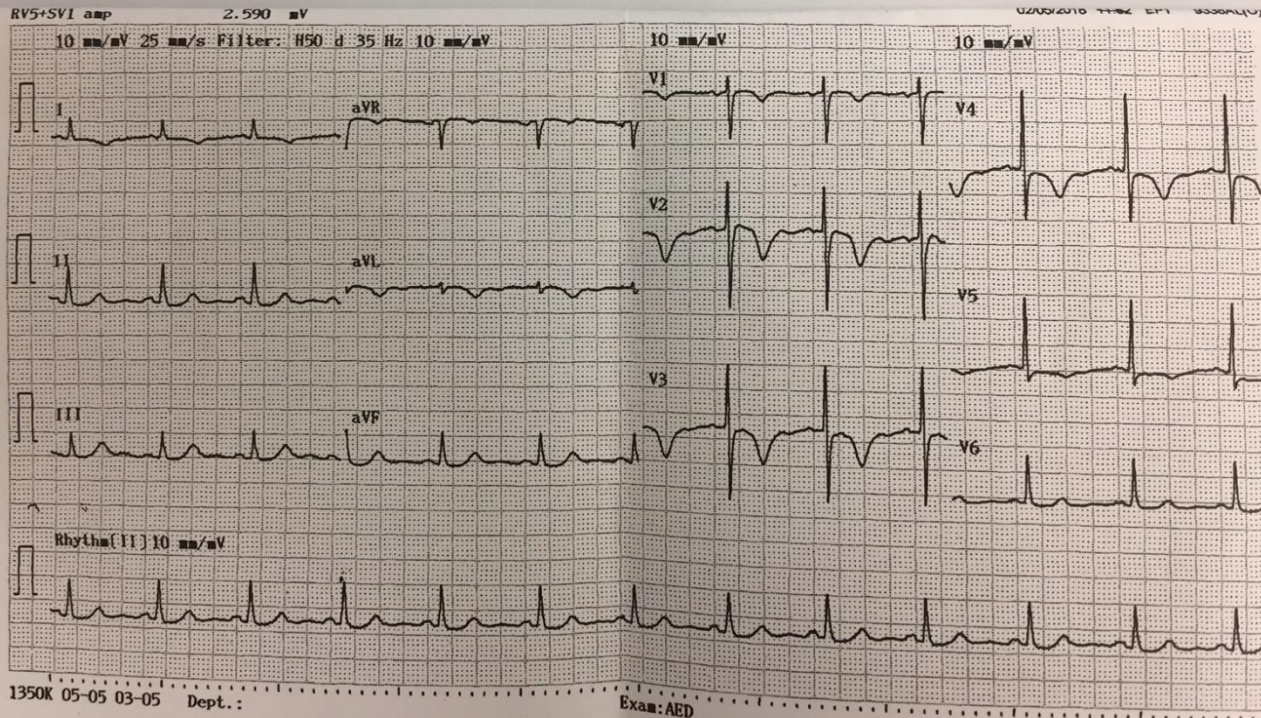
F/61, HT/IFG, presented with chest pain, hsTnT 63 / 44, CPK normal



- A. Brugada
- B. De-Winter
- C. Wolff-Parkinson-White
- D. Wellens'



Answer: D

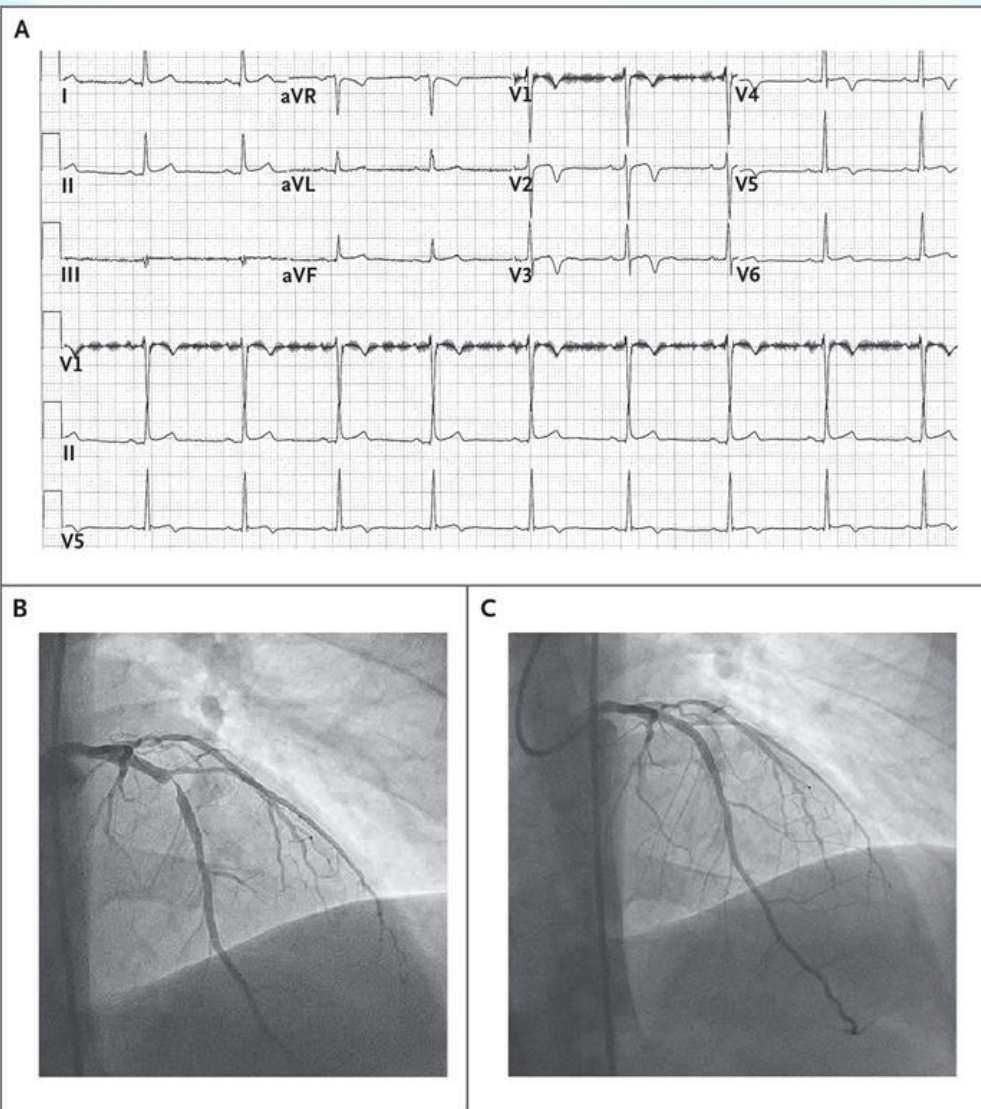


- A. Brugada
- B. De-Winter
- C. Wolff-Parkinson-White
- D. Wellens'

Wellens' Syndrome

- Wellens syndrome was first described in the early 1980s by de Zwaan
- Some patients with ischemic chest pain present with deep "coronary" T wave inversions in multiple precordial leads (e.g., V1 to V4) with or without cardiac enzyme elevations and with minimal or no ST elevations.
- called the Wellens' pattern or syndrome or the left anterior descending coronary artery (LAD)-T wave inversion pattern, is typically caused by a tight stenosis in the LAD coronary artery system. The natural history of this pattern is unfavorable, with a high incidence of recurrent symptoms and myocardial infarction.
- Emergency angiography is justified, with the hope of avoiding an extensive anterior wall MI through early intervention.





Coronary angiography performed 12 hours after presentation showed stenosis of 95% of the mid-left anterior descending artery

Wellen's syndrome criteria

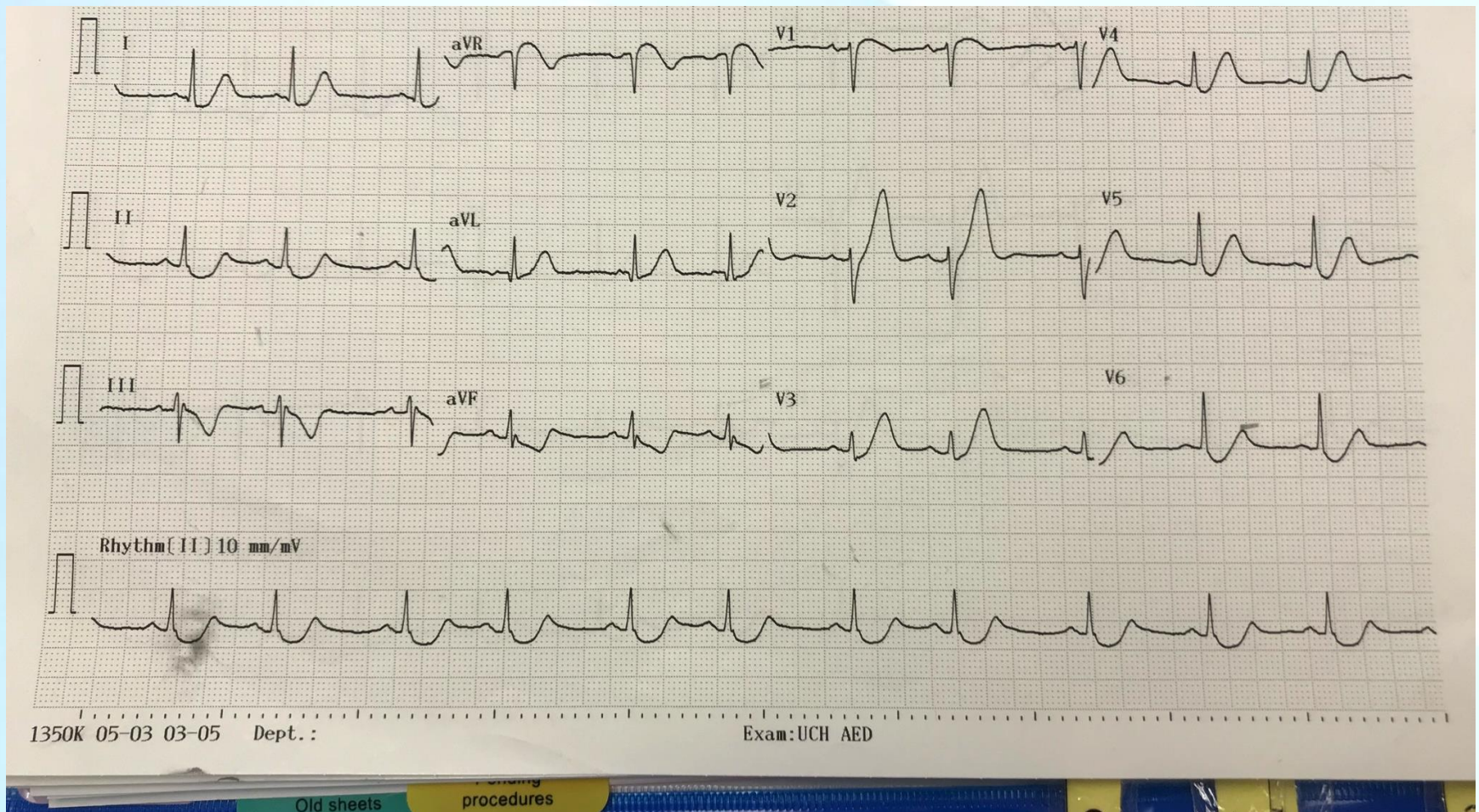
- Prior history of chest pain
- During chest pain: ECG is normal or with mild ST elevation or depression, or with terminal negative deflection of the T wave in V1 and V2
- Cardiac enzymes are normal or mildly elevated
- No pathologic precordial Q-waves
- No loss of precordial R-waves
- Deeply inverted or biphasic T-waves in V2 and V3, possibly V1, V4, V5 and/or V6 when pain free



