INVESTIGATIONS IN CARDIOLOGY

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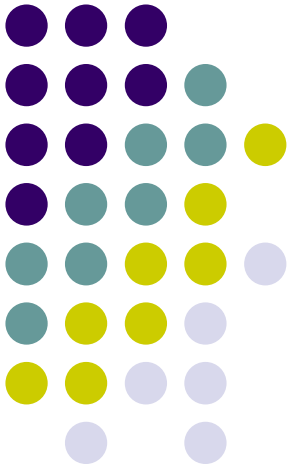
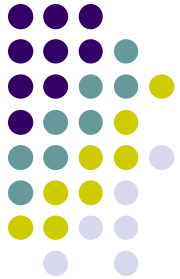


INTRODUCTION

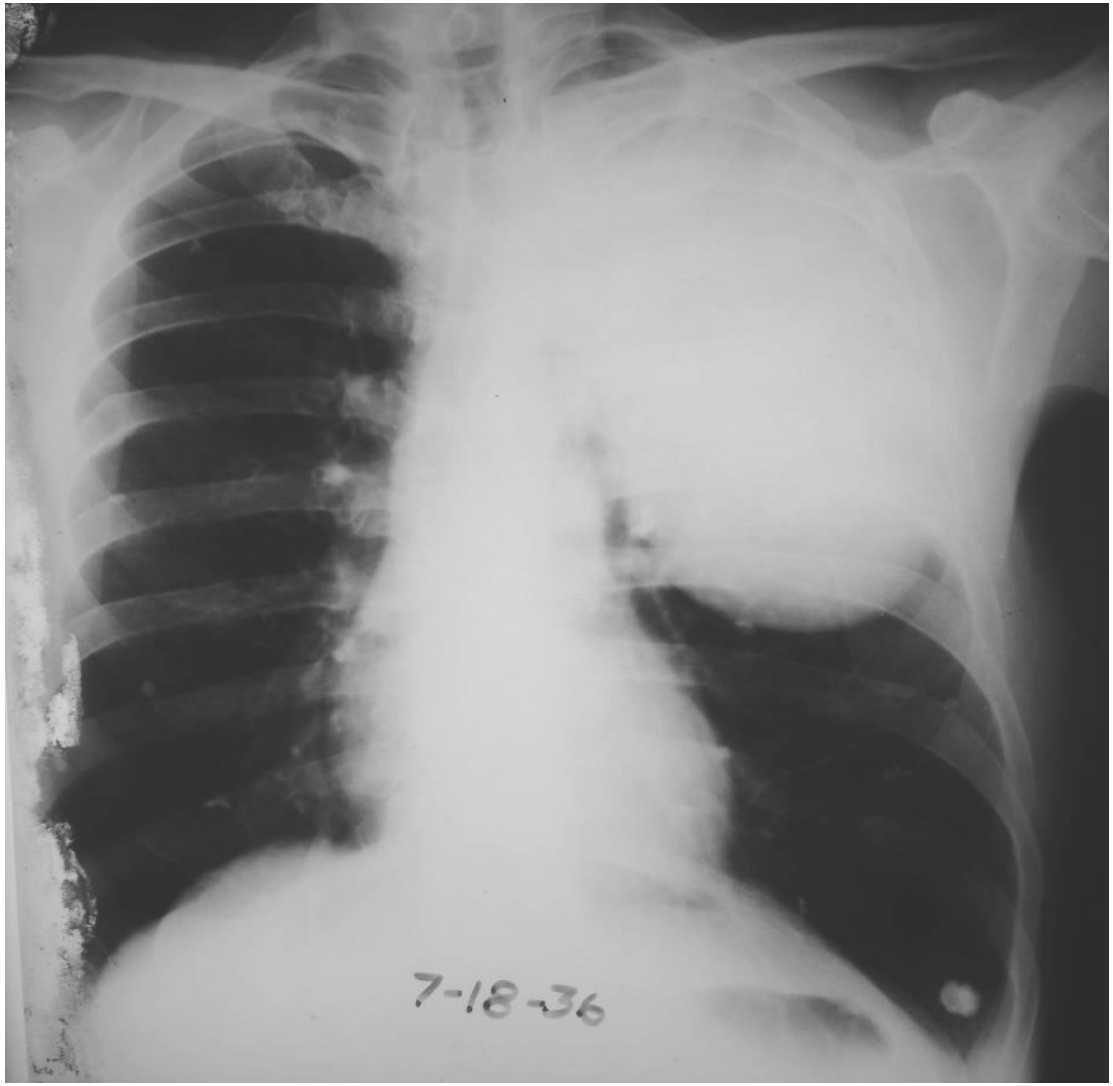


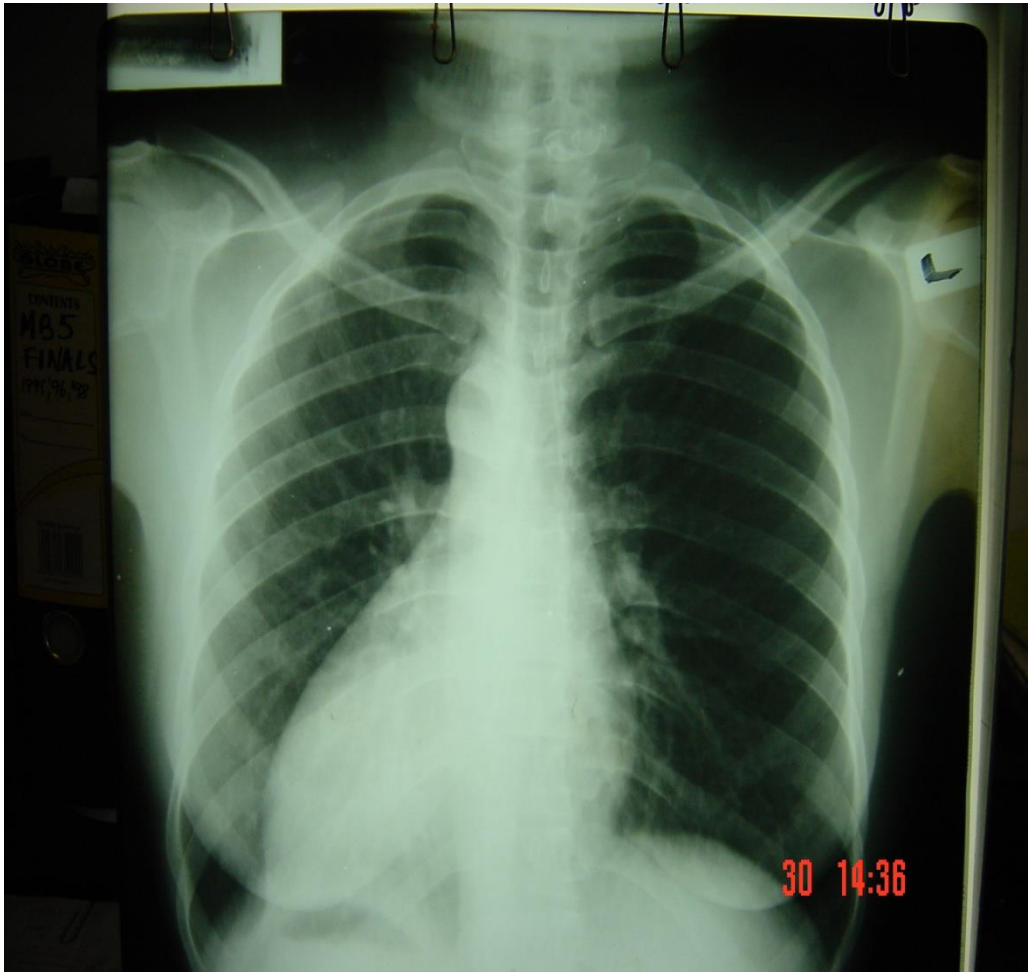
- Ix serve to confirm diagnosis
- Do not replace clinical skills
- Must be interpreted within the clinical context
- Include CXR, ECG (resting and stress), ECHO, cardiac catheterization, coronary angiogram, myocardial perfusion scans MRI, CT scan, LAB tests
- Always treat the patient not the result

CXR

- Cardiac silhouette gives important information on size and state of heart
  - Should occupy less than ½ of hemi thorax
  - Know the chambers forming the borders
    - LT – LV
    - RT- RA
    - LT UPPER BORDER – LA/ pulmonary bay, aortic knuckle
  - Lung fields – help differentiate cardiac and respiratory causes of dyspnoea e.g, pulmonary oedema / pl. effusion
  - Ribs – e.g, notching in COA

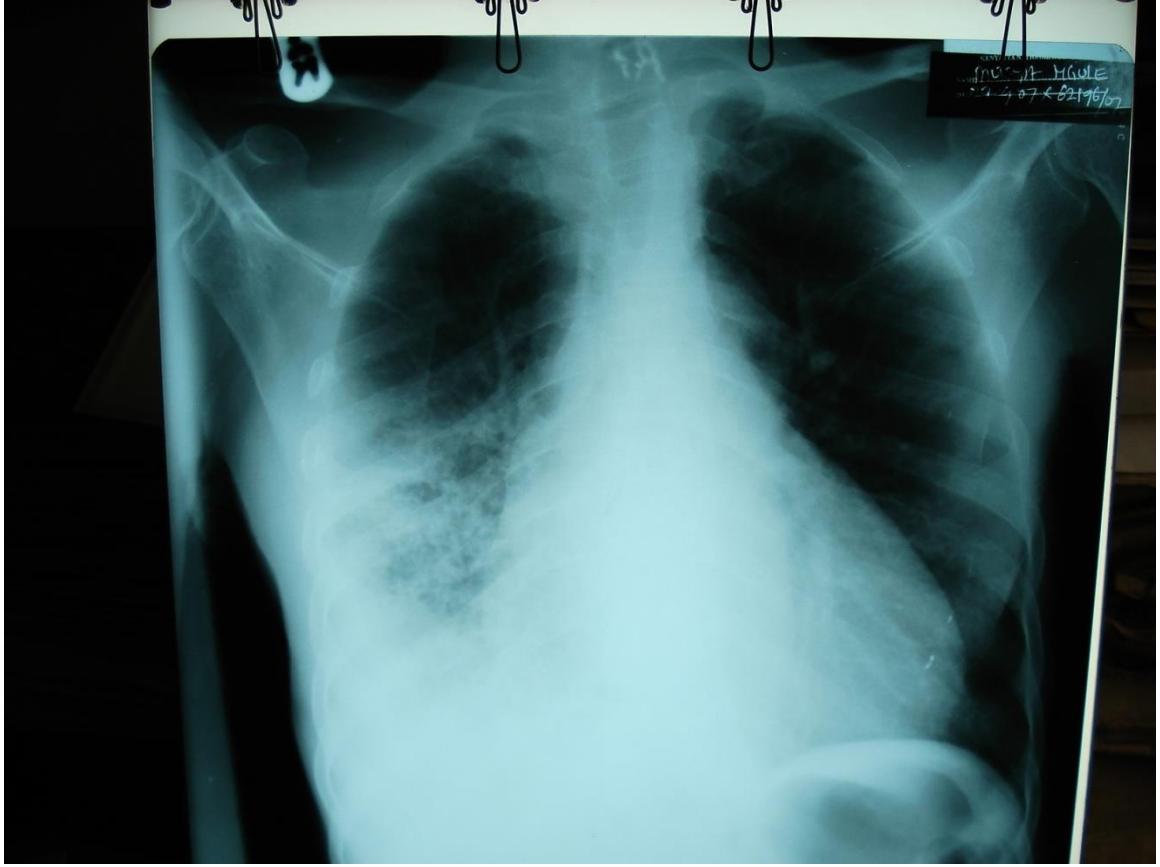
CXR of a Pastoralist from Lokichogio





- **There is Dextrocardia**

- **The patient has Situs inversus; see gastric bubble**



This CXR shows

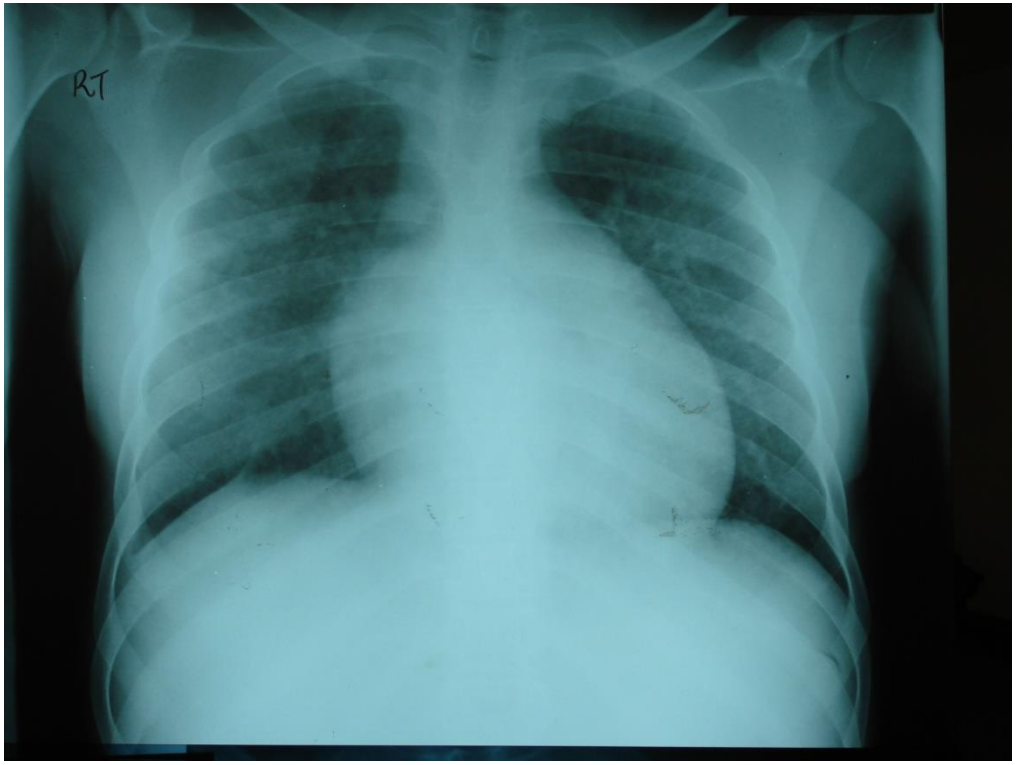
- Cardiomegally
- Right sided consolidation and effusion

MASSIVE CARDIOMEGALLY

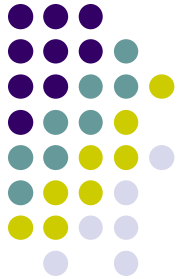
Differential diagnoses include:

Dilated cardiomyopathy

Pericardial effusion

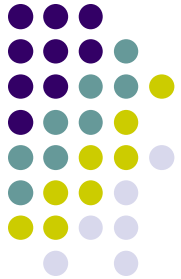


ELECTROCARDIOGRAM



- Very important
- SAN, atrial tissue, AVN, His purkinje system, Lt/Rt bundles, ventricular tissue
- Aids in diagnosis of
  - Ischemic heart disease especially ACS
  - Chamber enlargement
  - Arrhythmias
  - Electrolyte imbalance
  - Pericardial disease - effusion
  - Pulmonary thromboembolism

Arrhythmias
Diagnosis entirely based on ECG

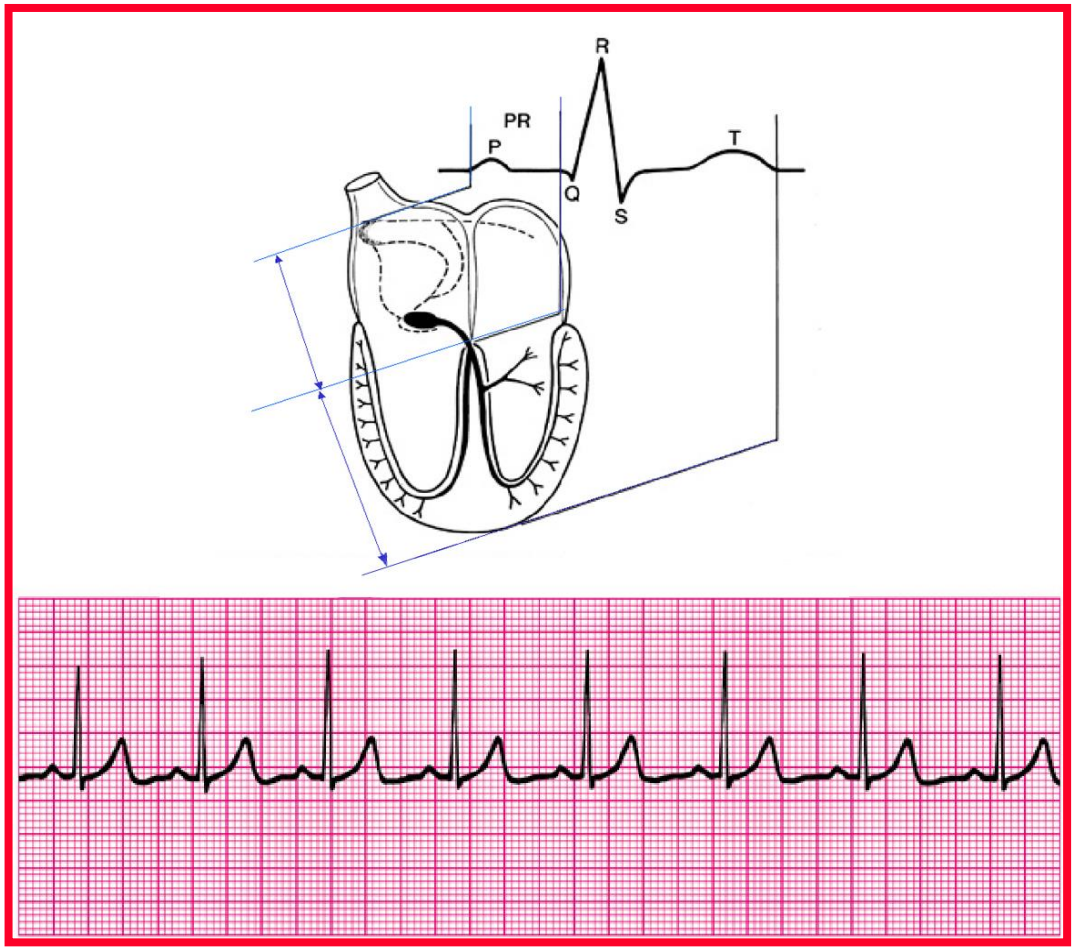


- Can arise from any part of the conduction system
- Classified depending on origin
  - Supraventricular
  - Ventricular
- Causes include
  - **Increased automaticity;** ischaemia, chamber enlargement, heightened SNS activity
  - **Conduction abnormalities**; degenerative disease/ischaemia at the AVN- **Heart block**
  - **Reentry** e.g. WPW syndrome
- Sinus nodal dysfunction

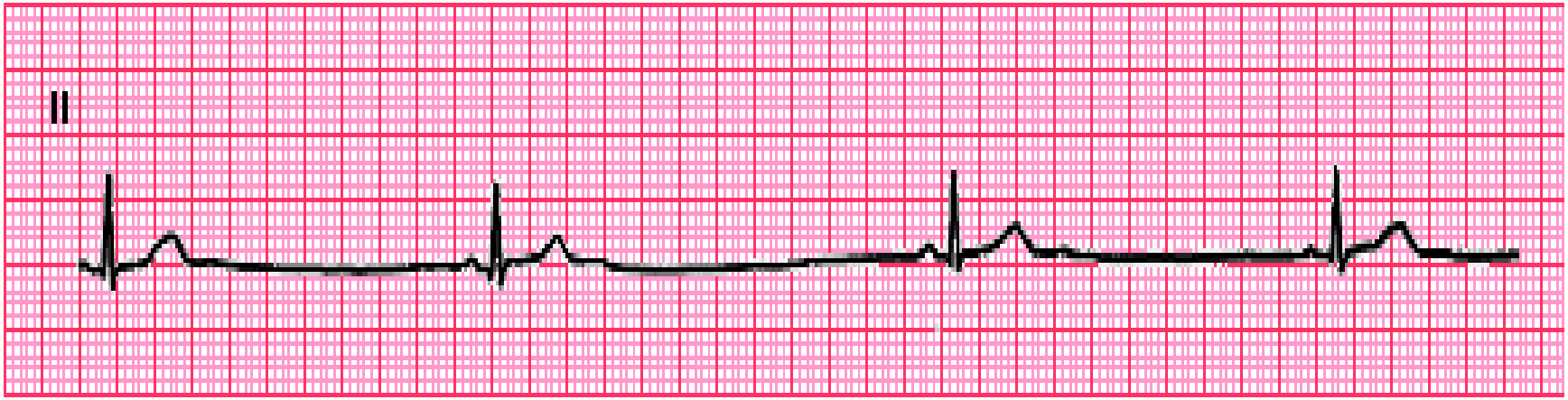
Analyzing ECG

- Key questions
  - Are QRS complexes present?
  - Are P waves present?
  - How is the P wave related to the QRS complex?
  - Is every P wave followed by a QRS complex with a normal P–R interval?
  - What is the R-R interval?- heart rate
  - What is the axis?