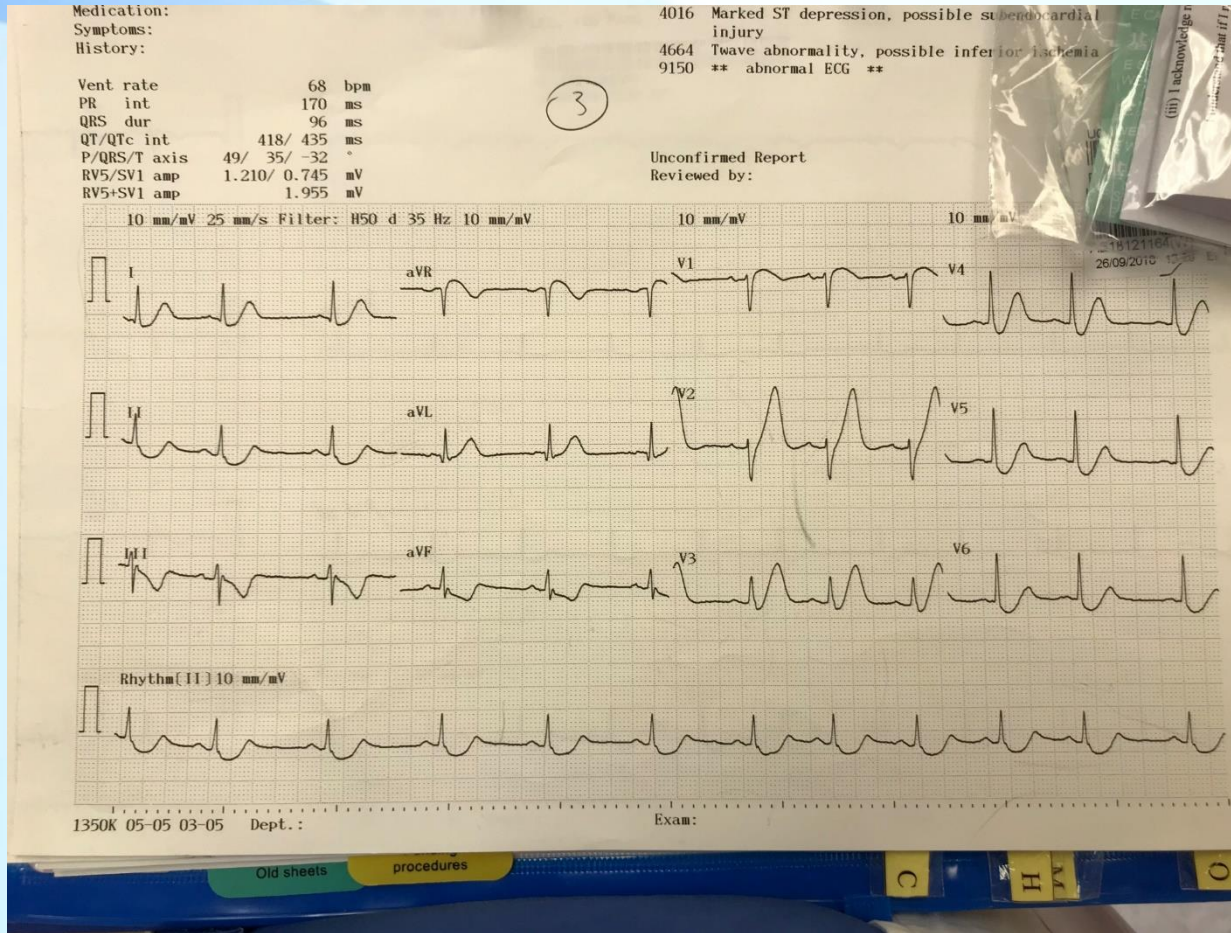
Case 3

Case 3

M/70. presented with chest pain



What is the ECG look like?

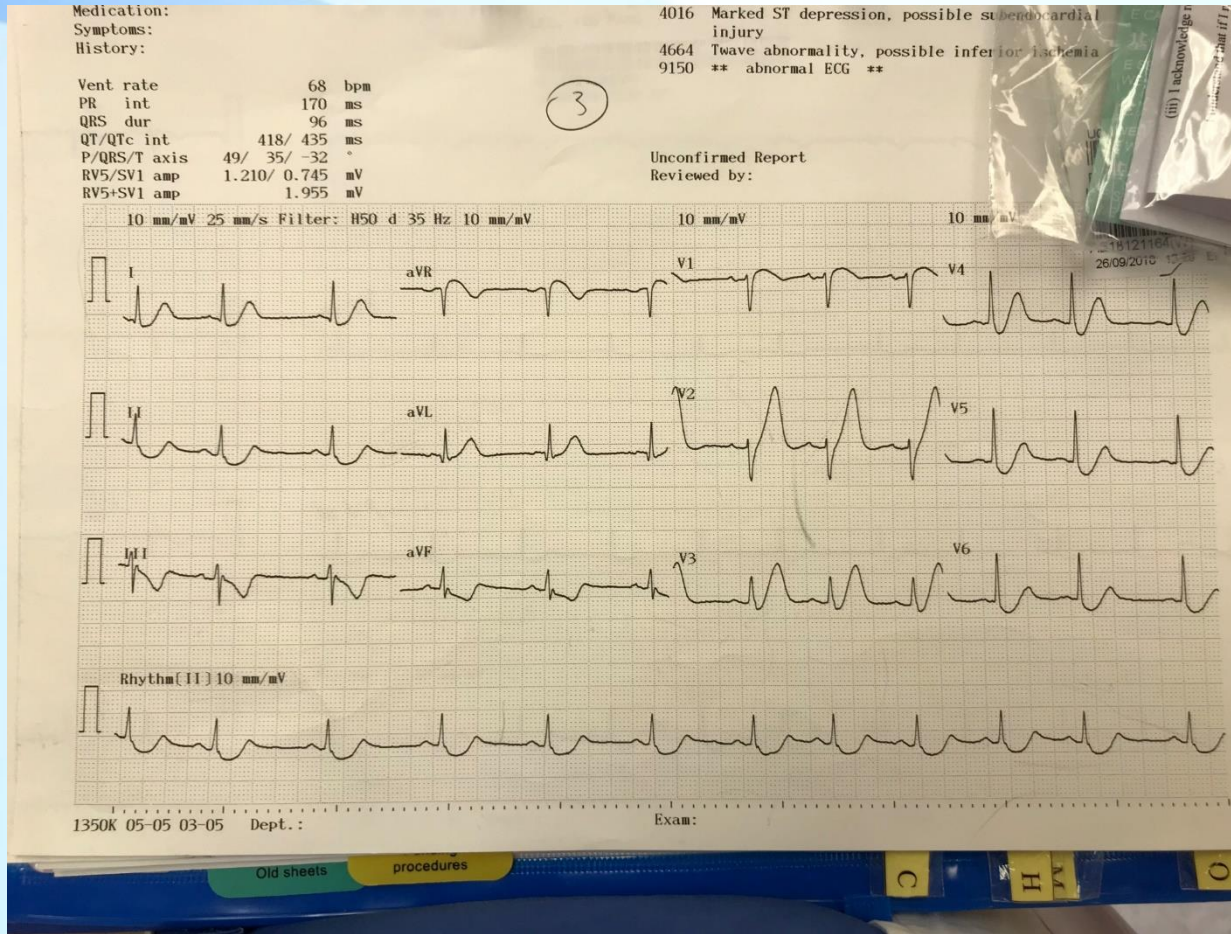


- A. Brugada
- B. De-Winter
- C. Wolff–Parkinson–White
- D. Wellens'



Answer: B

What is the ECG look like?



A. Brugada

B. De-Winter

C. Wolff–Parkinson–White

D. Wellens'

De-Winter's sign

- The de-Winter ECG pattern is an anterior STEMI equivalent that presents without obvious ST segment elevation. First reported by de Winter in 2008
- The de-Winter ECG pattern is an **anterior STEMI equivalent** that presents *without* obvious ST segment elevation.
- Key diagnostic features include **upsloping ST depression**↓ and **peaked T waves**↑ in the **precordial leads**.
- The de Winter pattern is seen in **~2% of acute LAD occlusions** and is under-recognised by clinicians
- It is important for cardiologists and emergency care physicians to recognise this distinct ECG pattern, so they can triage such patients for immediate reperfusion therapy.