NSR

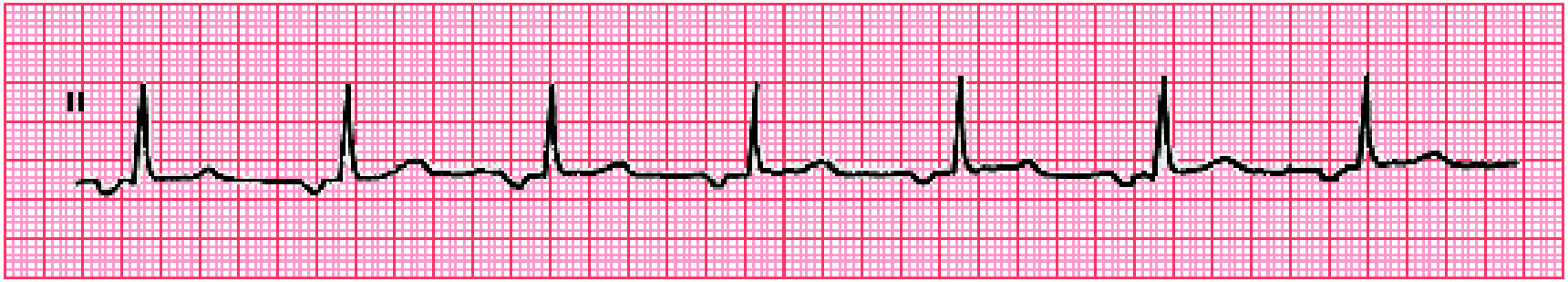


Sinus bradycardia



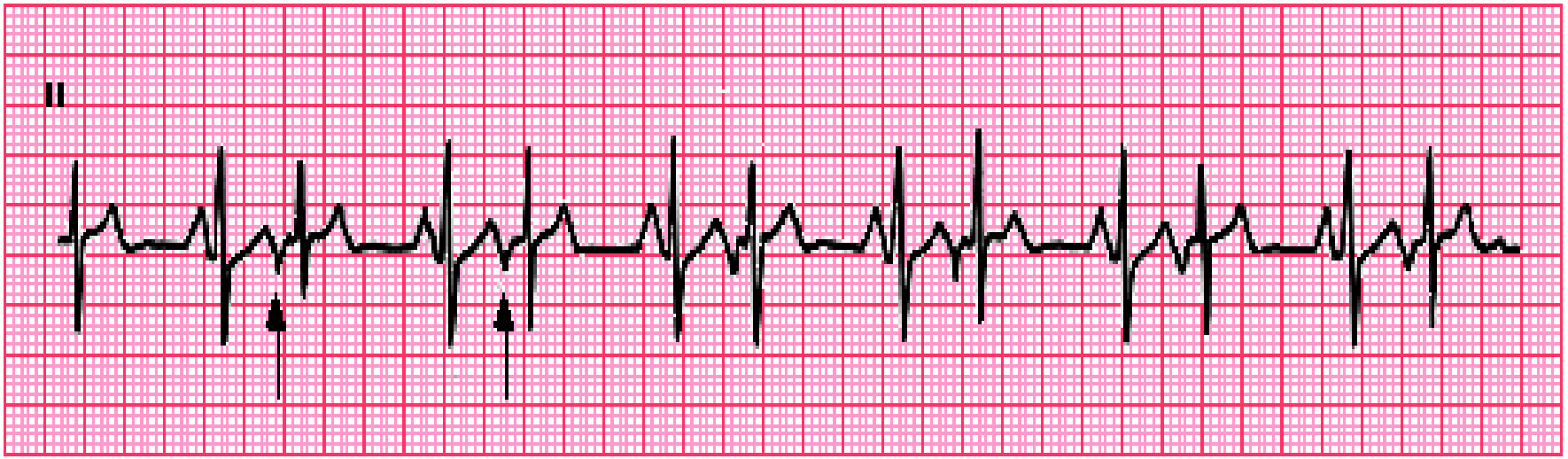
Sinus bradycardia Marked sinus bradycardia at a rate of 25 to 30 beats/min. The normal P waves (upright in lead II) and PR interval and consistent with a sinus mechanism with normal atrioventricular (AV) conduction. Courtesy of Ary Goldberger, MD.

Atrial ectopic rythm



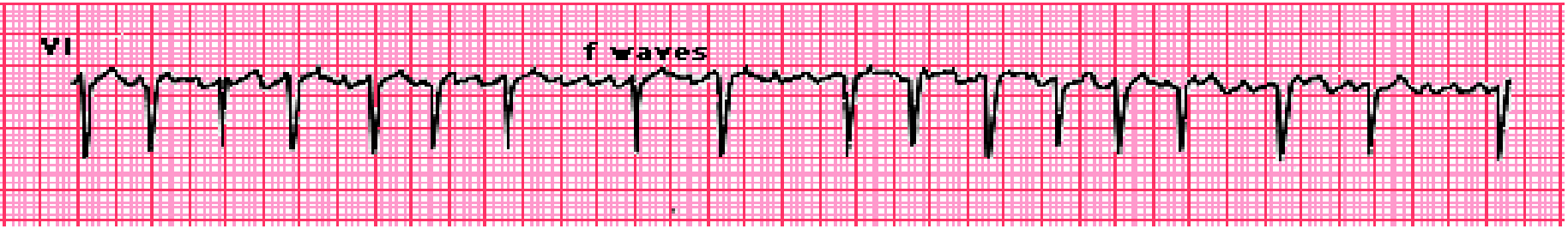
Ectopic atrial rhythm Rhythm strip in lead II showing ectopic atrial rhythm with inverted P waves which represent a low atrial focus with retrograde activation of the atrium. Courtesy of Ary L Goldberger, MD.

Atrial bigeminy



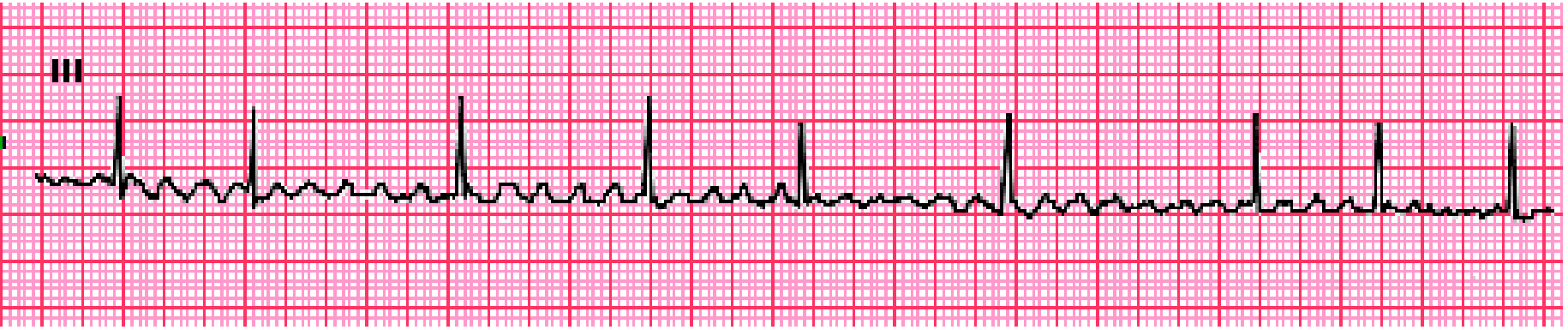
Atrial bigeminy Atrial bigeminy in which each sinus beat is followed by a premature atrial complex. The premature P waves (arrows) appear to be inverted in lead II, suggesting that they originate from a low atrial ectopic focus. Courtesy of Ary Goldberger, MD.

Atrial fibrillation

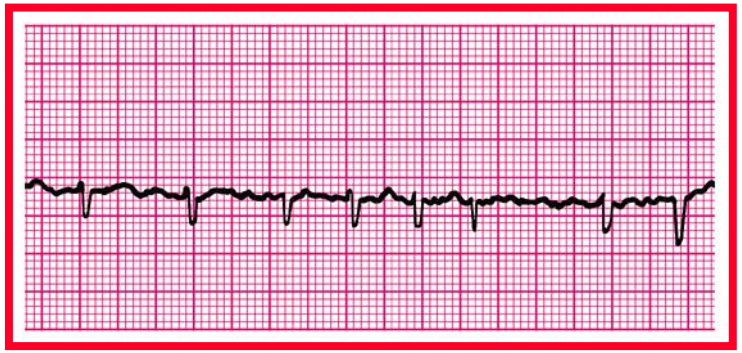


Atrial fibrillation Lead V1 showing coarse atrial fibrillation with moderate ventricular response. The two characteristic findings in AF are present: the very rapid atrial fibrillatory waves (f waves) which are variable in appearance; and the irregularly irregular ventricular response as the R-R interval between beats is unpredictable. Coarse atrial fibrillation may appear similar to atrial flutter. However, the variable height and duration of the f waves differentiate them from atrial flutter (F) waves which are identical in appearance and occur at a constant rate of about 250 to 350 beats/min. Courtesy of Ary Goldberger, MD.

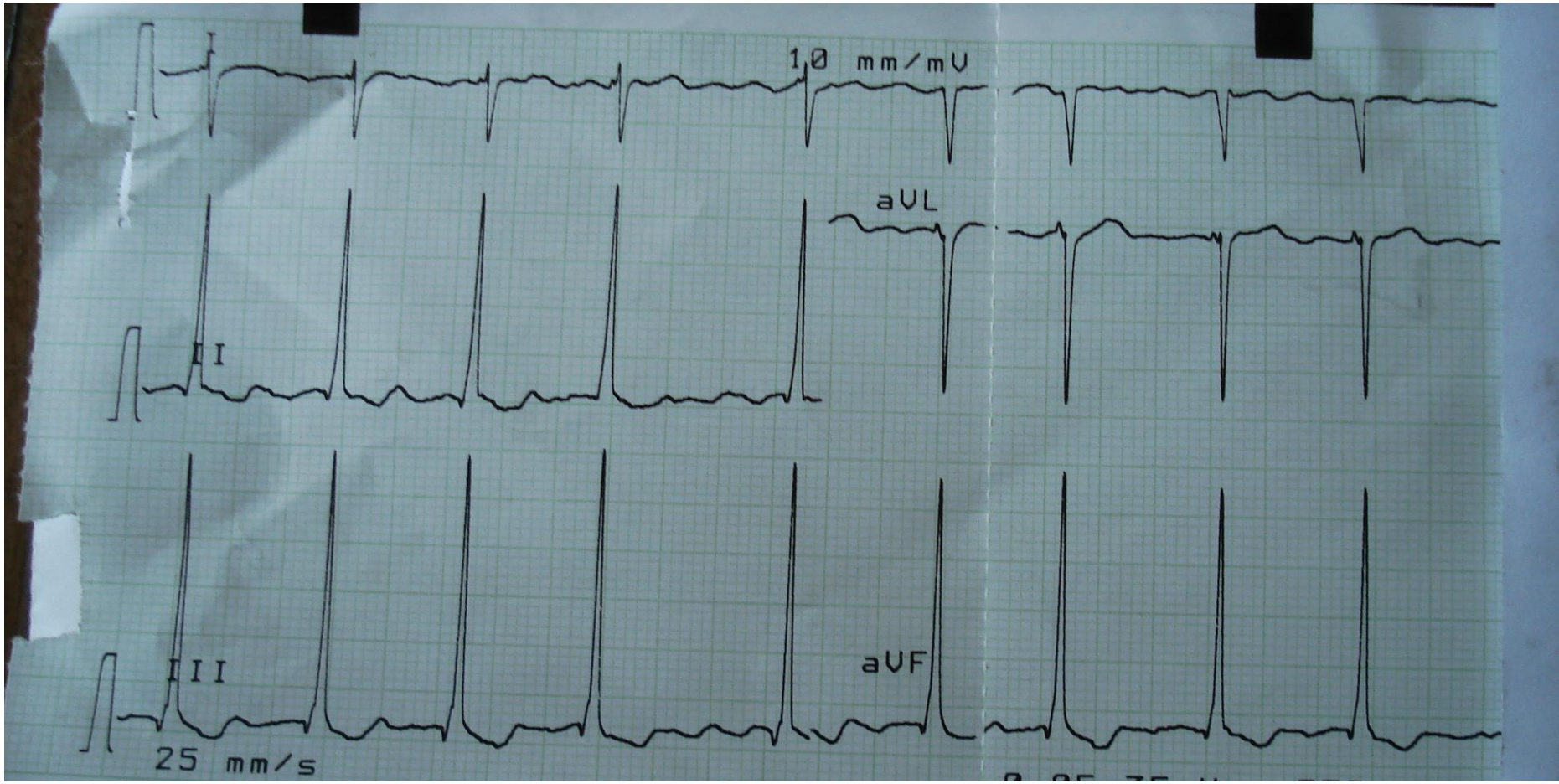
Atrial fibrillation



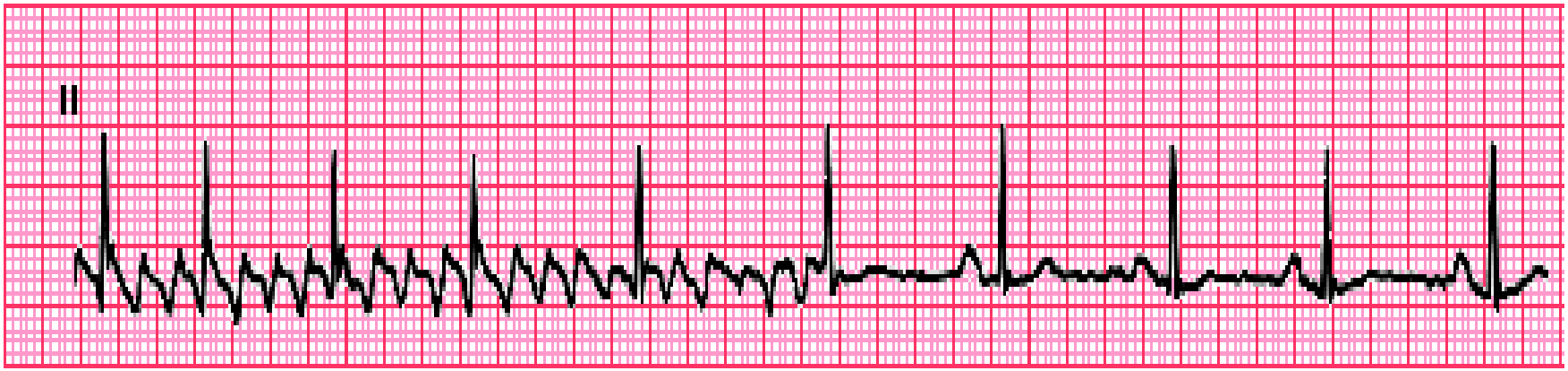
Coarse atrial fibrillation Coarse atrial fibrillatory waves are seen with an irregular ventricular response at a moderate rate. This arrhythmia may be mistaken for atrial flutter. However, the atrial waves are all identical in atrial flutter; in comparison, the rapid atrial waves vary in rate and shape from one section of the rhythm strip to the next in atrial fibrillation. Courtesy of Ary Goldberger, MD.



Atrial fibrillation

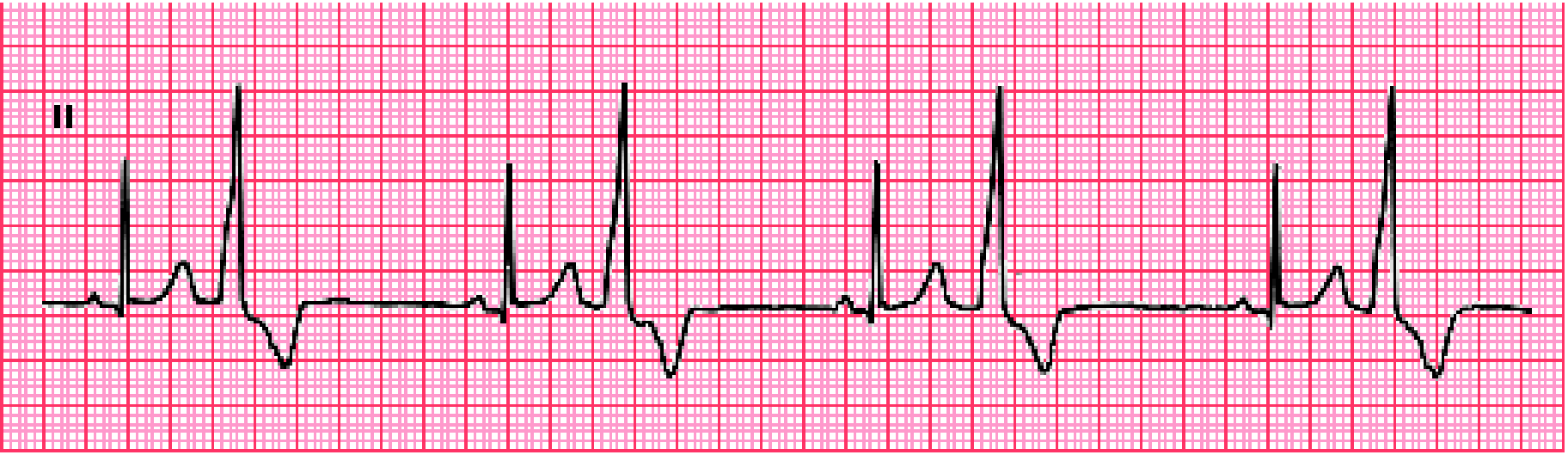


Atrial flutter



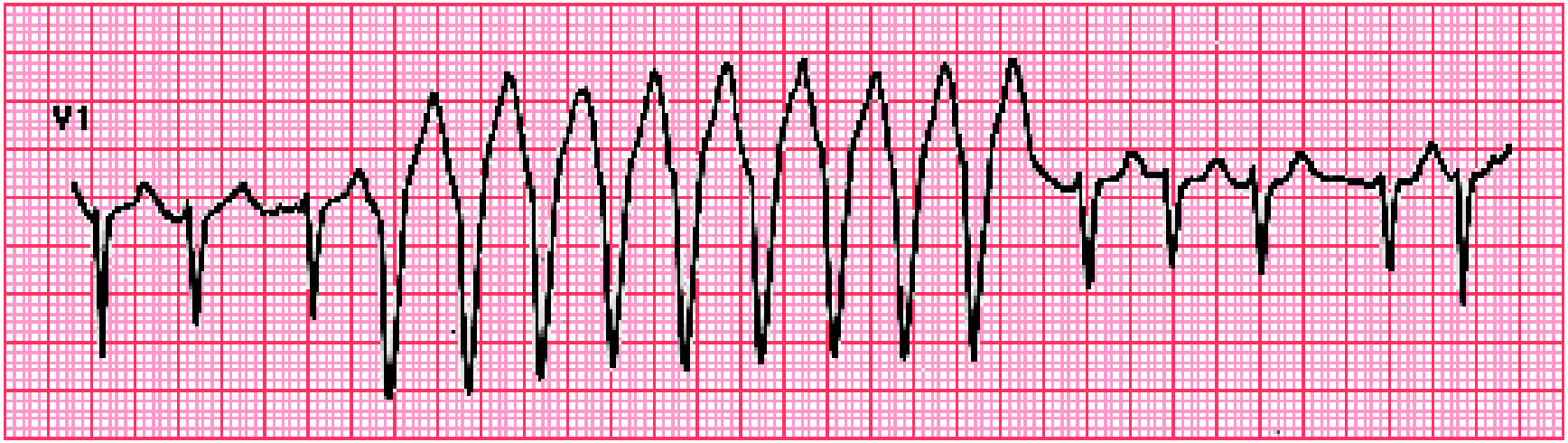
Atrial flutter to sinus rhythm Spontaneous conversion of atrial flutter (note the regular flutter waves) with variable degrees of atrioventricular block to normal sinus rhythm. Courtesy of Ary Goldberger, MD.

Ventricular bigeminy



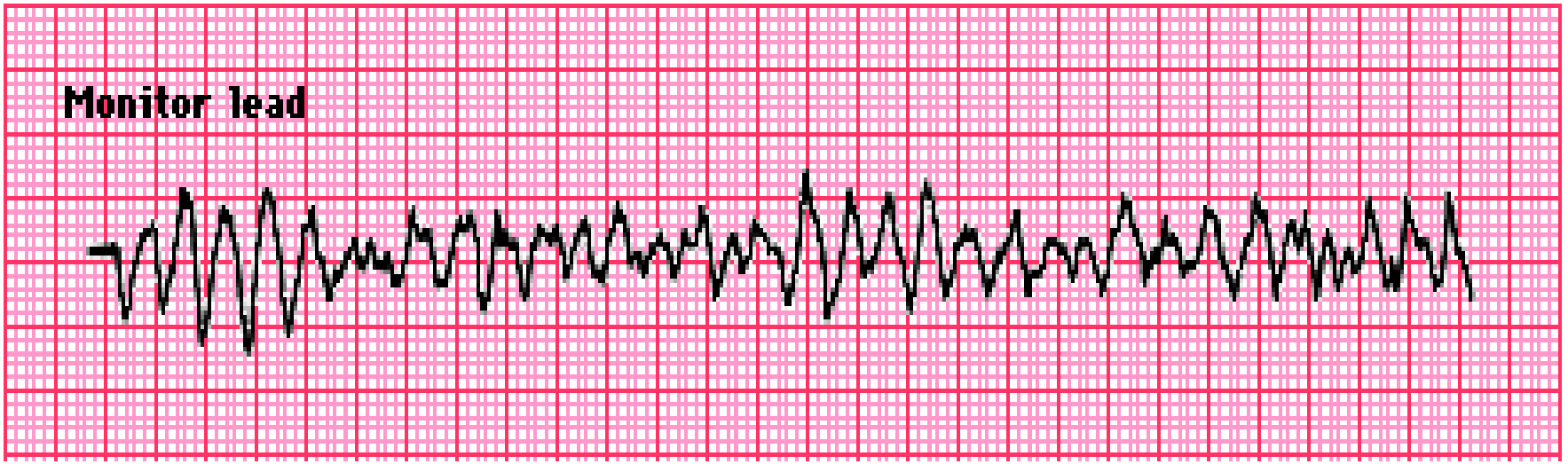
Sinus rhythm with ventricular bigeminy Sinus rhythm with ventricular bigeminy. Each sinus beat is followed by a uniform premature complex with prolonged duration and no apparent P wave; these findings are indicative of ventricular ectopic beats. Courtesy of Ary Goldberger, MD.

Ventricular tachycardia



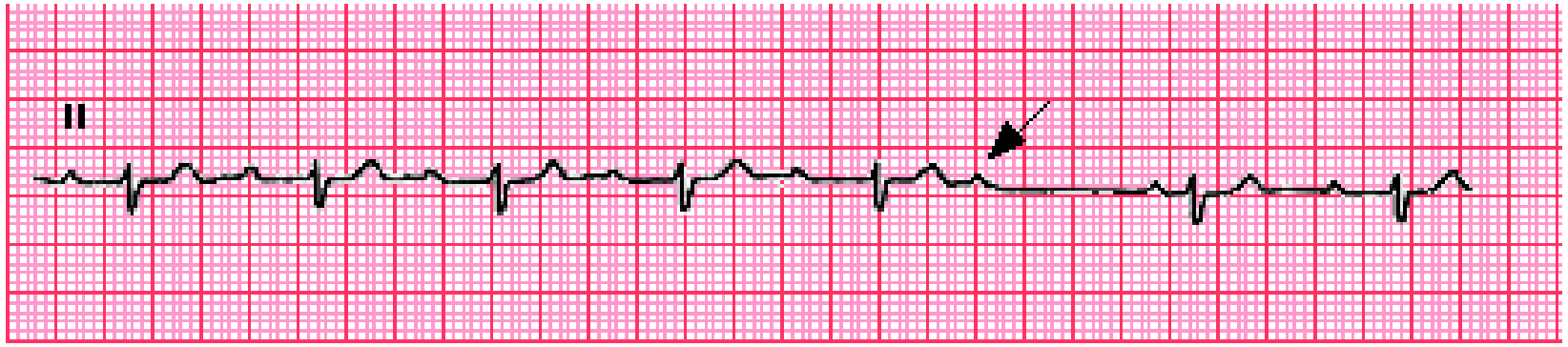
Nonsustained ventricular tachycardia Nonsustained ventricular tachycardia in a patient with underlying atrial fibrillation. The ventricular arrhythmia consists of nine beats at an approximate rate of 170 beats/min. Courtesy of Ary Goldberger, MD.

Ventricular fibrillation



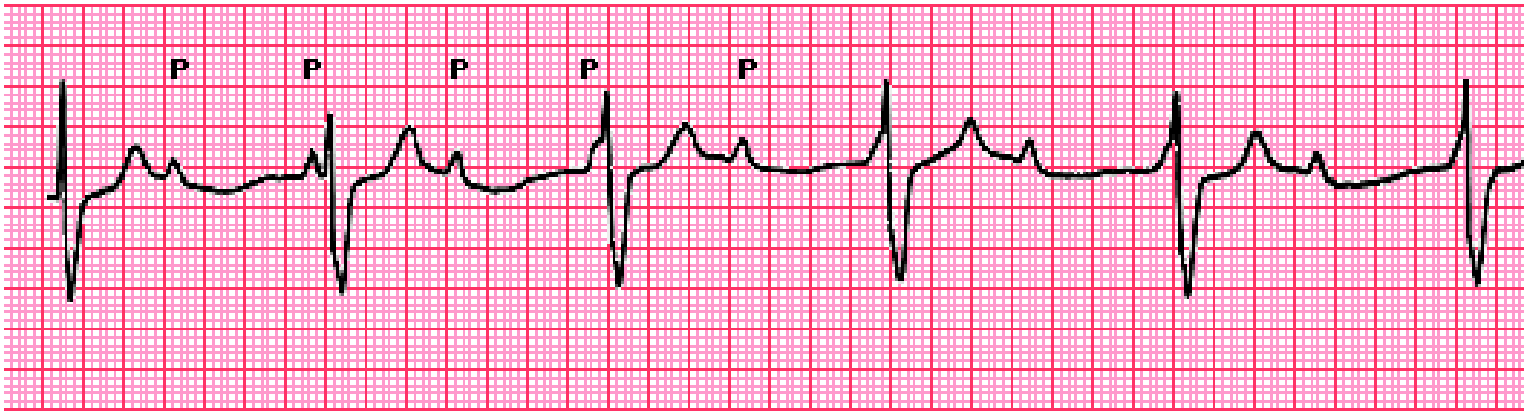
Ventricular fibrillation Extremely rapid, erratic ventricular activity due to ventricular fibrillation during cardiac arrest. Courtesy of Ary Goldberger, MD.

2nd degree heart block

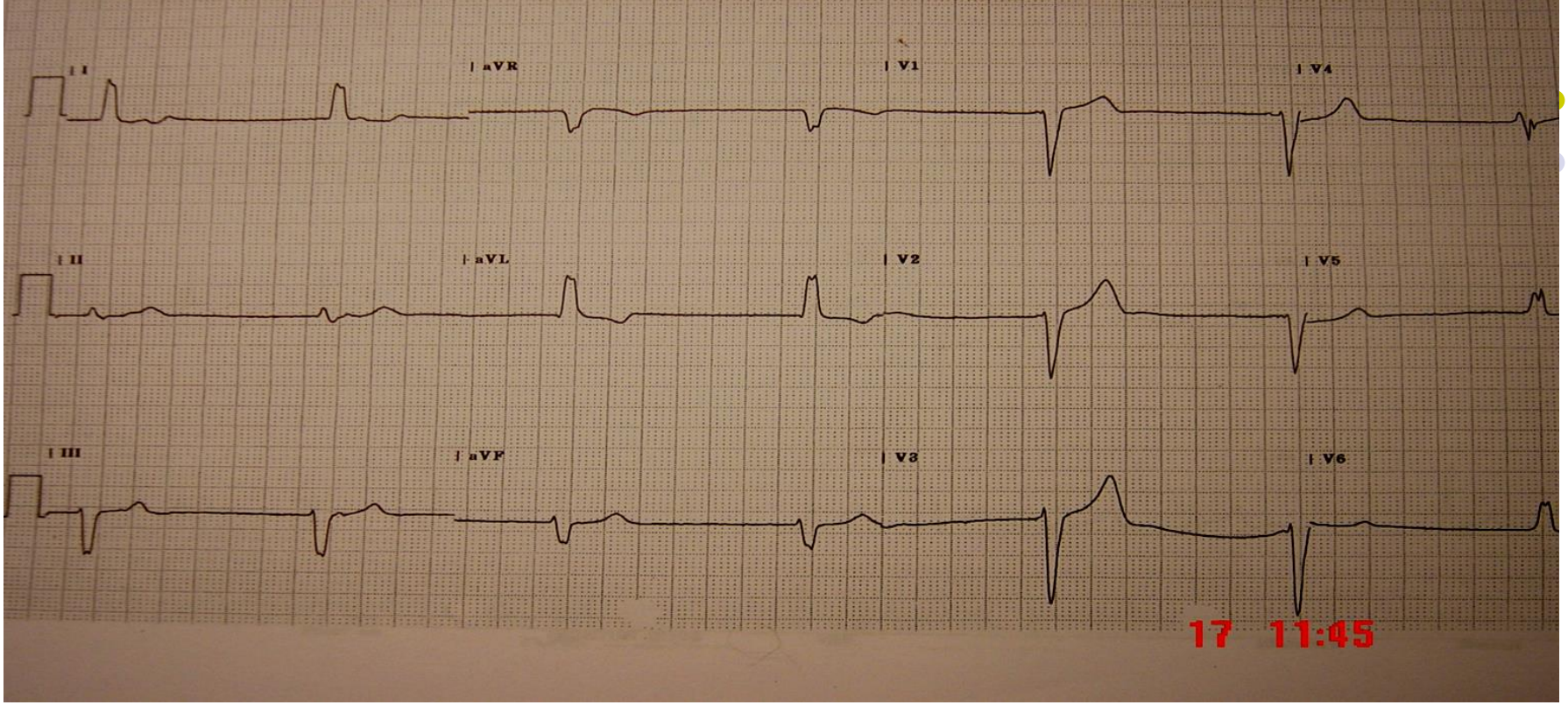


Mobitz type I (Wenckebach) AV block Sinus rhythm with Mobitz type I (Wenckebach) atrioventricular block. Note the progressive prolongation of the PR interval with the sixth P wave not being conducted (arrow). This is followed by resumption of AV conduction as the seventh P wave is conducted with a relatively short PR interval that then begins to increase with the next P wave. Courtesy of Ary Goldberger.

Complete heart block



Sinus rhythm with complete heart block Sinus rhythm with complete (third-degree) heart block. There is independent atrial (as shown by the P waves) and ventricular activity, with respective rates of 83 and 43 beats/min. The wide QRS complexes may represent a junctional escape rhythm with underlying bundle branch block or an idioventricular pacemaker. Courtesy of Ary Goldberger, MD.