De Winter ST/T-Waves



@ECGTraining

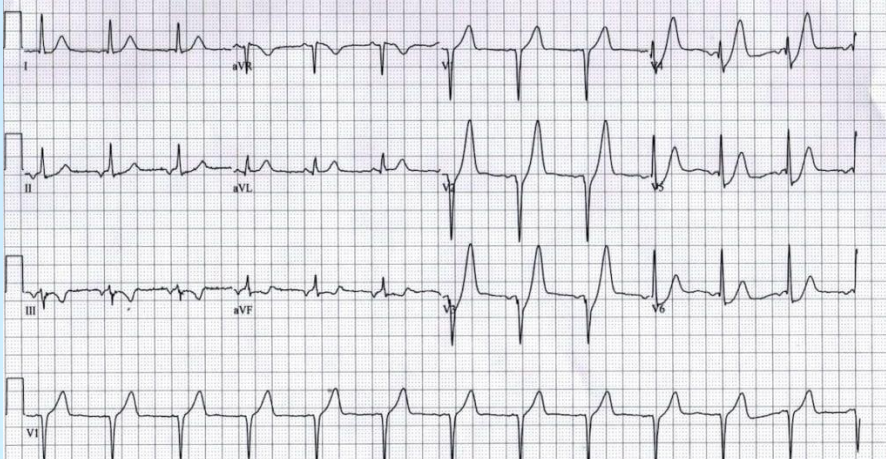
ECGMedicalTraining.com



More examples

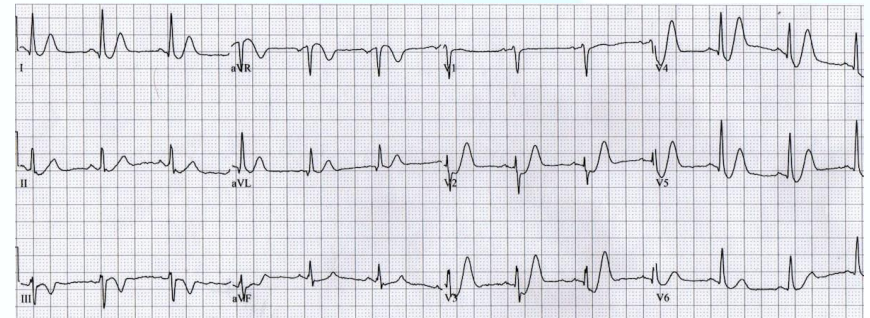
De Winter T waves

- Upsloping ST depression in the precordial leads ($> 1\text{mm}$ at J-point).
- Peaked anterior T waves (V2-6), with the ascending limb of the T wave commencing below the isoelectric baseline.
- Subtle ST elevation in aVR $> 0.5\text{mm}$.

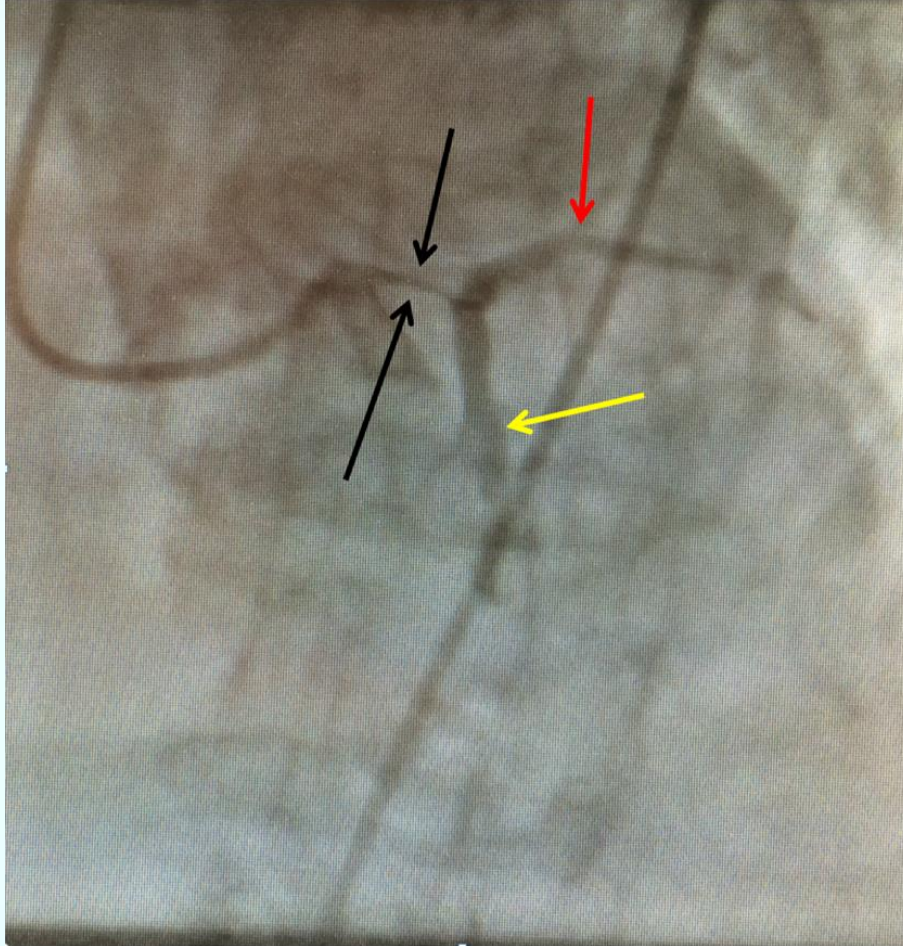


De Winter T waves

- Upsloping ST depression in the precordial leads ($> 1\text{mm}$ at J-point).
- Peaked anterior T waves (V2-6), with the ascending limb of the T wave commencing below the isoelectric baseline.
- Subtle ST elevation in aVR $> 0.5\text{mm}$.



Angiogram : one of example with De-Winter's

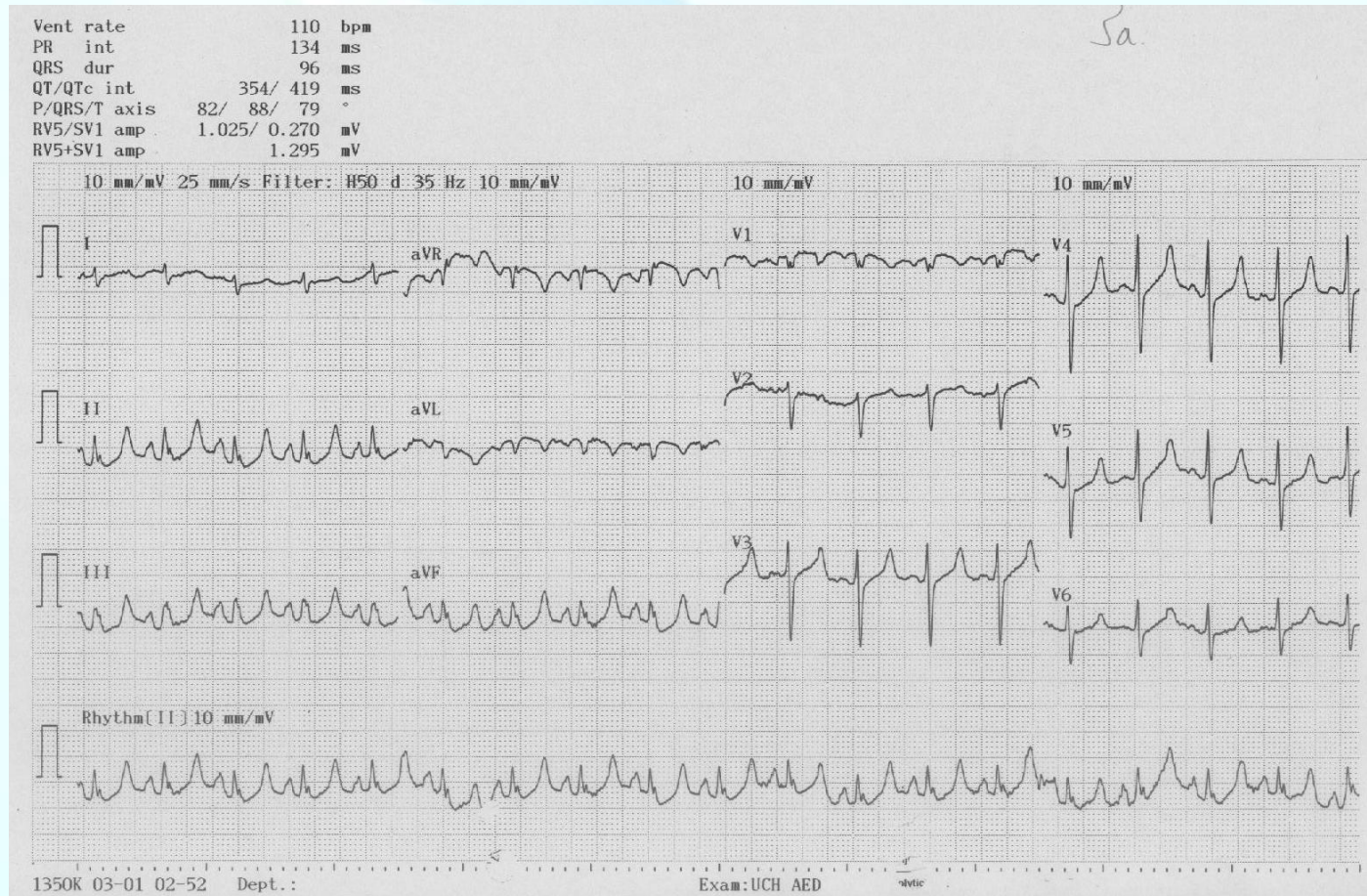


- Black arrows are very narrowed left main, red arrow is LAD with flow, and yellow arrow is circumflex with flow.
- **Interpretation: 95% left main thrombus with TIMI-2 flow, with thrombus extending to the ostial LAD and the circumflex.**

Case 4

Case 4

M/65 History of CRF and CCF. Admitted due to heart failure exacerbation. What is the most alarming ECG change?