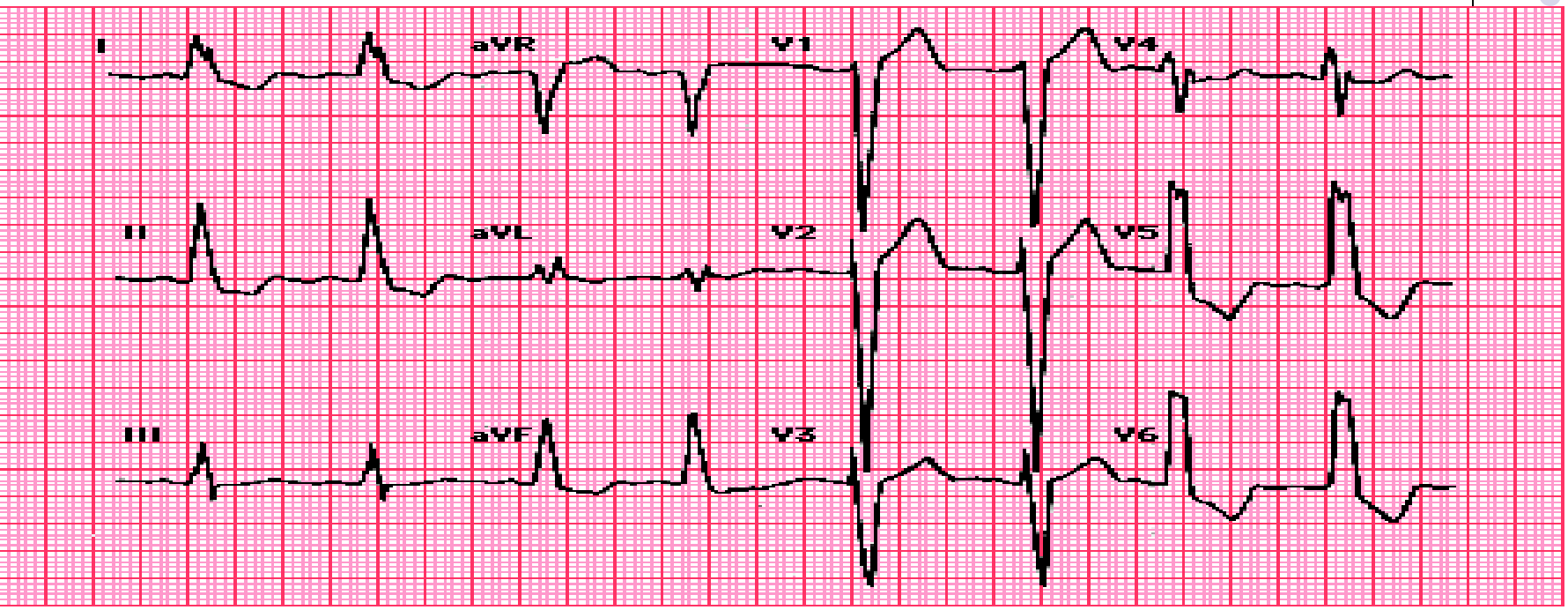


12 lead resting ECG tracing of a 37 year lady who presents with fatigue

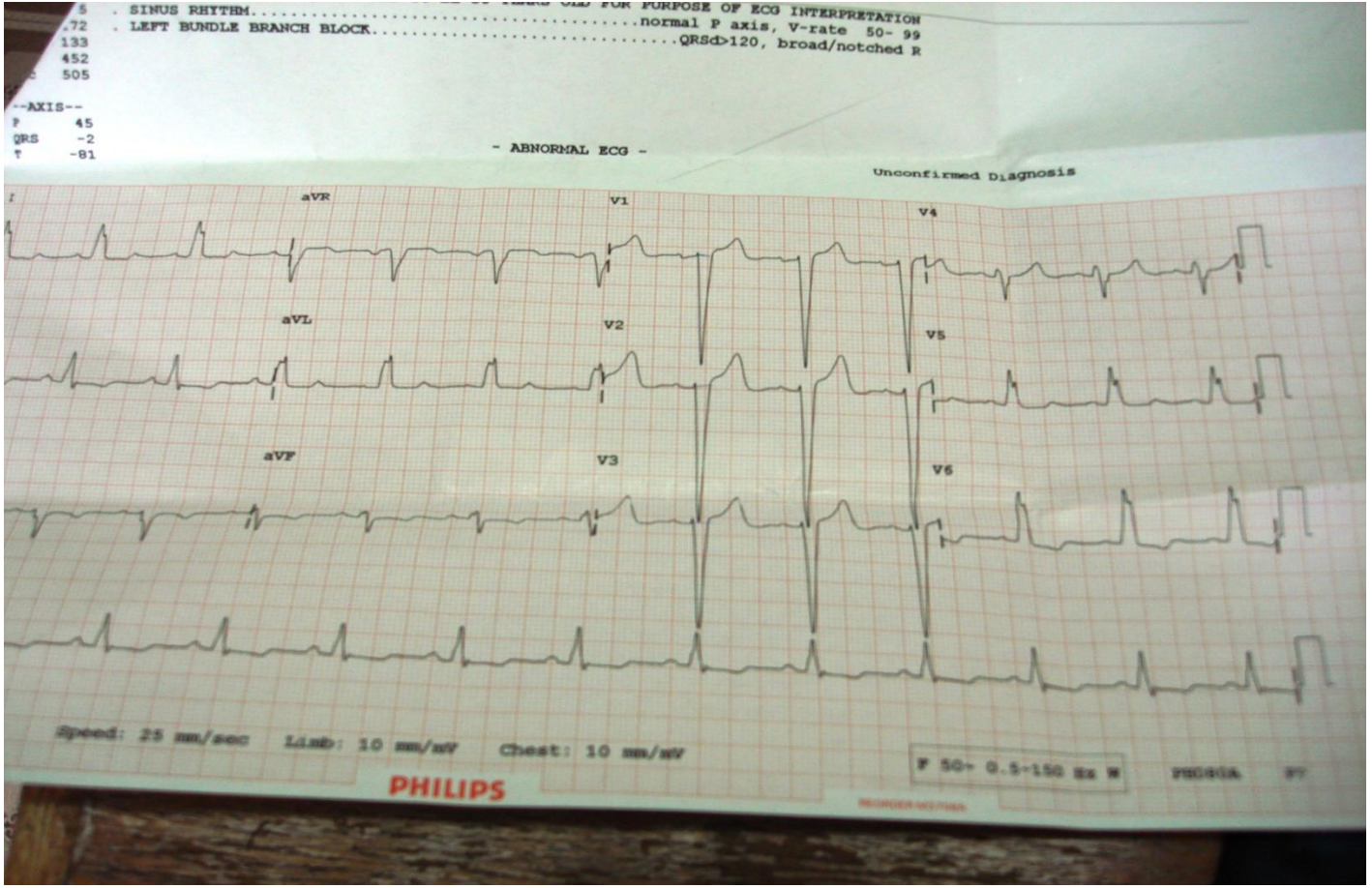
- **Complete heart block**

- **Permanent pacing is indicated**

Left bundle branch block

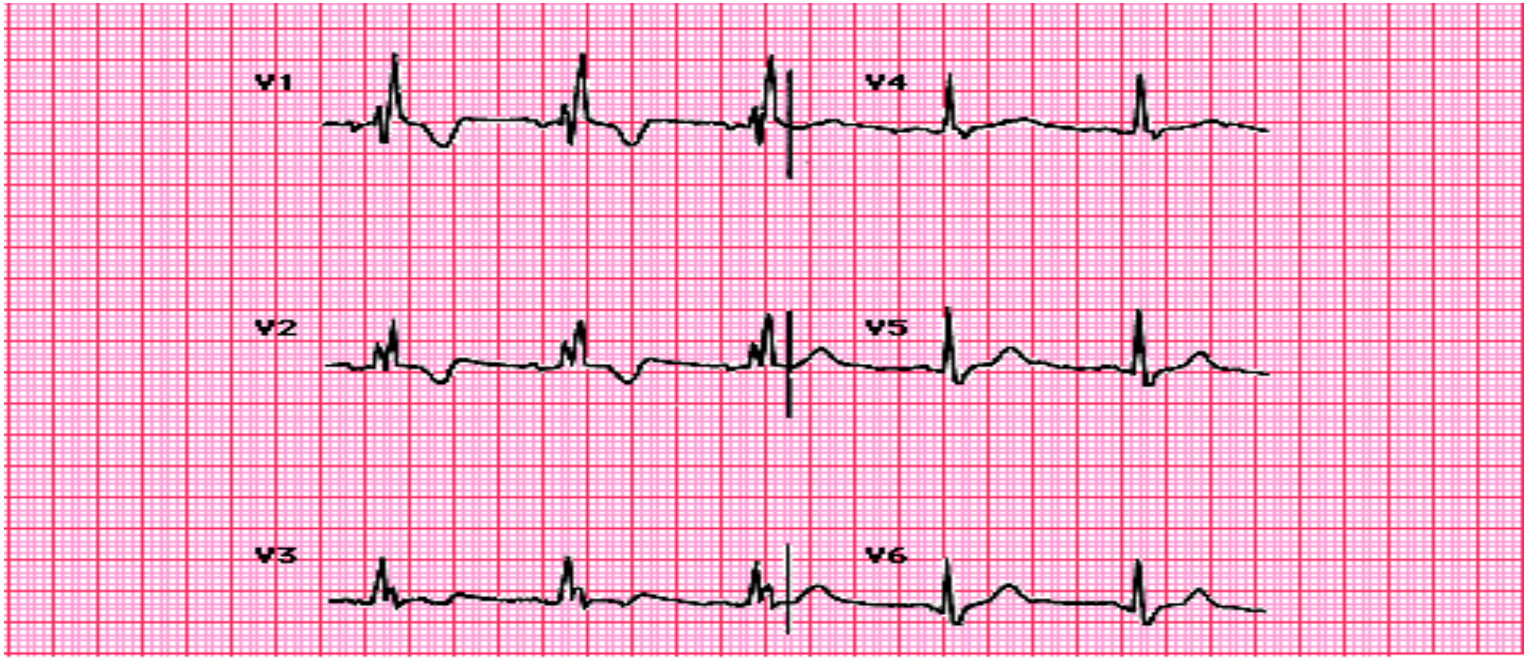


Typical left bundle branch block Electrocardiogram in typical complete LBBB. The asynchronous activation of the two ventricles increases the QRS duration (0.16 sec). The abnormal initial vector results in loss of "normal" septal forces as manifested by absence of q waves in leads I, aVL, and V6. The late activation of the left ventricle prolongation of the dominant leftward progression of the middle and terminal forces, leading to a positive and widened R wave in the lateral leads. Both the ST segment and T wave vectors are opposite in direction from the QRS, a "secondary" repolarizationa abnormality. Courtesy of Ary Goldberger, MD.

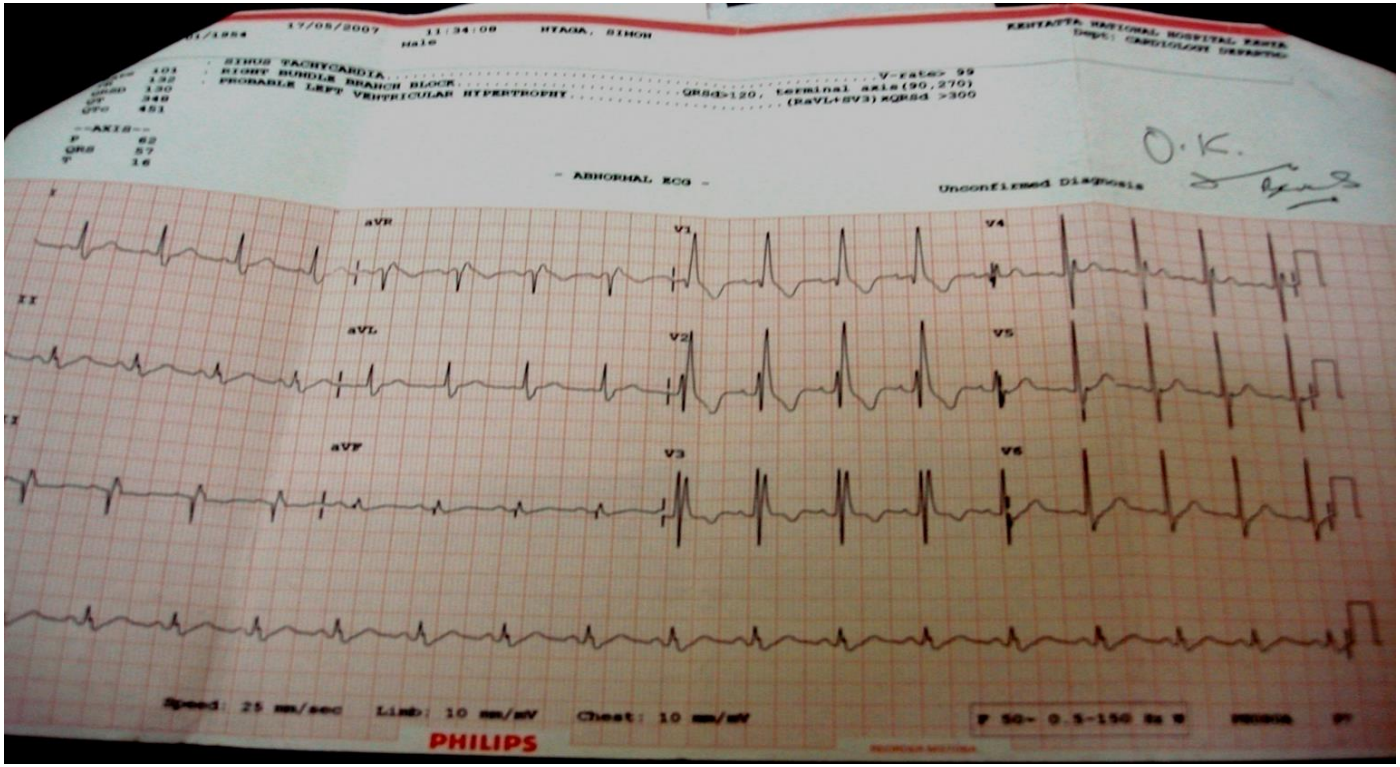


ECG of a 65 year patient with acute pulmonary edema.

Right bundle branch block

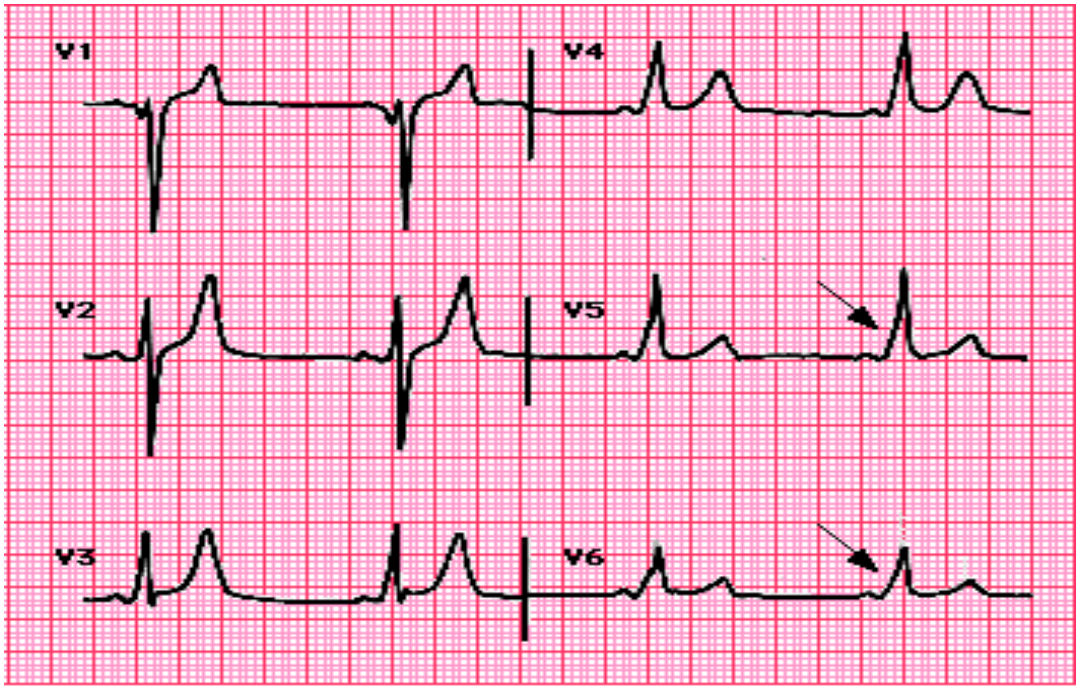


Common RBBB Electrocardiogram showing characteristic changes in the precordial leads in common RBBB. The asynchronous activation of the two ventricles increases the QRS duration (0.13 sec). The terminal forces are rightward and anterior due the delayed activation of the right ventricle, resulting in an rsR' pattern in the anterior-posterior lead V1 and a wide negative S wave in the left-right lead V6 (and, not shown, in lead I). Courtesy of Ary Goldberger, MD.

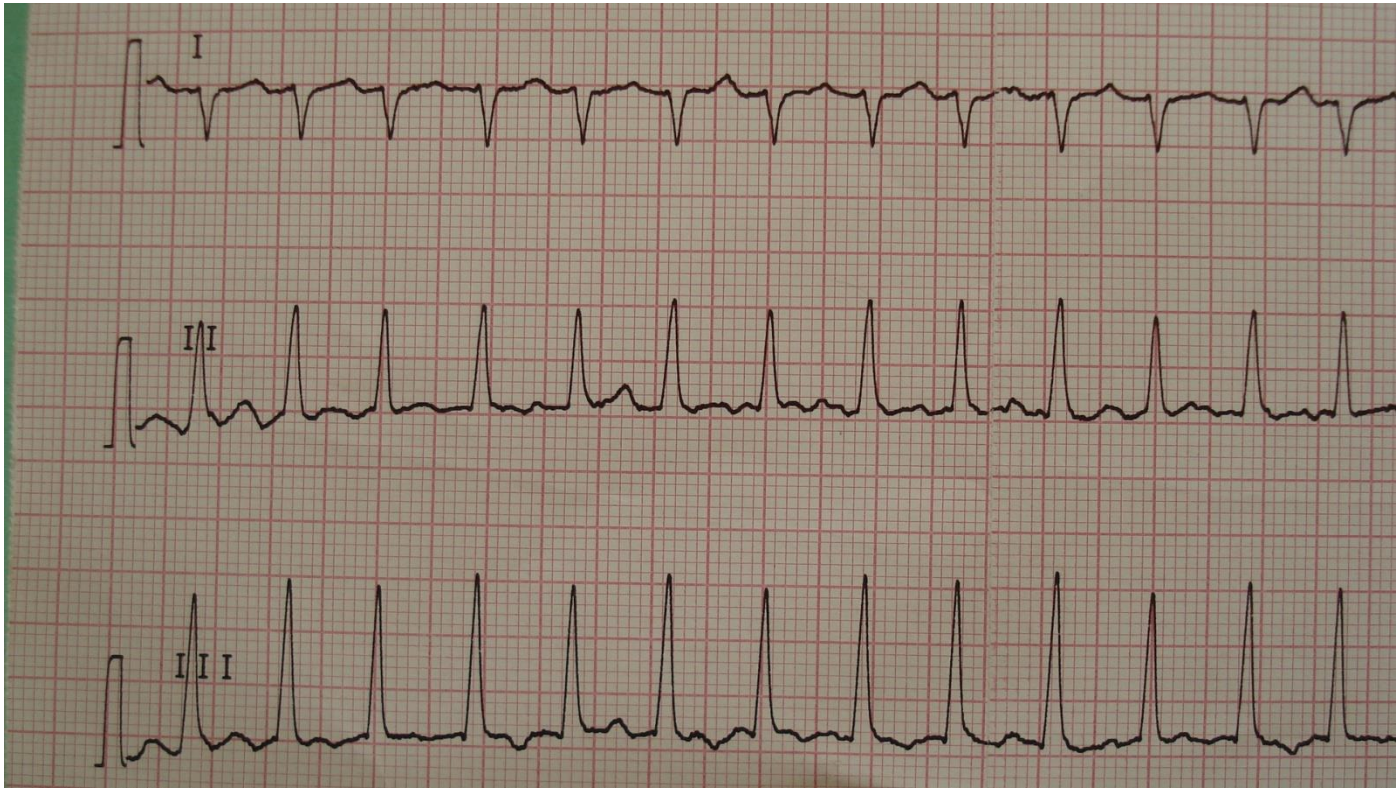


Shows right bundle branch block

Wolff parkinson white syndrome



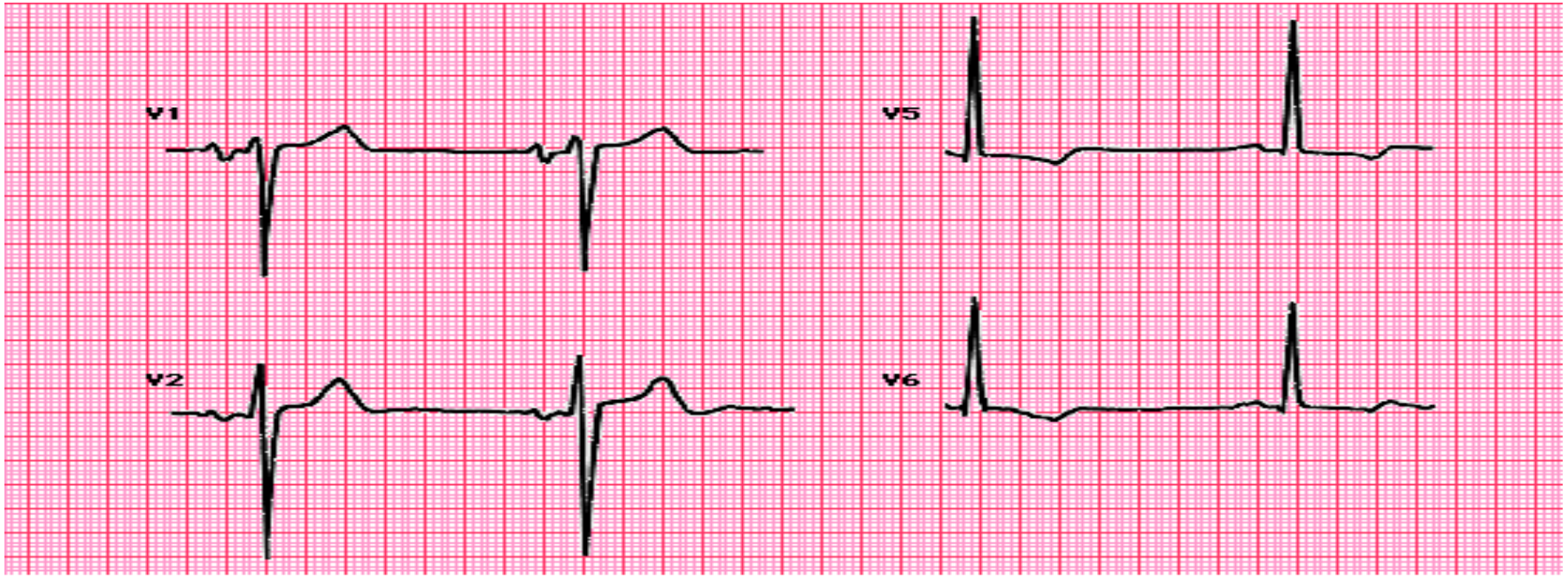
Wolff–Parkinson–White pattern Precordial leads in a patient with preexcitation due to the WPW syndrome. The three characteristic findings are the short PR interval (0.09 sec in this case), the wide QRS, and the delta wave (slurring of the QRS upstroke) that is best seen in leads V5 and V6 (arrows). Courtesy of Ary Goldberger, MD.



An ECG of a patient who presented with syncope.

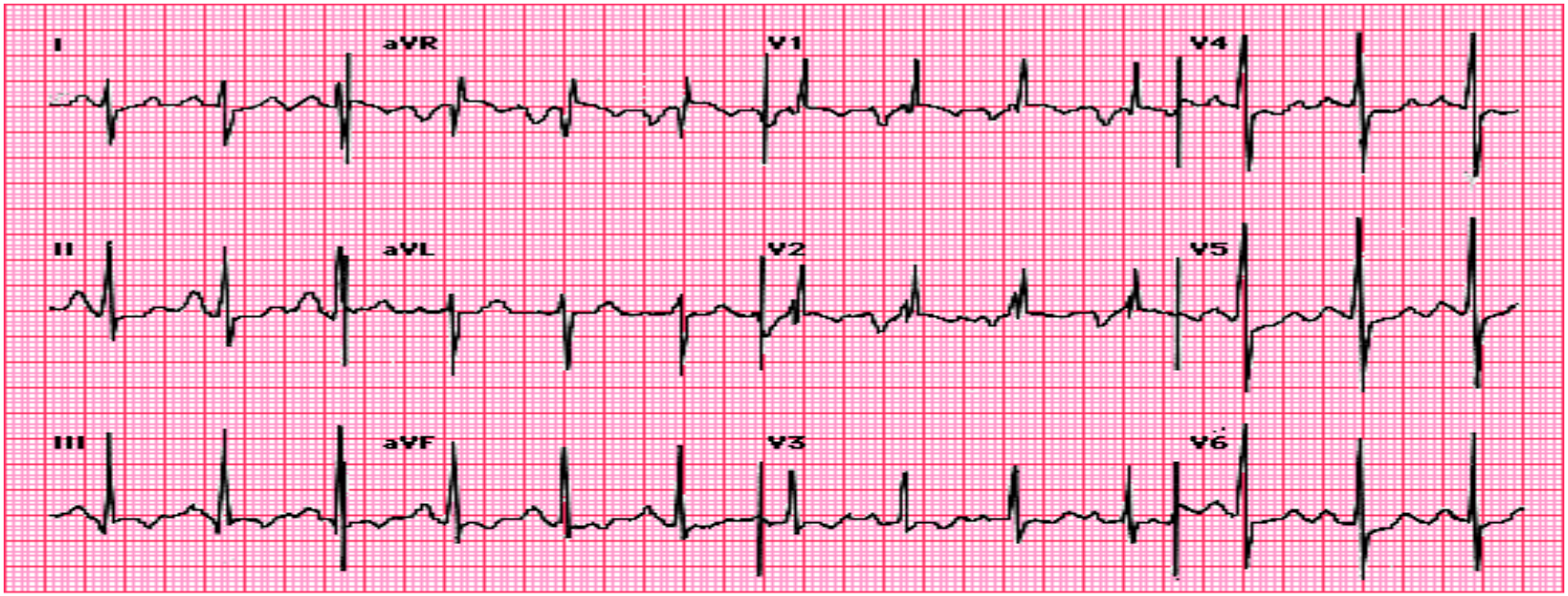
- **The patient has supraventricular tachycardia.**
- **Intravenous adenosine is the treatment of choice.**
- **Carotid sinus massage is useful.**

Left ventricular hypertrophy

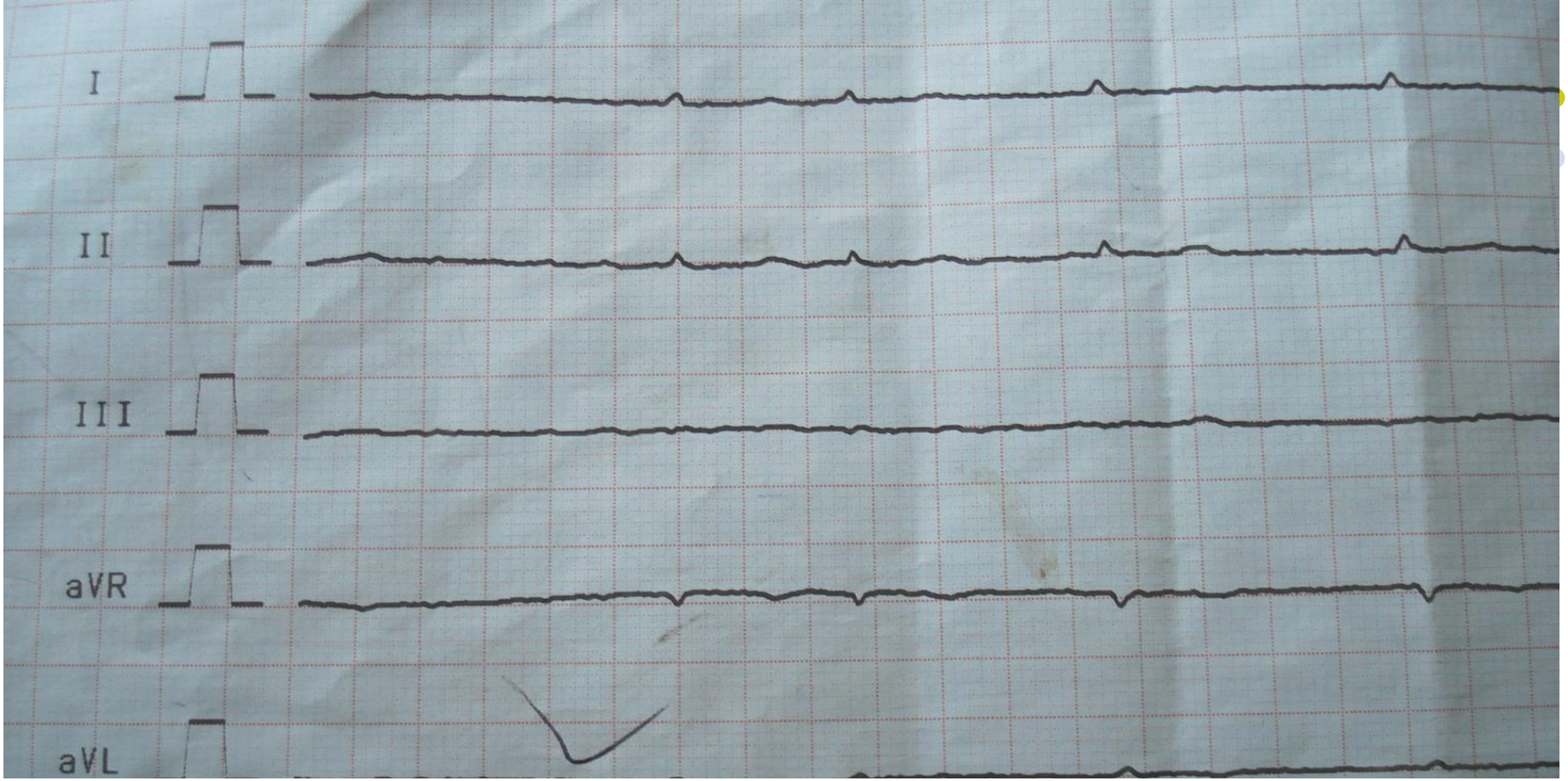


LVH with strain pattern Precordial leads in a hypertensive patient with left ventricular hypertrophy (LVH). The ECG far exceeds commonly used voltage criteria for LVH in the precordial leads: the S in V1 plus the R in V5 equals 50 mm (upper limit of normal 35 mm). Voltage criteria alone may not be diagnostic of LVH. However, the associated repolarization abnormality ("strain" pattern) in leads V5 and V6, characterized by downward sloping ST segment depression and T wave inversion, make LVH highly likely. Left atrial abnormality consistent with enlargement is also present as shown by the biphasic P wave with a broad negative deflection in lead V1. These changes can be induced by a chronic pressure load (hypertension or aortic stenosis) or a chronic volume load (aortic or mitral regurgitation or dilated cardiomyopathy). Courtesy of Ary Goldberger, MD.

Right ventricular hypertrophy

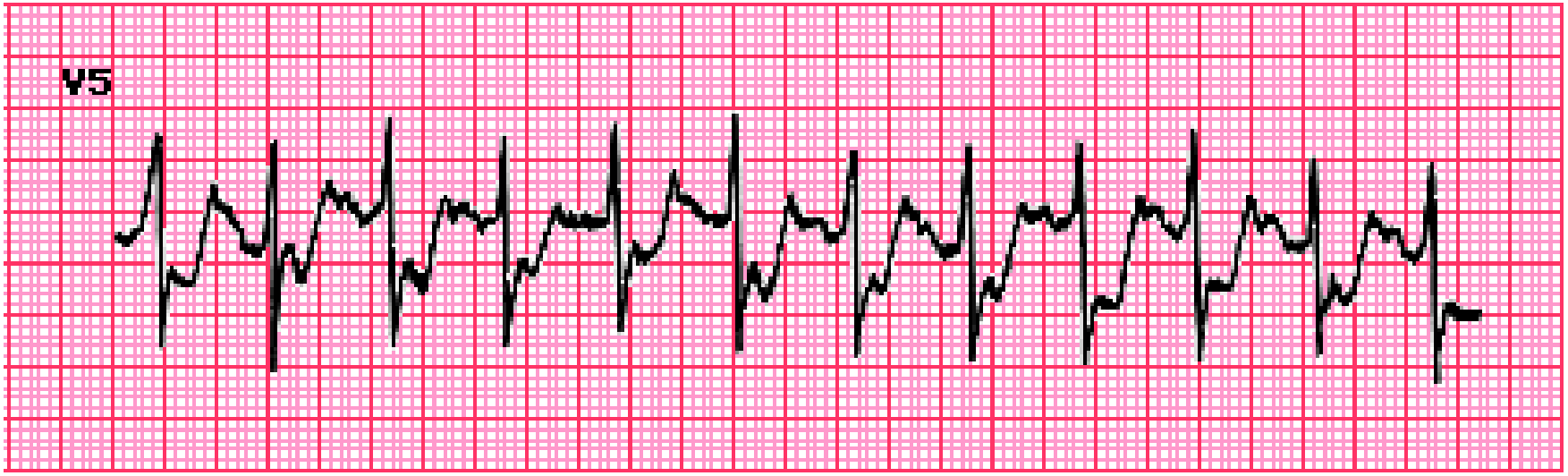


Mitral stenosis Electrocardiogram showing the two major findings of mitral stenosis: right ventricular hypertrophy and left atrial abnormality (enlargement). The presence of right ventricular hypertrophy is indicated by the tall R in V1 (as part of a qR complex) with right axis deviation. Incomplete right bundle branch block is also seen as evidenced by the rsR' in lead V2, and the prominent S waves in leads V5 and V6 with normal QRS duration. The large negative component of the P wave in lead VI (an anterior–posterior lead) indicates left atrial enlargement; the tall (>2.5 mm) P wave in lead II suggests concurrent right atrial enlargement due, in this case, to secondary pulmonary hypertension. The ST and T wave abnormalities are nonspecific. Courtesy of Ary Goldberger.



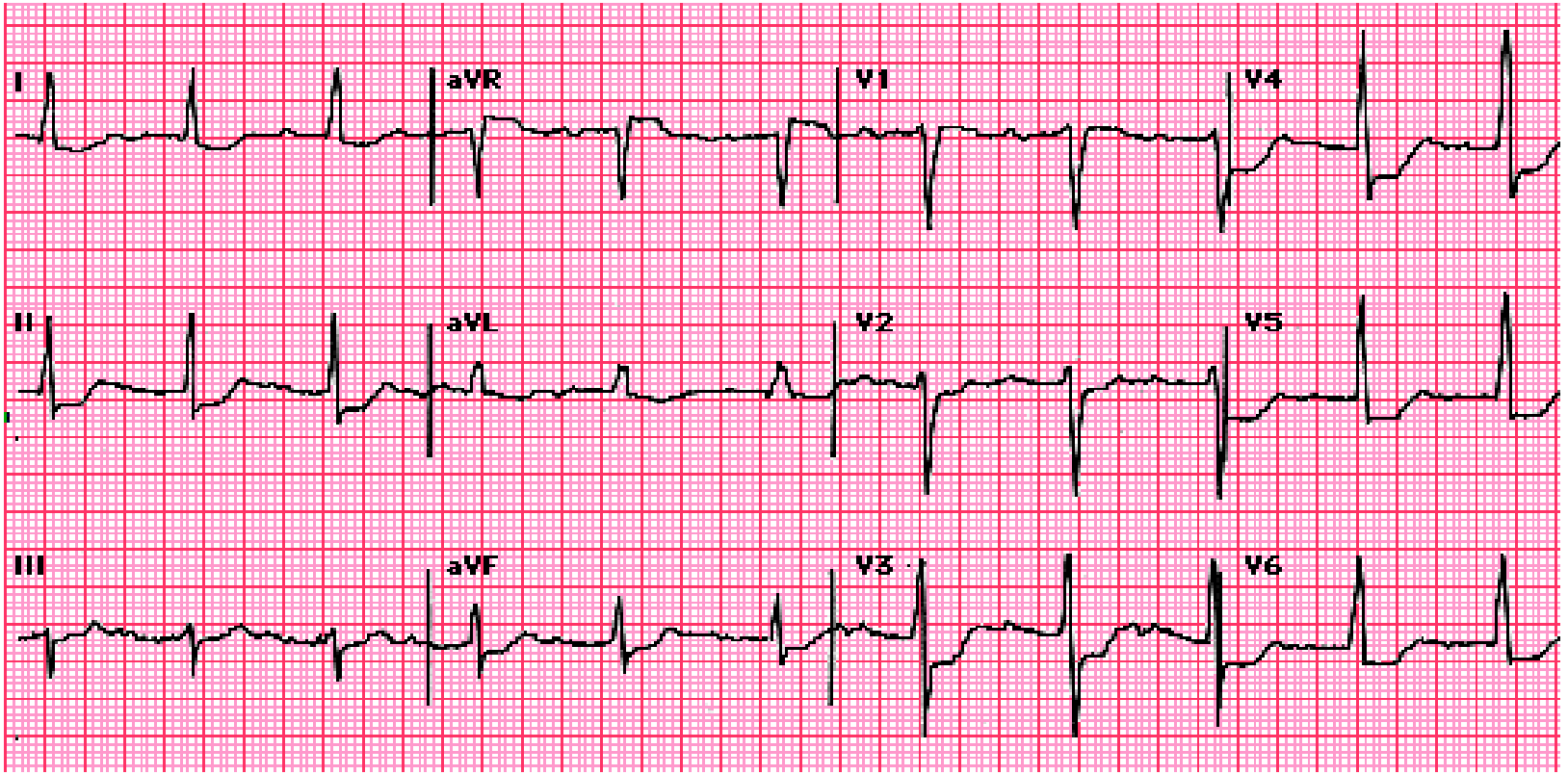
Pericardial effusion

Ischemia



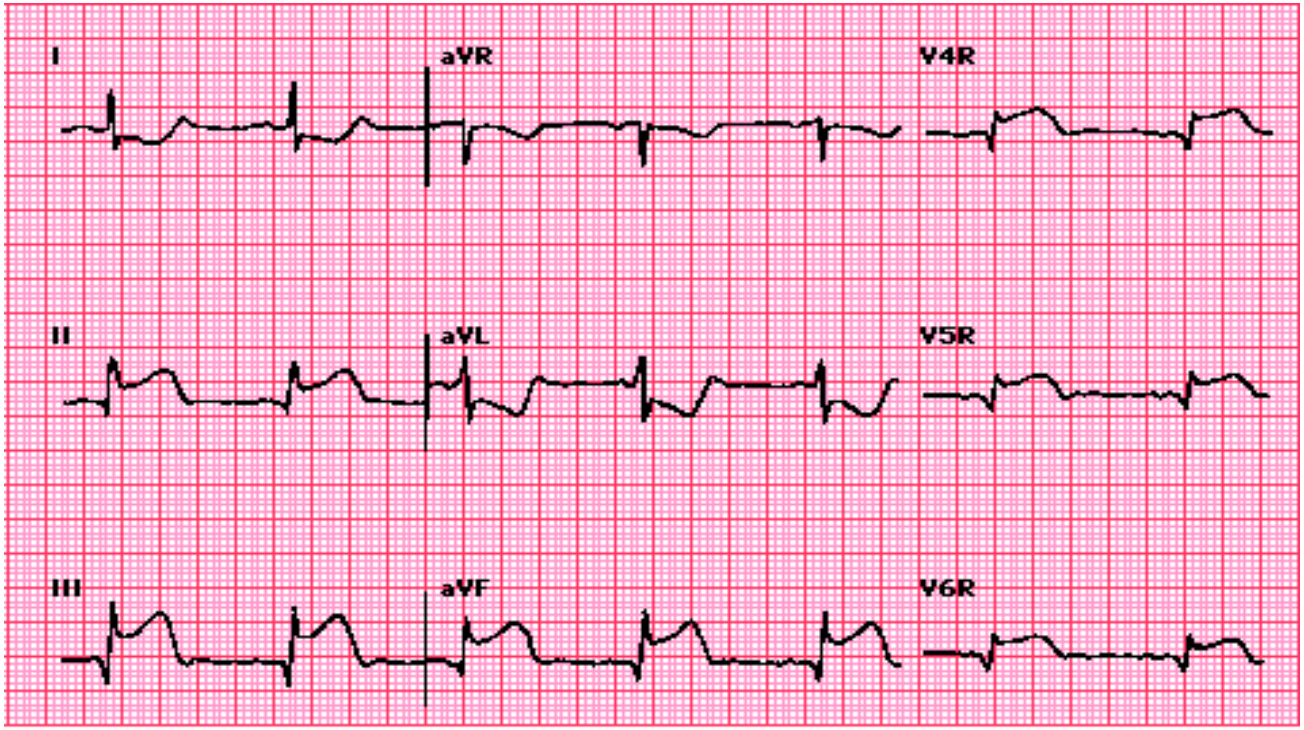
Subendocardial ischemia Sinus tachycardia with marked (5 mm) ST depression that is horizontal or downsloping. This finding is consistent with severe subendocardial ischemia. Courtesy of Ary Goldberger, MD.