- A. Tall T waves
- B. Tachycardia
- C. SVT
- D. None of above



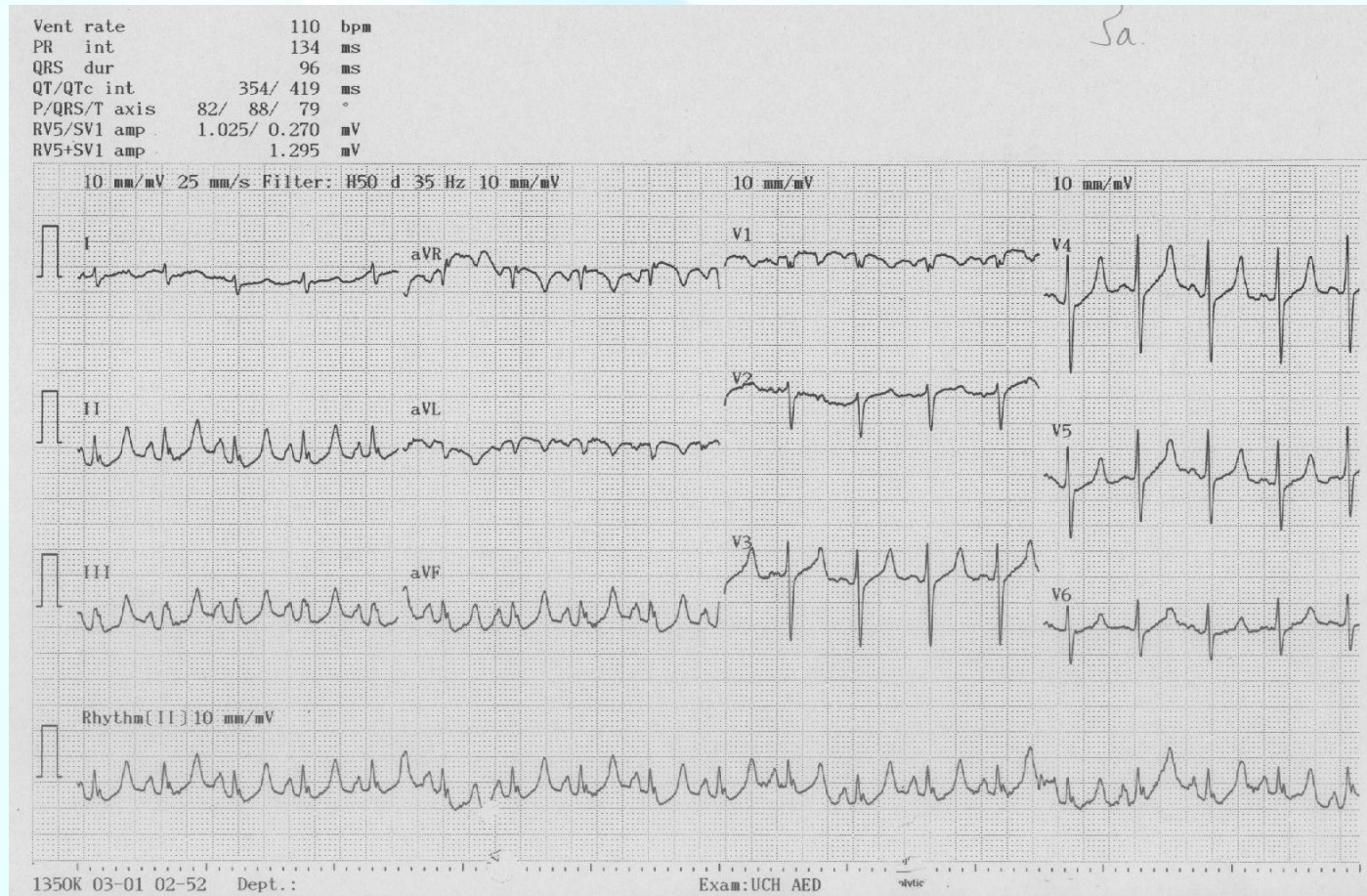
Answer: A , Tall T waves (earliest sign of $\uparrow K$, $K = 6.0 \text{ mmol/l}$)
M/65 History of CRF and CCF. Admitted due to heart failure exacerbation. What is the most alarming ECG change?

A. Tall T waves

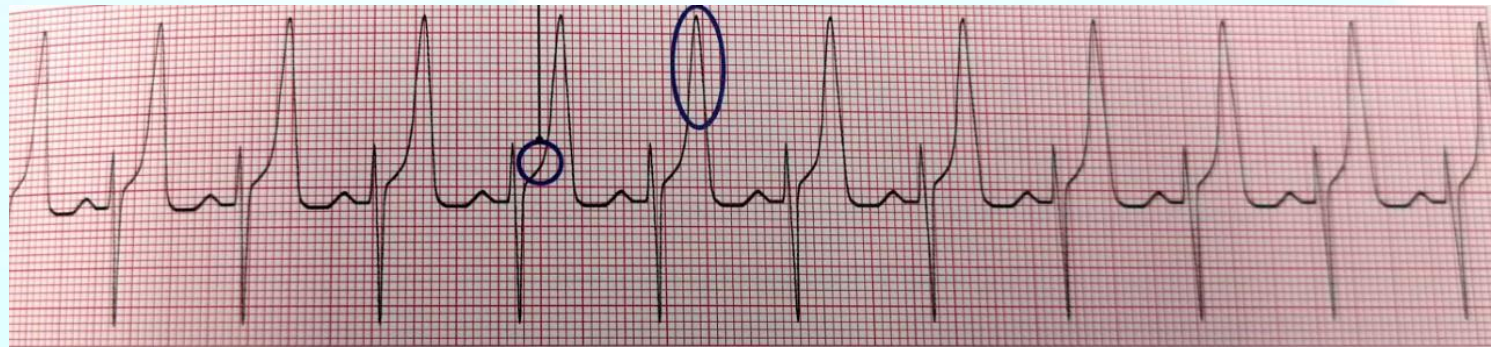
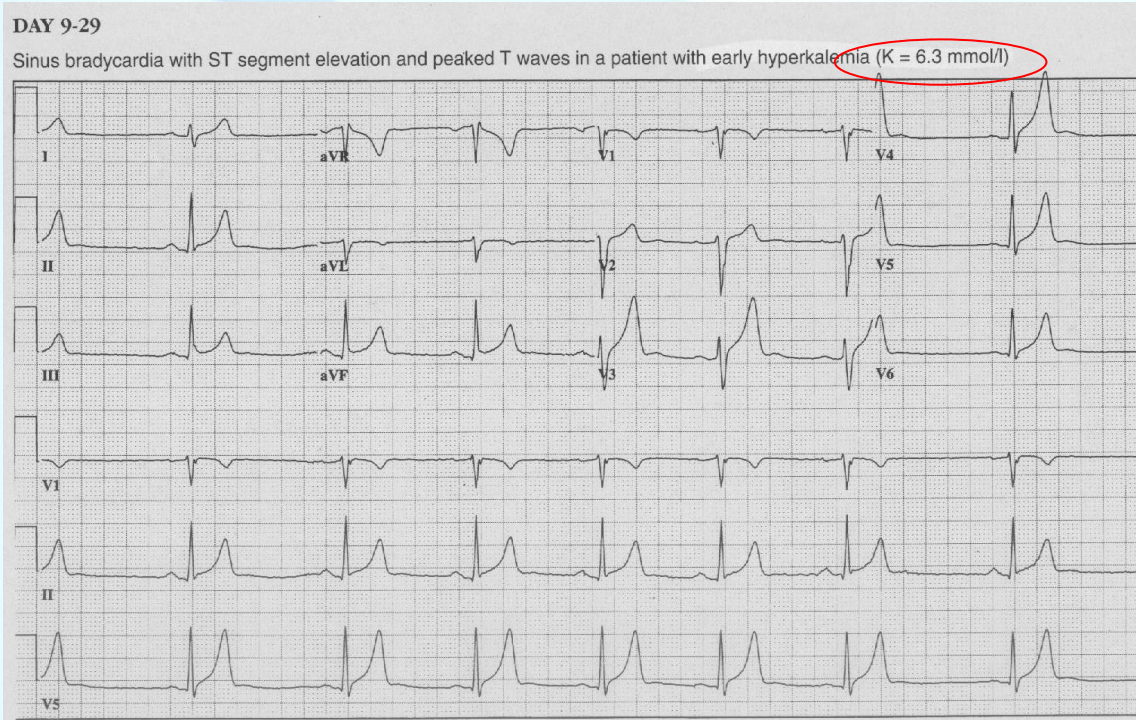
B. Tachycardia