Ischaemia



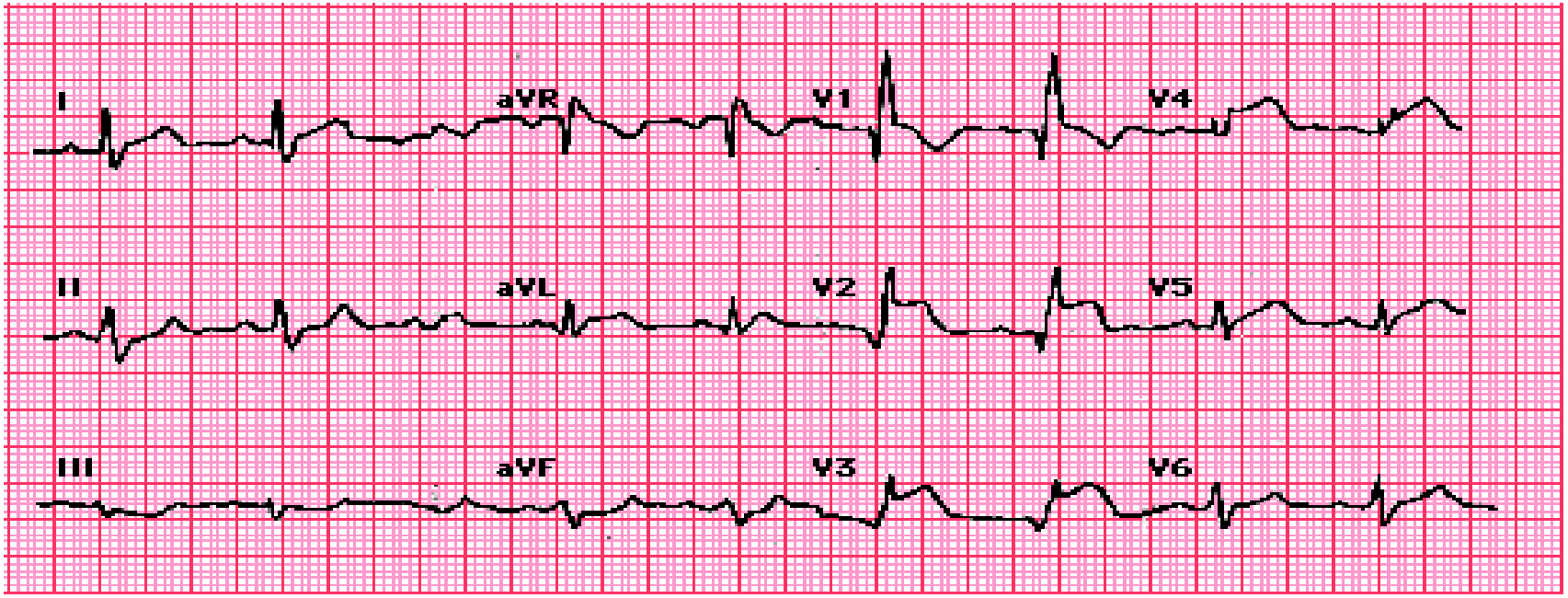
Diffuse subendocardial ischemia Diffuse subendocardial ischemia manifested by prominent ST depressions in leads I, II, aVL, aVF, and V2 to V6, with ST elevation in aVR. A prolonged PR interval (0.28 sec) is also present. Courtesy of Ary Goldberger, MD.

Inferior and Right STEMI

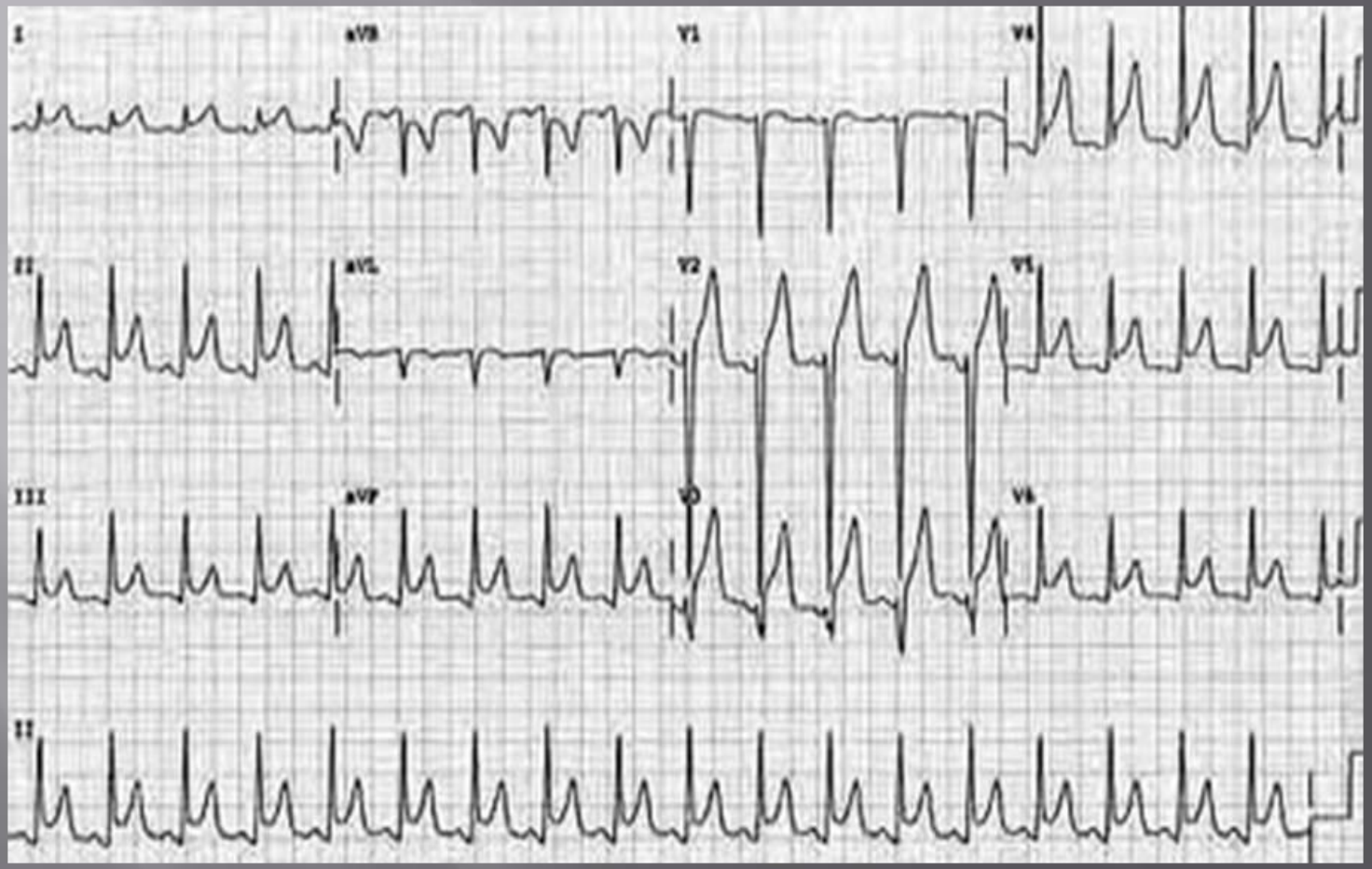


Acute inferior and right ventricular myocardial infarction
Electrocardiogram shows Q waves and prominent doming ST segment elevation in II, III, and aVF, findings which are characteristic of an acute inferior myocardial infarction. ST elevation in the right precordial leads – V4R, V5R, and V6R – indicates right ventricular involvement as well (arrows). The ST depressions in leads I and aVL represent reciprocal changes. Courtesy of Ary Goldberger, MD.

Anterior STEMI

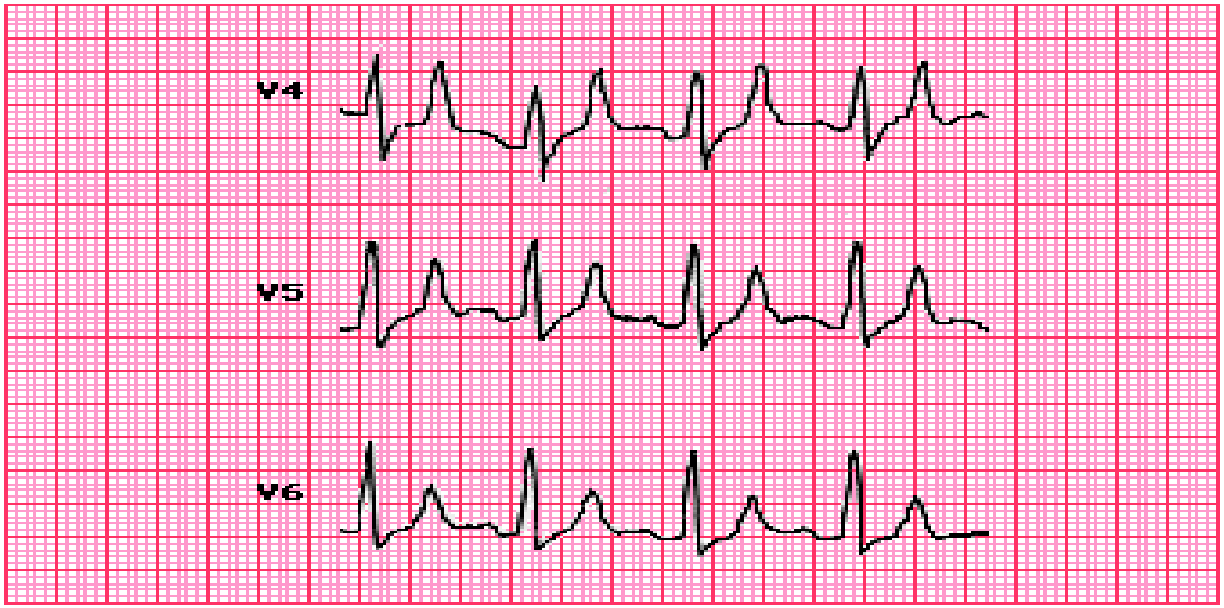


Anterior MI with right bundle branch block Electrocardiogram showing an acute anterior myocardial infarction (Q waves and ST elevations in leads V2 to V4, and ST elevations alone in leads I and aVL) and right bundle branch block (terminal R wave in lead V1 and terminal S wave in leads I and V6). Courtesy of Ary Goldberger, MD.



Pericarditis

Hyperkalemia



Hyperkalemia Lateral precordial leads showing peaked, narrow (tented) T waves and prolongation of the QRS complex (0.14 to 0.16 seconds) associated with moderate to severe hyperkalemia. Courtesy of Ary Goldberger, MD.

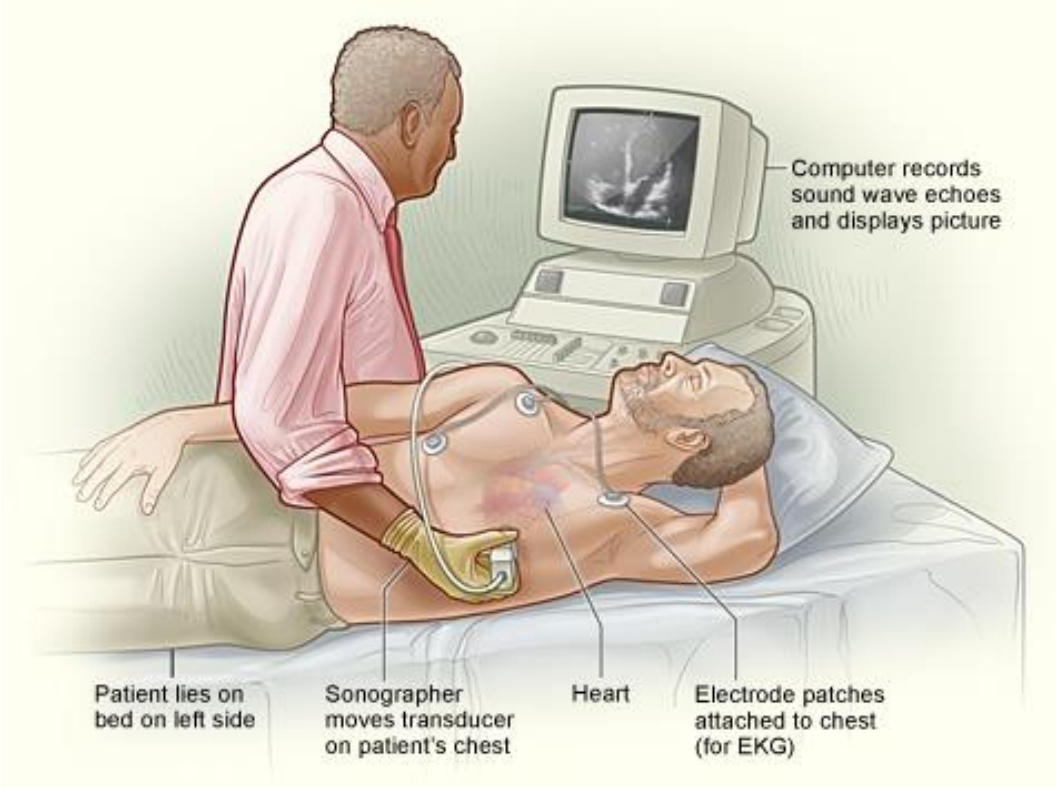
Stress ECG

- Diagnosis of CAD if suspected
- Follow up post ACS to assess exercise tolerance and advice on life style changes
- During myocardial perfusion scanning
- Standard Bruce protocol
- Pharmacological stress testing
  - ? adenosine
- Limitations; LBBB, limb amputation, clinical state of pt etc.

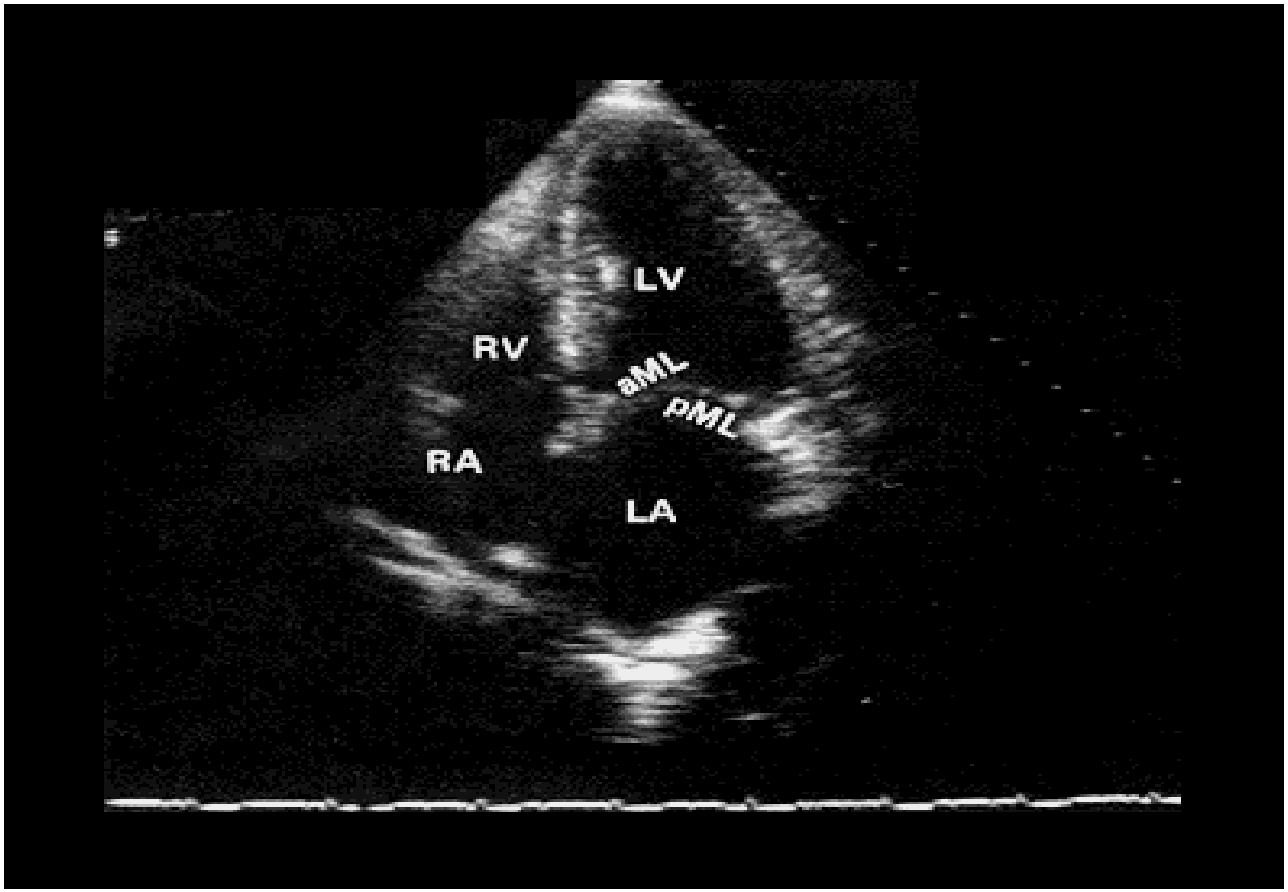
ECHOCARDIOGRAM

- Visualizes the structure of the heart
- Definitive in diagnosis of structural abnormalities of the heart
  - Valvular heart lesions – stenosis/ regurgitation
  - Cardiomyopathies – dilatation/hypertrophy
  - Pericardial diseases – effusion, constriction
  - Congenital abnormalities
  - Diseases of the aorta
- Key in planning and follow up of OHS