C. SVT

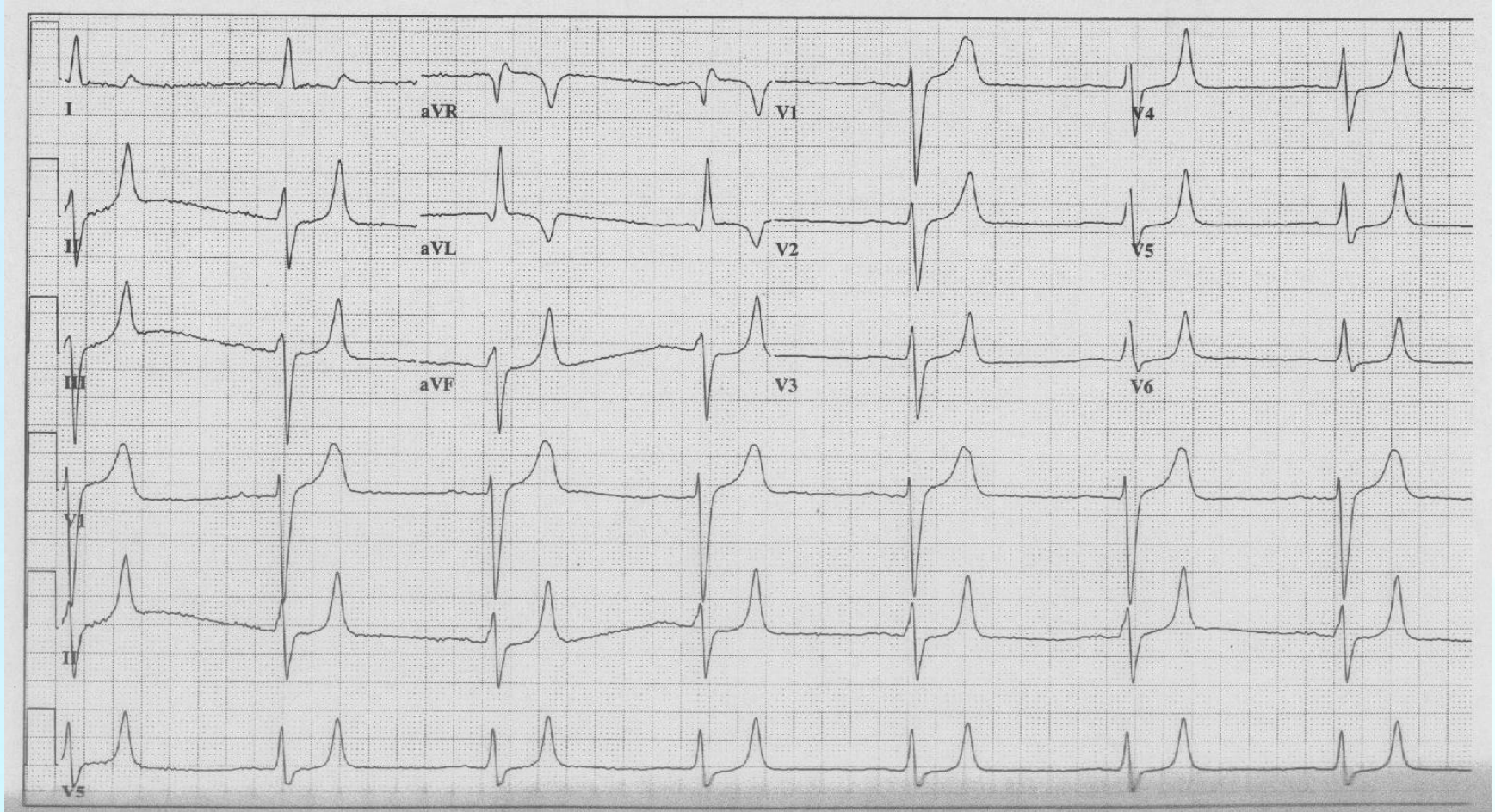
D. None of above



ECG changes in early Hyperkalemia



ECG: K = 7.0 mmol/l (flattened or absent P wave, widened QRS and bradycardia)



ECG changes: Hyperkalaemia (\uparrow K)

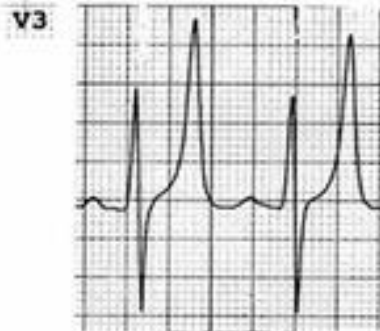
- Peaked and Tented T waves
- Widening of the QRS (severe \uparrow K, sinusoidal shape)
- \downarrow R wave amplitude
- Loss of ST segment
- \uparrow PR interval
- Loss of P wave
- Loss of U wave
- Bradycardia and AV block, BBB, ventricular standstill, VT/VF



Causes of Tall T waves

Different Causes of Tall T Waves

Hyperkalemia



Symmetric, narrow-based, pointed, tenting

Hyperacute Ischemia



Symmetric, broad-based, not tented, not pointed
QT interval tends to be long
(not in this example)

Normal Variant

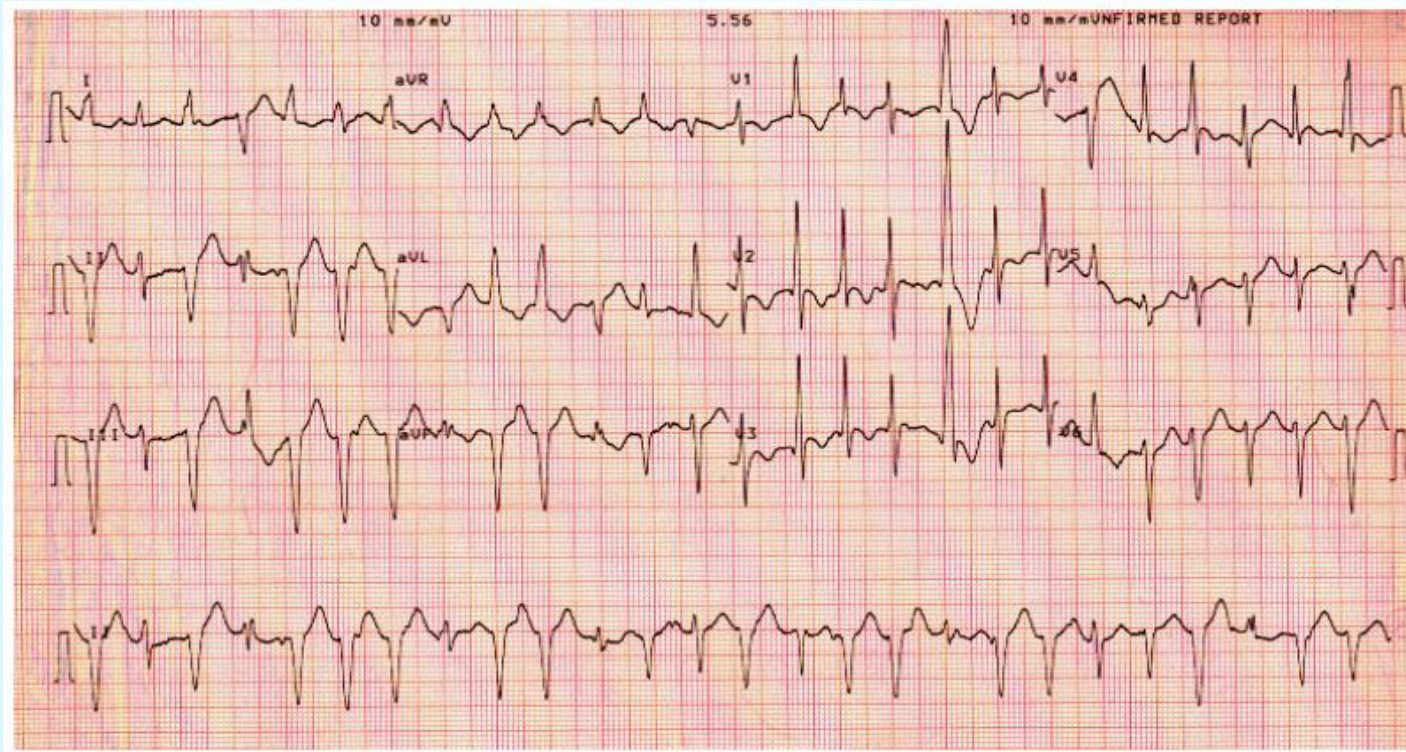


Asymmetric and not narrow

Case 5

Case 5

M/70 DM, HT, IHD, presented to AED due to palpitation, dizziness and numbness over mouth and limbs. BP ~95/60. History reviewed: taken some herbal medicine for myalgia and joint pain ~ 3hrs ago. What is the ECG diagnosis?

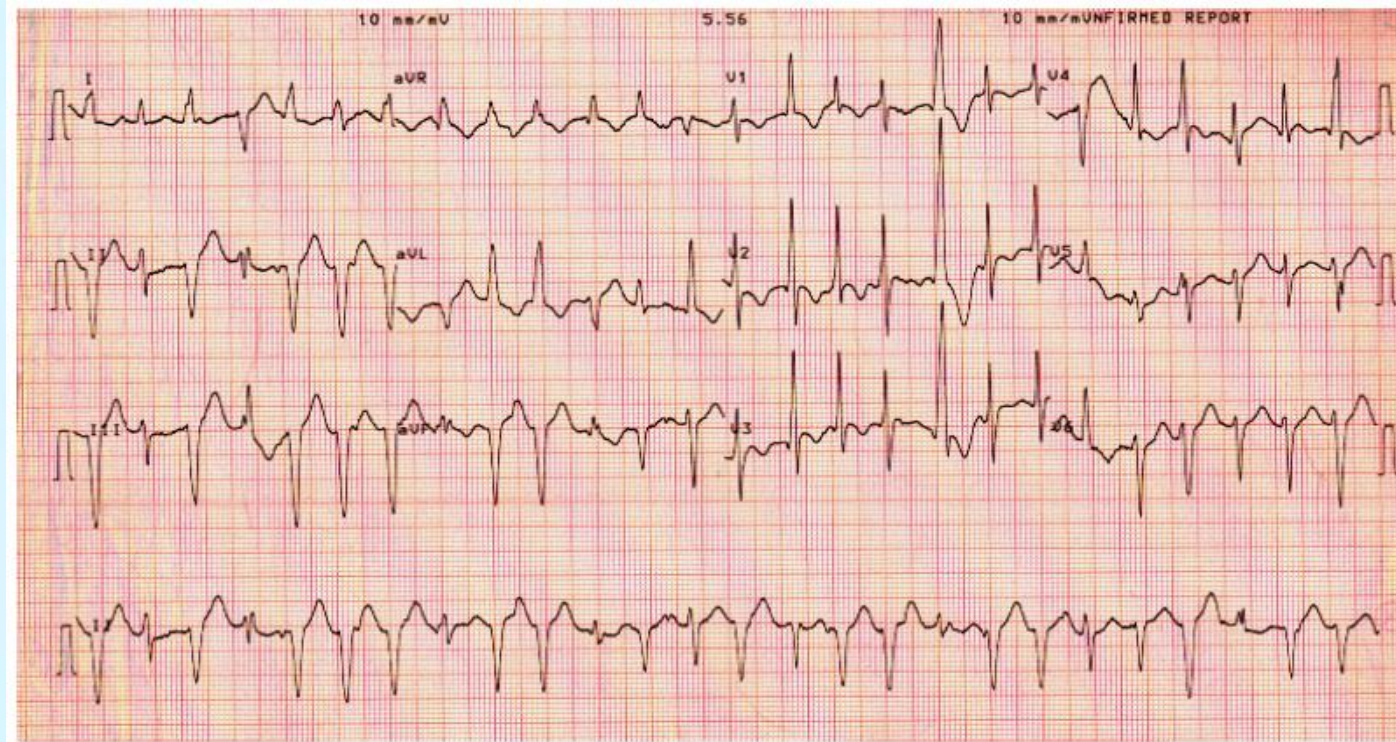


- A. Non-sustain VT
- B. Polymorphic VT
- C. Tachycardia
- D. SVT



Answer: B

M/70 DM, HT, IHD, presented to AED due to palpitation, dizziness and numbness over mouth and limbs. BP ~95/60. History reviewed: taken some herbal medicine for myalgia and joint pain ~ 3hrs ago. What is the ECG diagnosis?



- A. Non-sustain VT
- **B. Polymorphic VT**
- C. Tachycardia
- D. SVT

Polymorphic VT rate 150/min

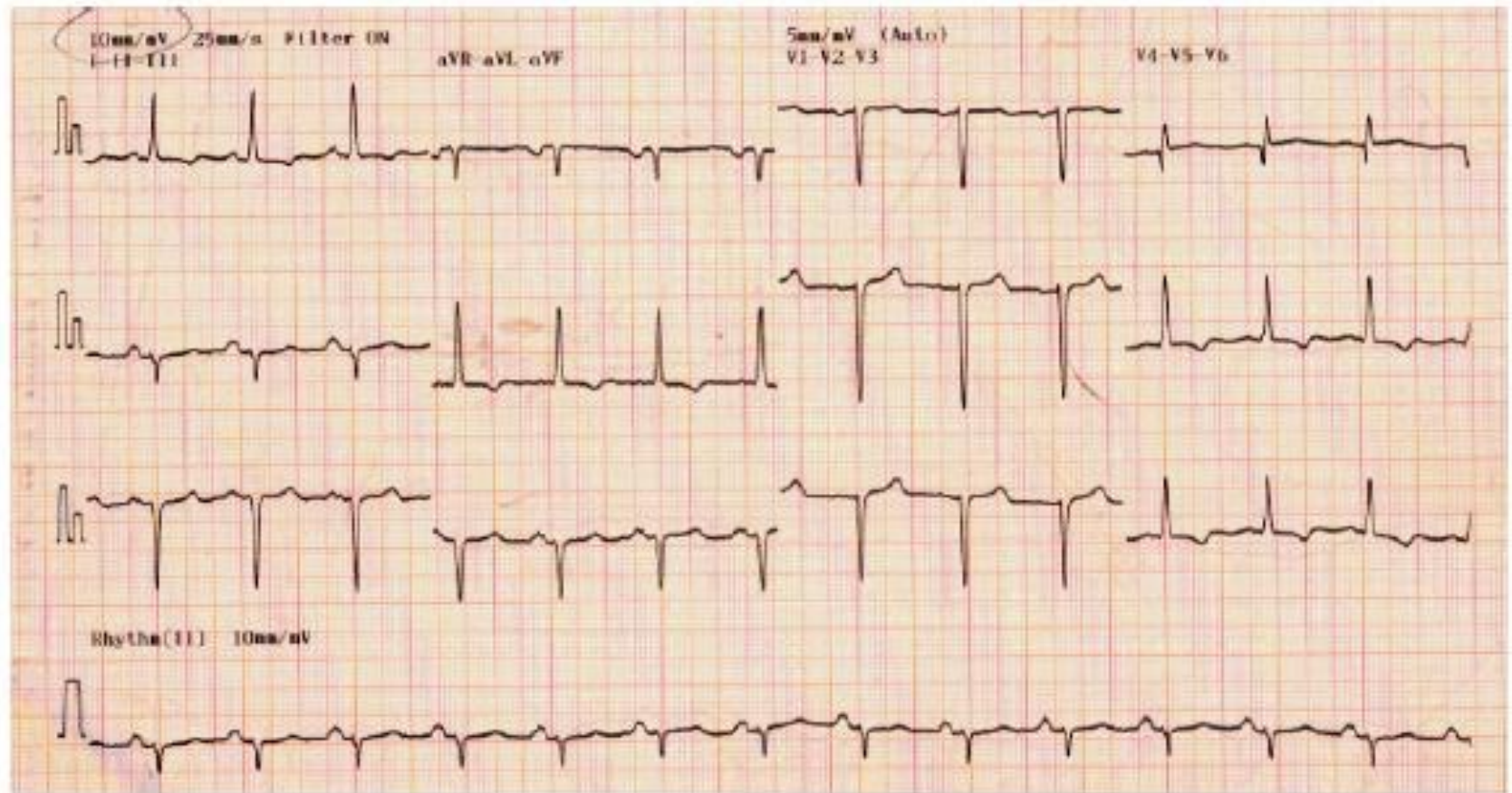
Herbal medicine: Chuanwu (川烏)

Fuzi (附子)

Aconite poisoning



ECG showed reversion to sinus rhythm 2 hrs after admission



Electrocardiogram showed reversion to sinus rhythm.

- The wild plant (especially the roots and root tubers) is extremely toxic. Severe aconite poisoning can occur after accidental ingestion of the wild plant or consumption of an herbal decoction made from aconite roots. In traditional Chinese medicine, aconite roots are used only after processing to reduce the toxic alkaloid content. Soaking and boiling during processing or decoction preparation will hydrolyze aconite alkaloids into less toxic and non-toxic derivatives.
- Aconite roots contain cardiotoxins and neurotoxins. Patients present predominantly with neurological, cardiovascular, and gastrointestinal features.
- Management is supportive; the early use of cardiopulmonary bypass is recommended if ventricular arrhythmias and cardiogenic shock are refractory to first-line treatment.



Hidden Aconite Poisoning: a life-threatening presentation



Case

A 36-year-old woman was admitted for generalized weakness, vomiting, perioral and limb numbness 2 hours after consumption of a dose of Chinese herbal medicine (CHM). She was hypotensive upon presentation (BP 75/30) with ECG showing junctional rhythm with ventricular bigeminy. ICU admission was required. The clinical features are compatible with aconite poisoning. While no aconite herb has been prescribed, aconitum alkaloids were detected in her urine and herbal broth sample. Subsequently, one of the herbs dispensed to her – *Ligusticum sinensis* (藁本) – was found to be contaminated by aconite herb. She recovered fully and has been discharged.



Message

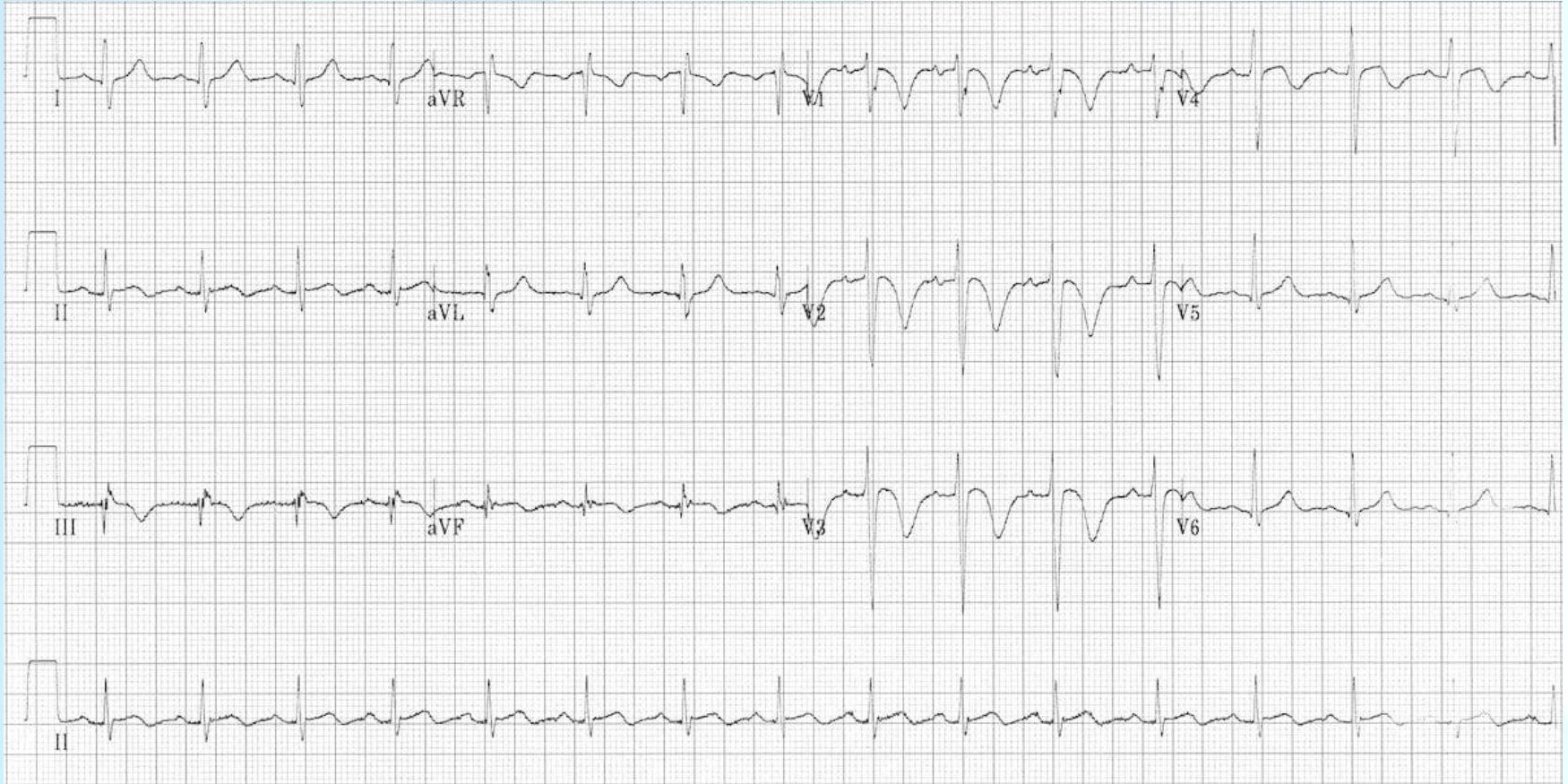
Aconite herbs like *Radix Aconiti* (川烏), *Radix Aconiti Kusnezoffii* (草烏) and *Radix Aconiti Lateralis* (附子) are the most common causes of life-threatening herbal poisoning. Typical clinical features include gastrointestinal upset, perioral and limb numbness, weakness, bradycardia with different levels of AV blockage, hypotension and ventricular arrhythmia. More than 60 aconite poisoning cases have been confirmed by the HA Toxicology Services in the past 6 years; in 1/3 of them, the ingestion of aconite herb was unintentional – the herb was NOT prescribed. The source of the error was often undetermined; possibilities include contamination, misidentification and dispensing error. This form of aconite poisoning, as in this present case, is particularly dangerous. [Reference: HK Med J. 2006;12:456–9].

Recommended treatments include fluid replacement, atropine for bradycardia, inotropic support for hypotension, amiodarone for ventricular arrhythmia and magnesium if amiodarone is contraindicated. The HA Toxicology Services can help if similar case is encountered.

Case 6

Case 6

M/75 OAHR, IHD and old CVA with poor mobility. Admitted due to sudden onset of SOB. SpO₂ ~90% (50%O₂), BP ~100/60. What is the diagnosis?



- A. Acute Pulmonary Embolism
- B. Acute Myocardial Infarction
- C. Acute Pericarditis
- D. Acute SAH



Answer : A

M/75 OAHR, IHD and old CVA with poor mobility. Admitted due to sudden onset of SOB. SpO₂ ~90% (50%O₂), BP ~100/60. What is the diagnosis?

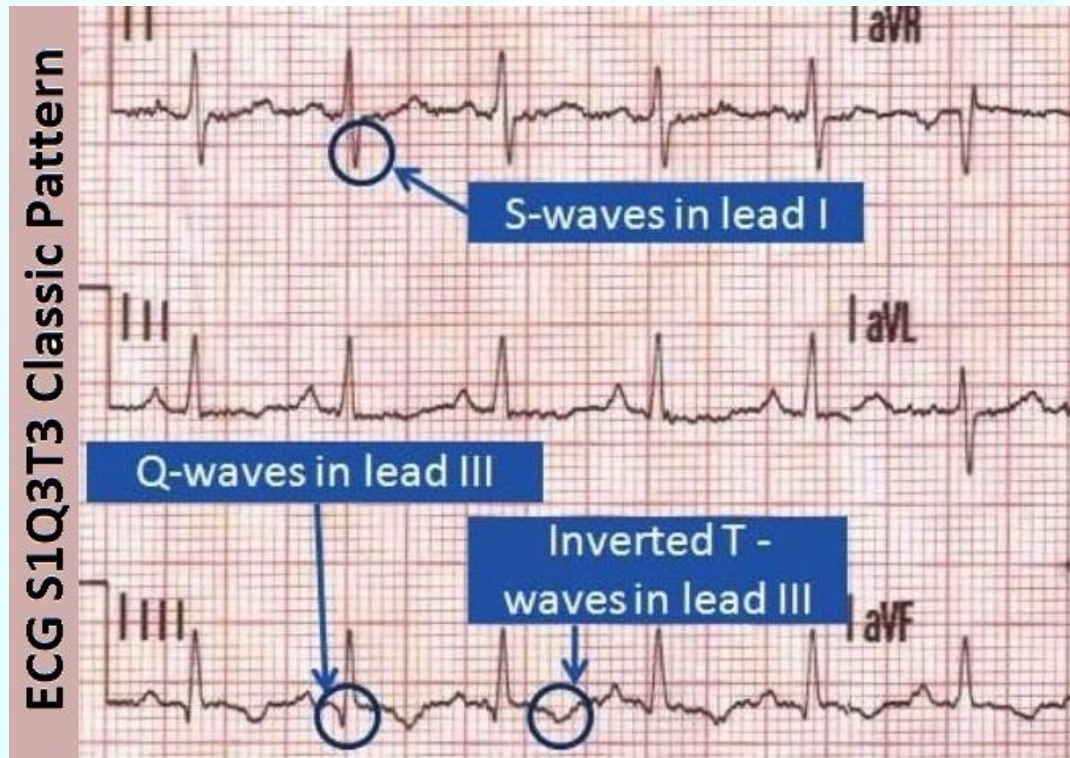


- A. Acute Pulmonary Embolism
- B. Acute Myocardial Infarction
- C. Acute Pericarditis
- D. Acute SAH

Acute Pulmonary Embolism (S_I Q_{III} T_{III})

CT thorax- bilateral pulmonary embolism
in proximal R and L pulmonary arteries

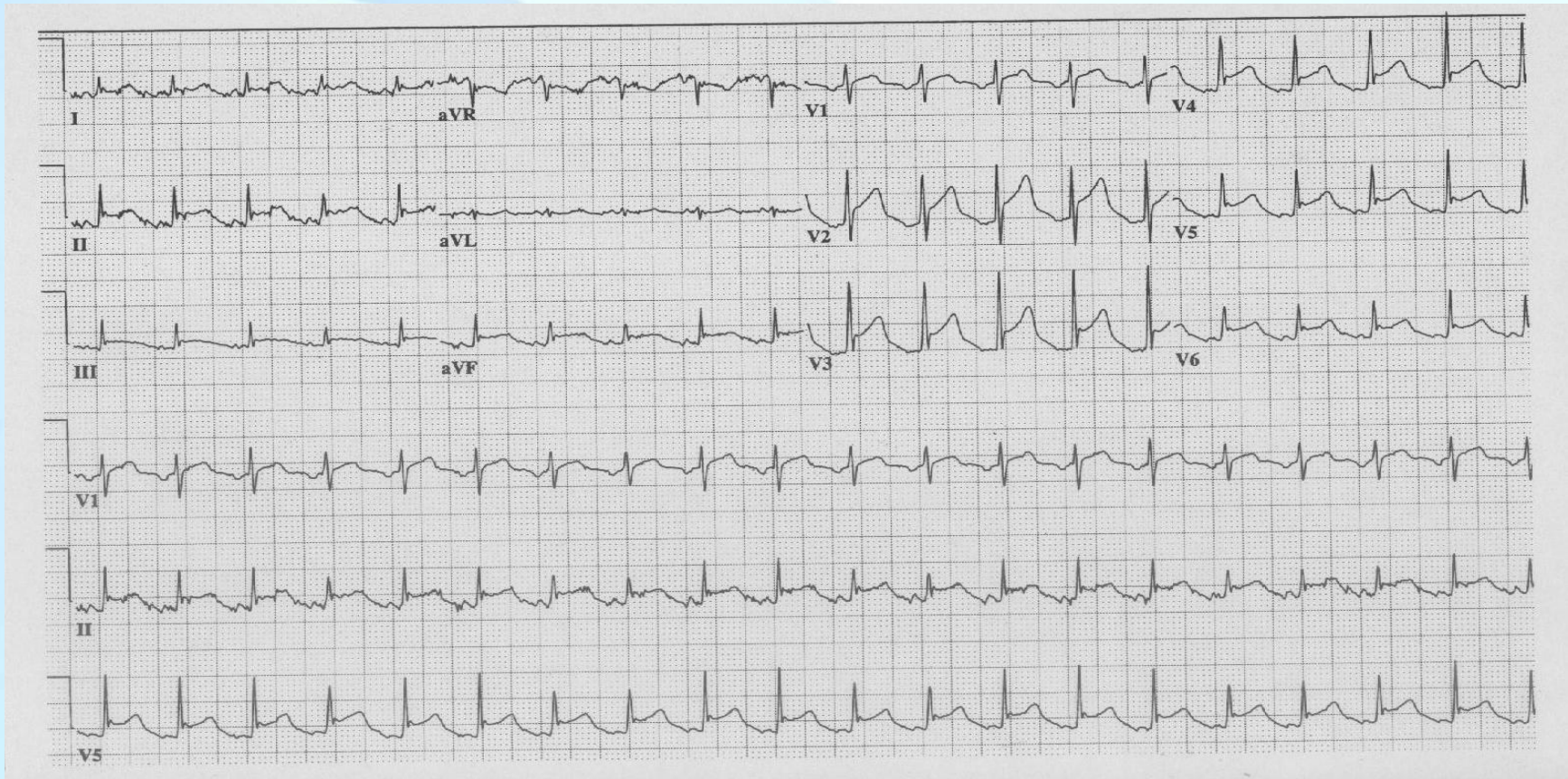
Doppler USG- DVT of right leg up to
femoral vein



Case 7

Case 7

M/40, smoker. Admitted due to acute onset of chest pain for 2 hrs.
What is the diagnosis?

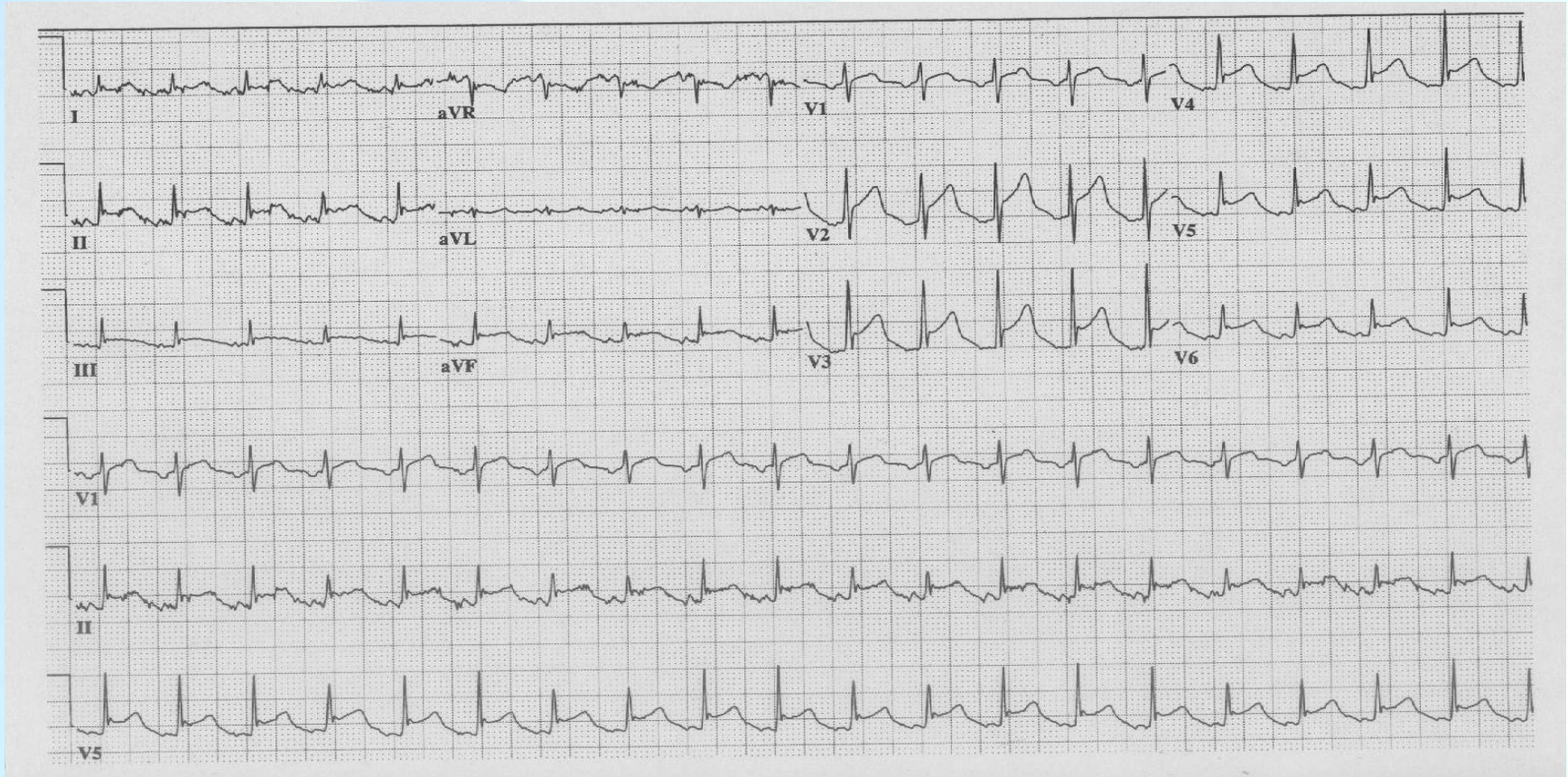


- A. Acute Pulmonary Embolism
- B. Acute Myocardial Infarction
- C. Acute Pericarditis
- D. Acute SAH



Answer: C

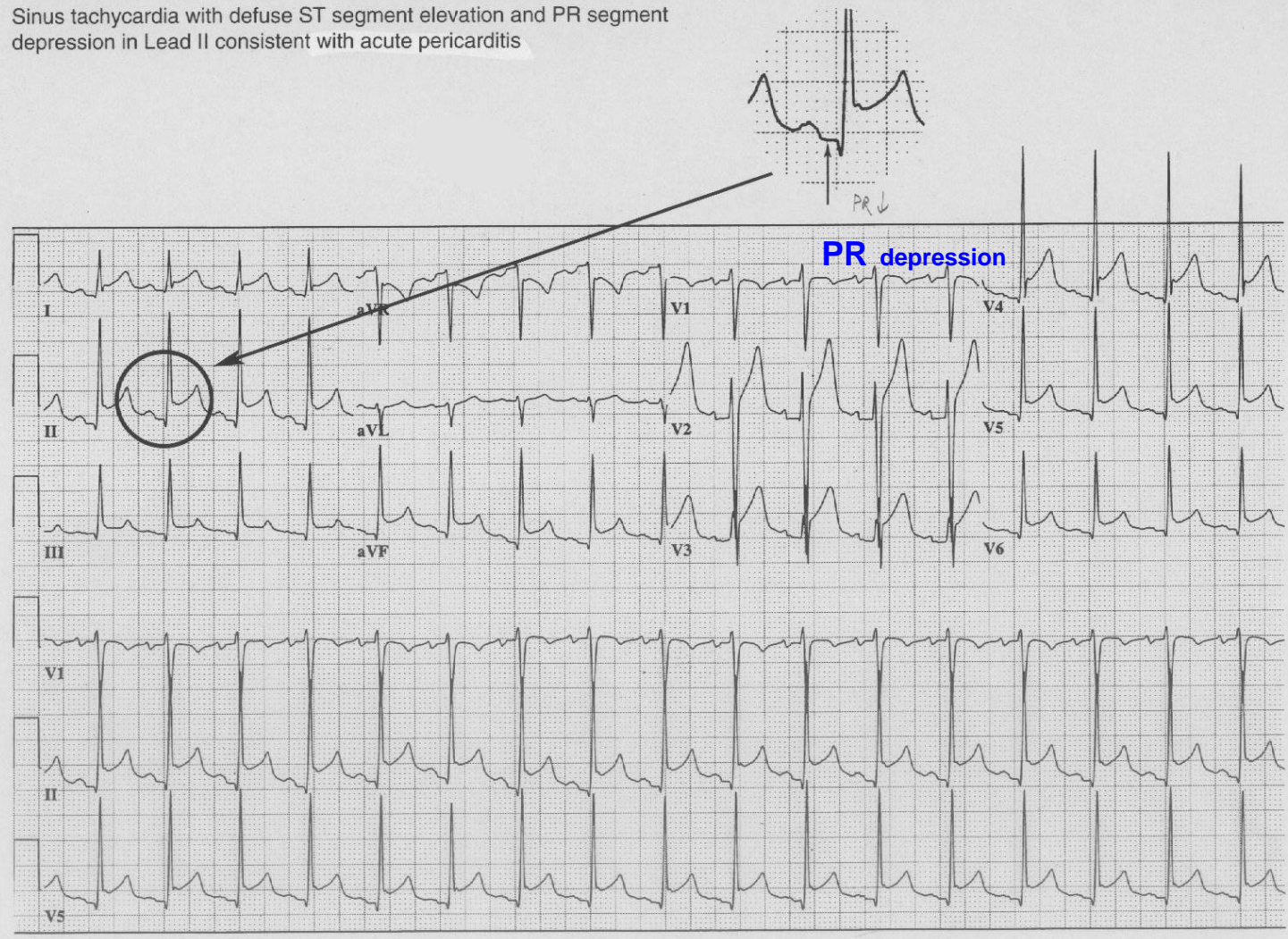
M/40, smoker. Admitted due to acute onset of chest pain for 2 hrs.
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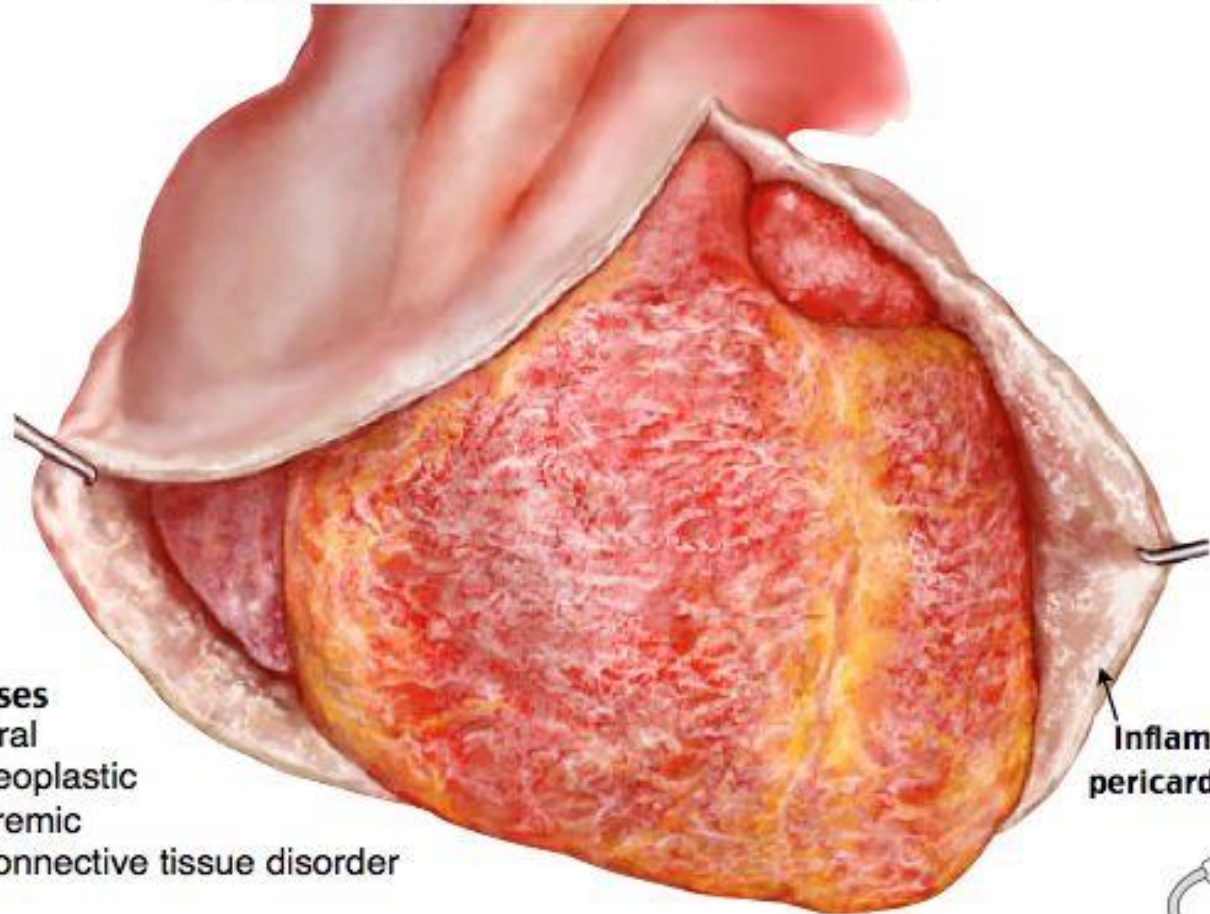
- A. Acute Pulmonary Embolism
- B. Acute Myocardial Infarction
- C. Acute Pericarditis
- D. Acute SAH

Rx: pain relief eg. NSAID

Sinus tachycardia with diffuse ST segment elevation and PR segment depression in Lead II consistent with acute pericarditis

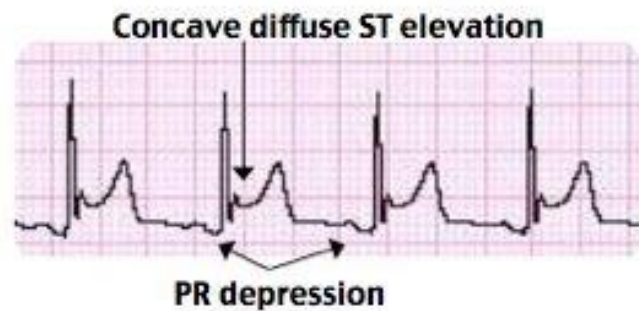


Acute Pericarditis



Causes

- Viral
- Neoplastic
- Uremic
- Connective tissue disorder



Acknowledgement

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THANK YOU!