Echo set up

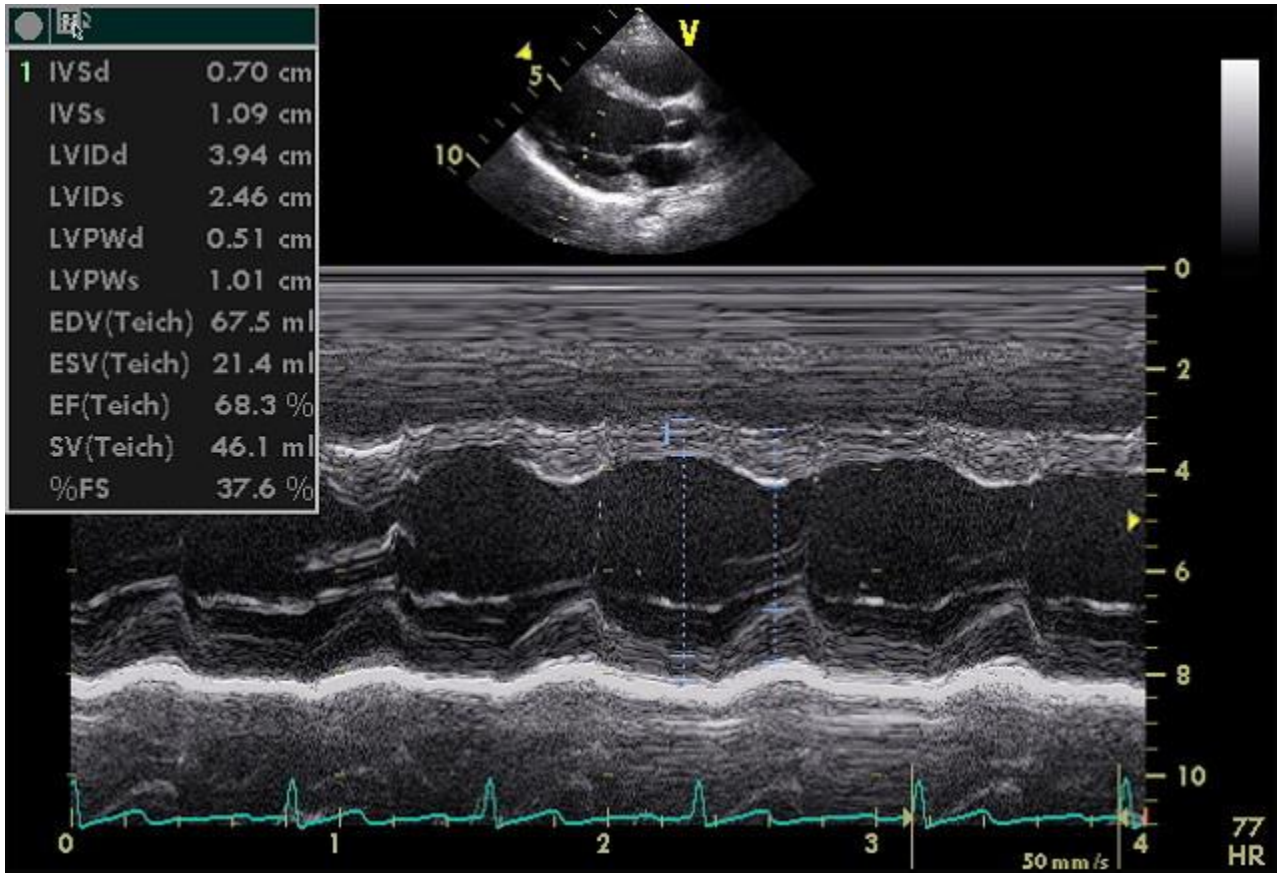

Computer records sound wave echoes and displays picture
Patient lies on bed on left side
Sonographer moves transducer on patient's chest
Heart
Electrode patches attached to chest (for EKG)

Normal 2D echo

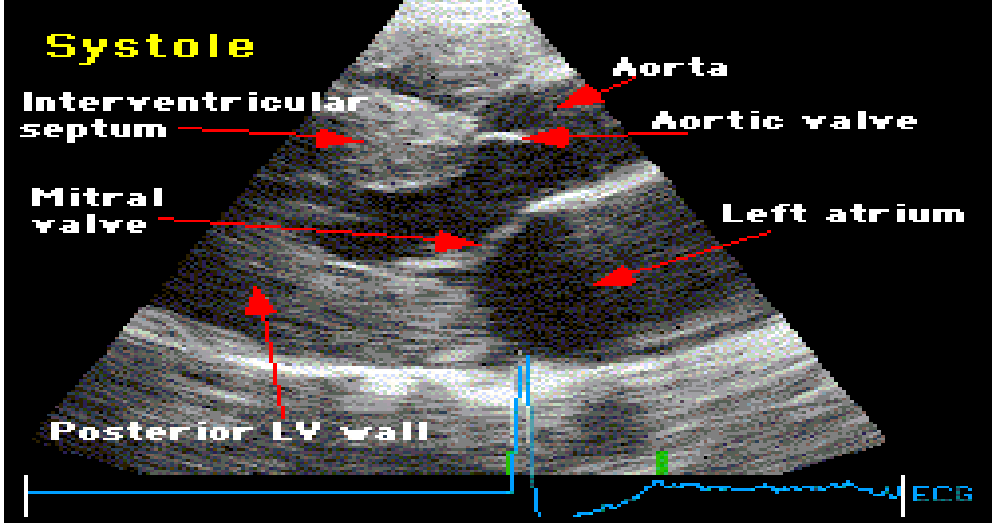


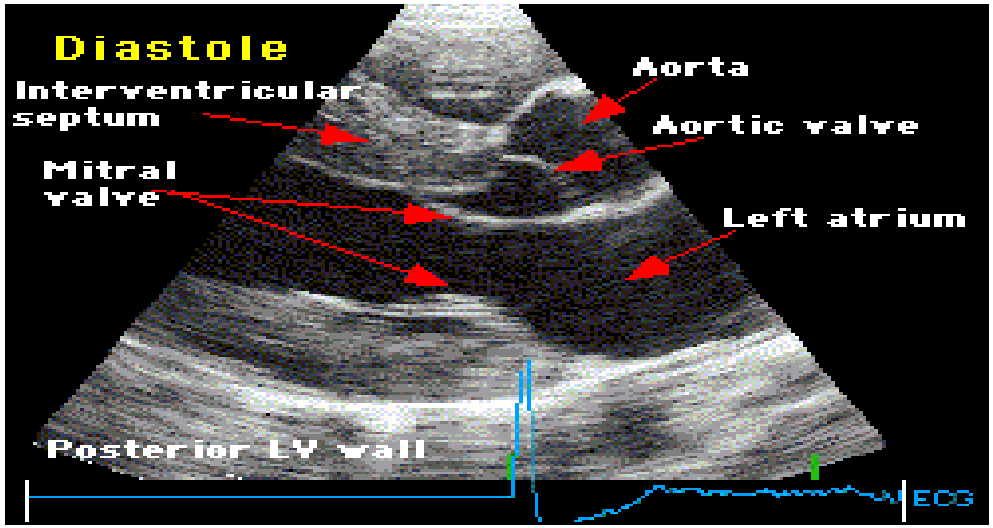
Apical four-chamber view Seen is the apical four-chamber view from a 2-D echocardiogram. The display shows the apex at the top of the screen with the left ventricle (LV) to the viewer's right. aML, anterior mitral leaflet; LA, left atrium; pML, posterior mitral valve leaflet; RA, right atrium; RV, right ventricle.

Normal M mode echo



Hypertrophic cardiomyopathy



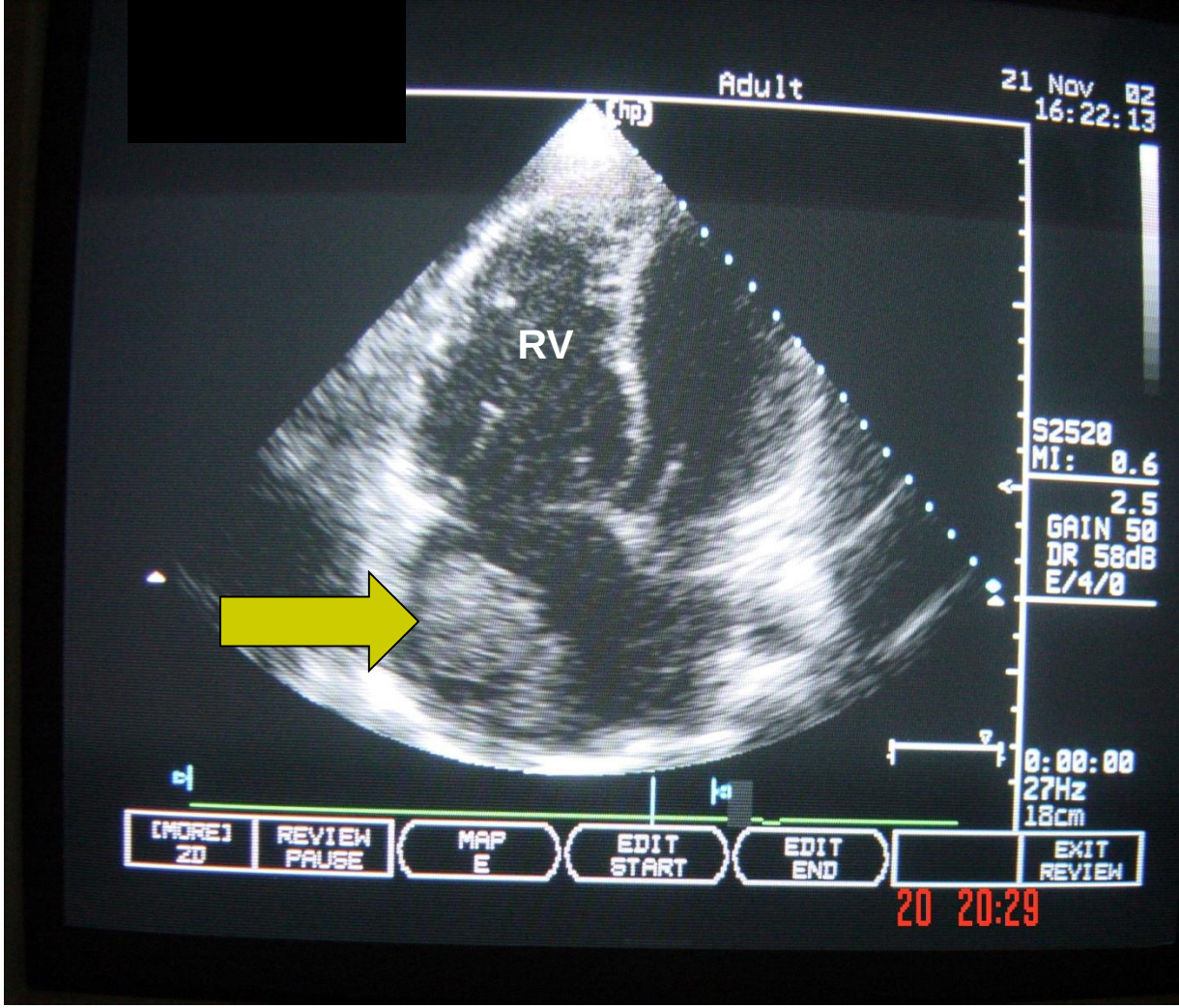


Left ventricular hypertrophy The parasternal long axis view from a 2-D echocardiogram shows marked thickening of the interventricular septum and posterior left ventricular (LV) wall as a result of hypertrophy. (Provided by Thomas Binder, MD, et al. Interactive Echocardiography. A Clinical Atlas. Futura, 1997).

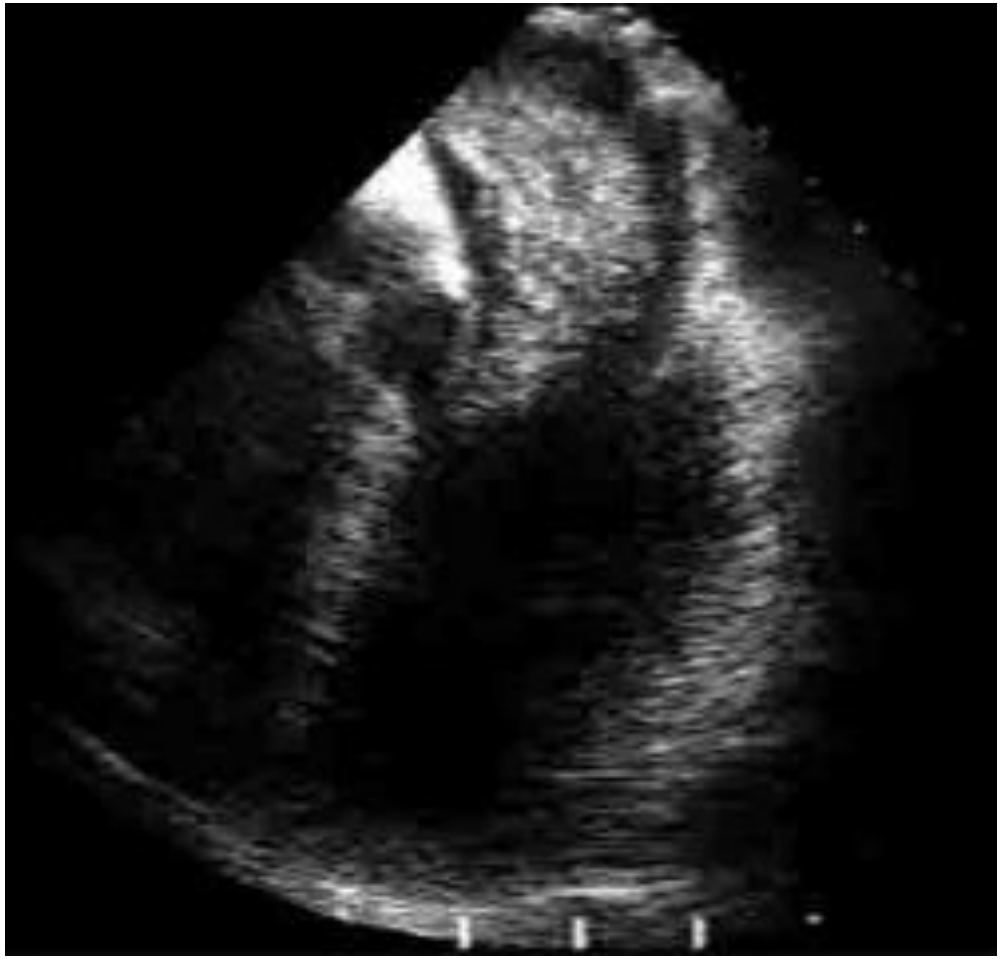
This 2D Echocardiographic finding is consistent with:

A. **Atrial myxoma.**

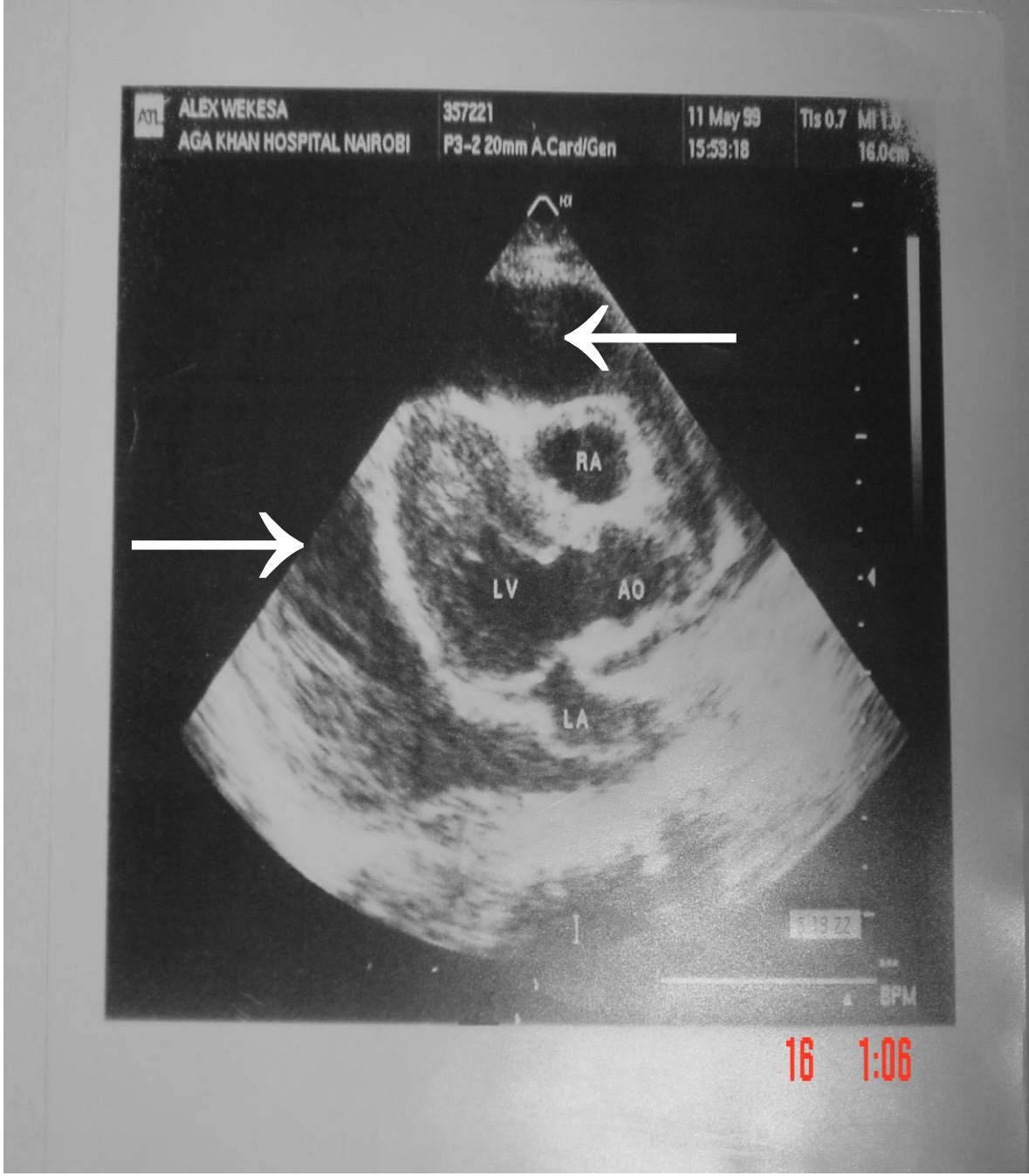
B. **Atrial thrombus**



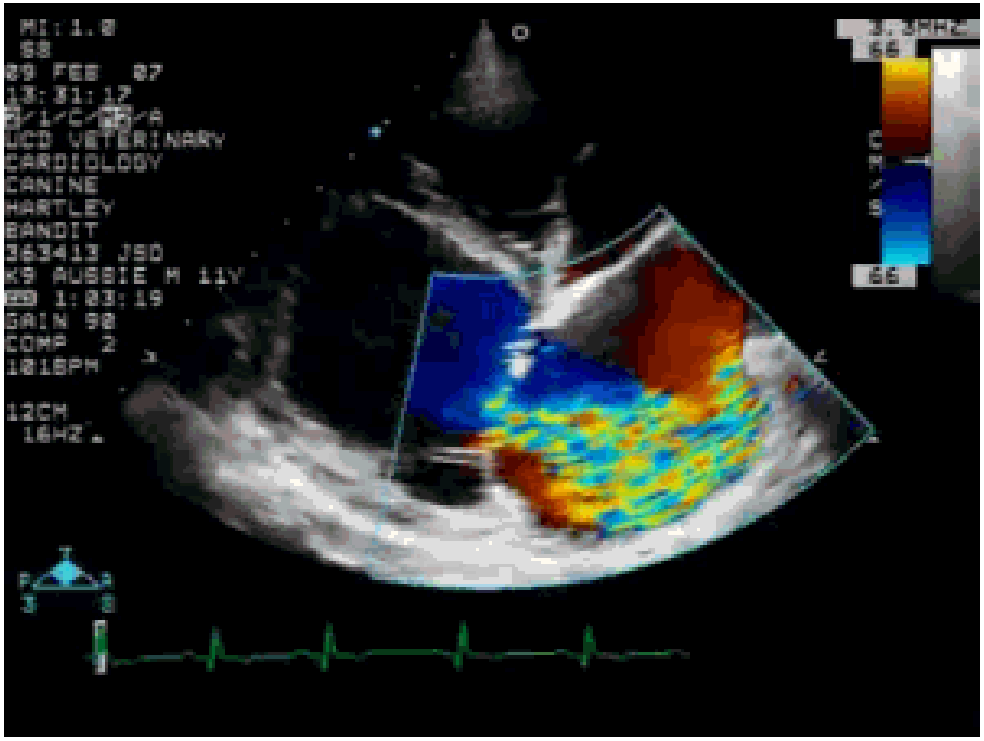
LA thrombus



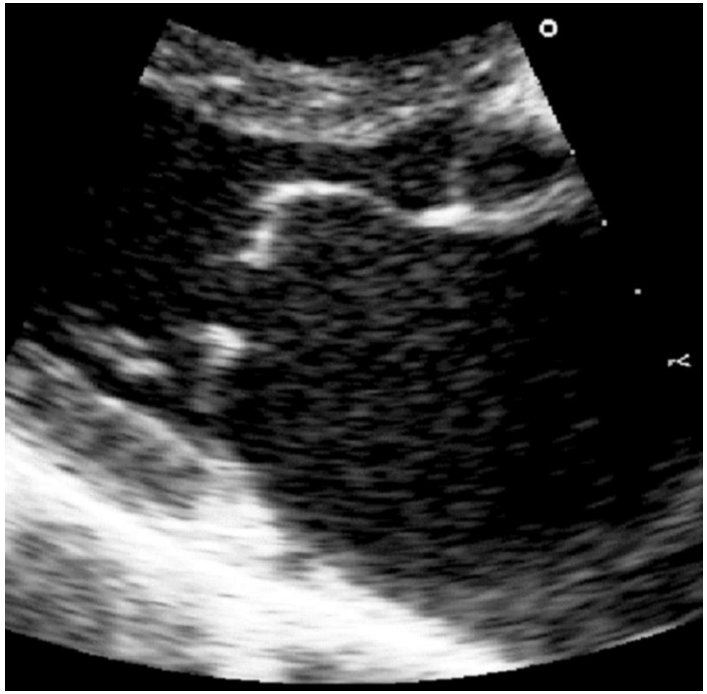
Pericardial effusion

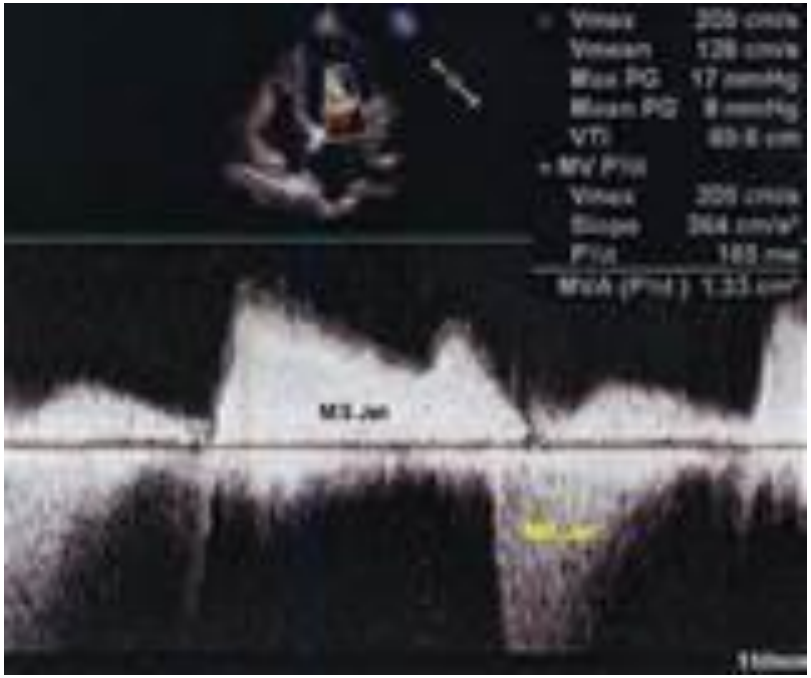


Color Doppler of severe mitral regurgitation

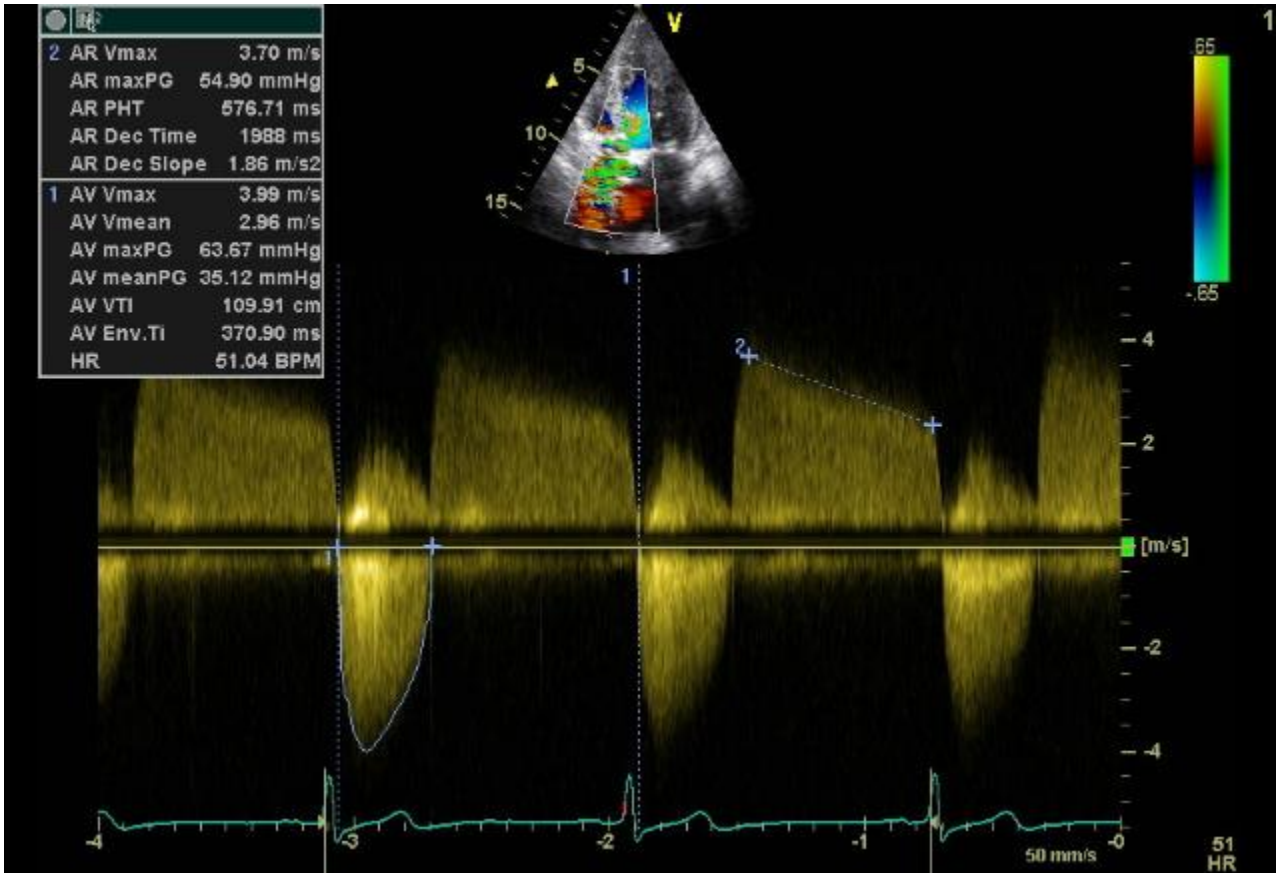


Mitral stenosis

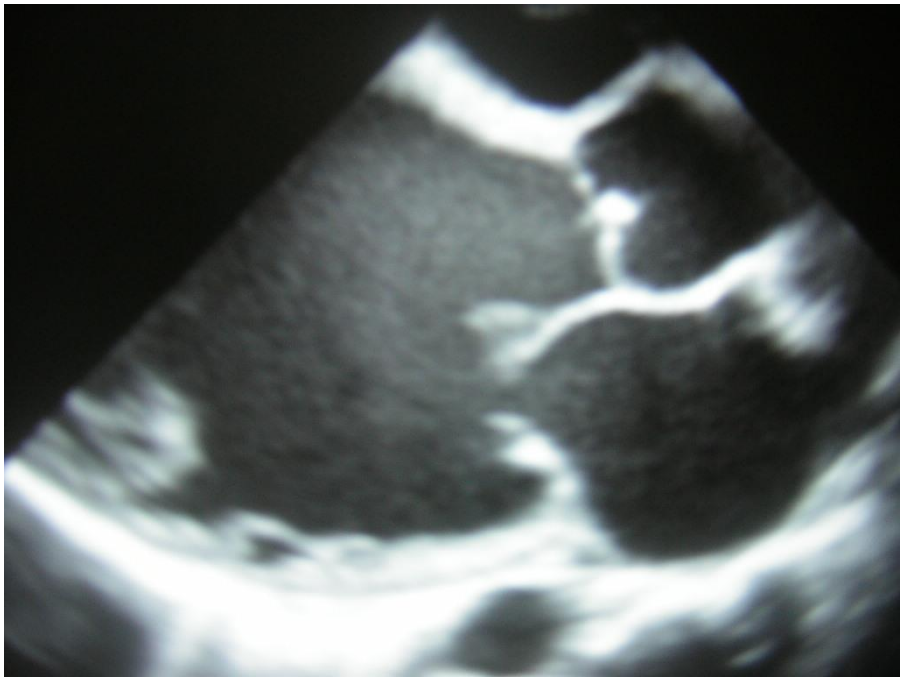




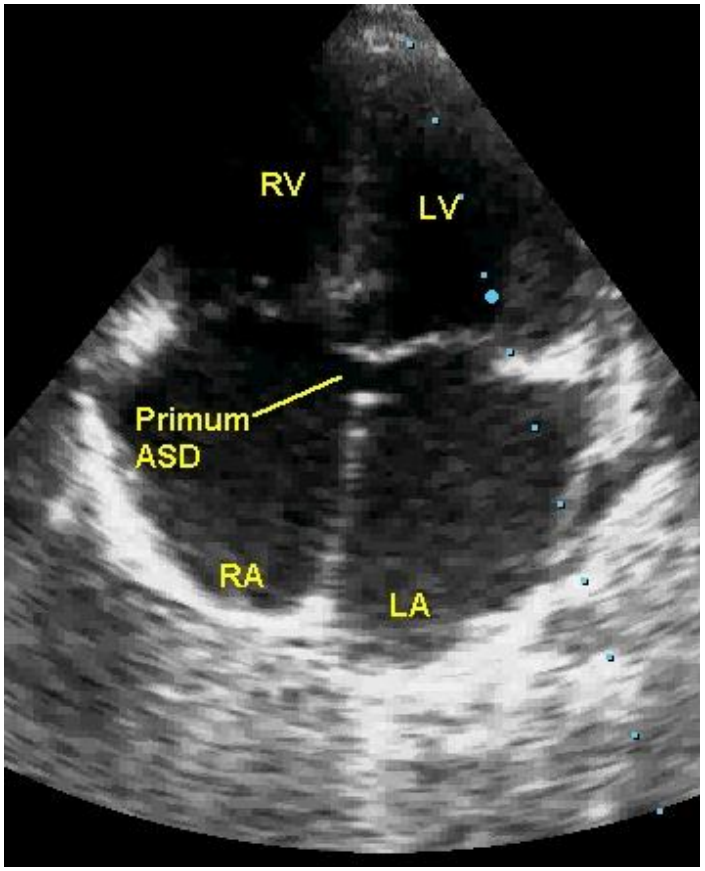
Aortic regurgitation and stenosis



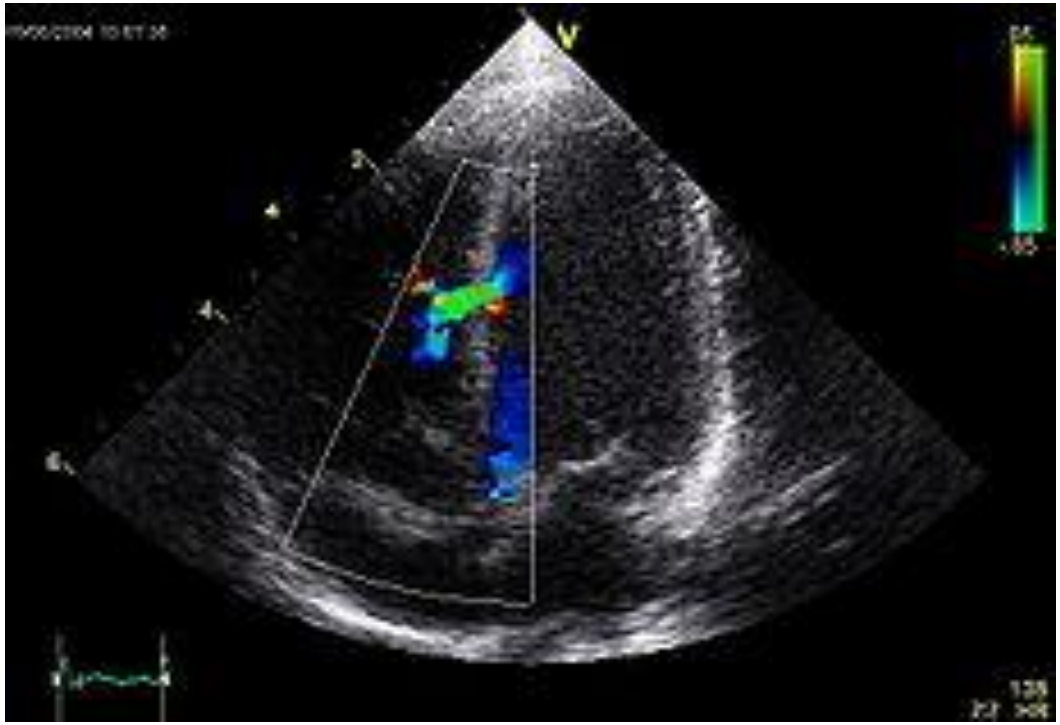
Vegetations in infective endocarditis



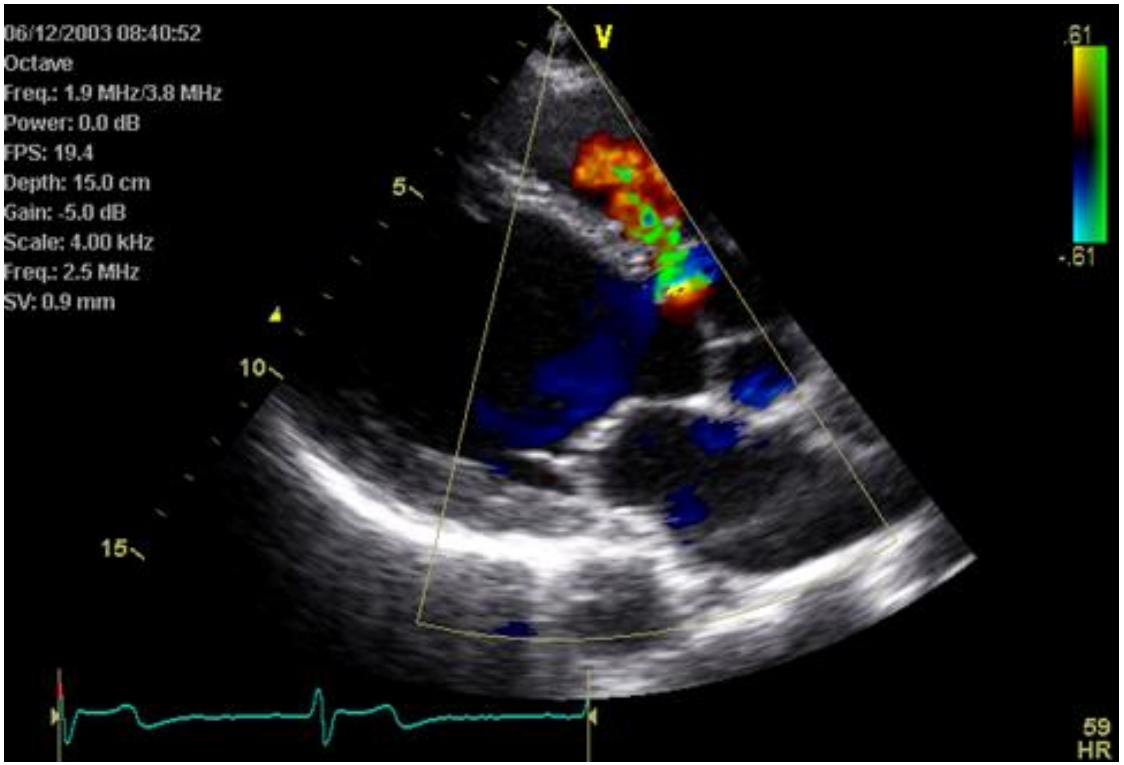
ASD



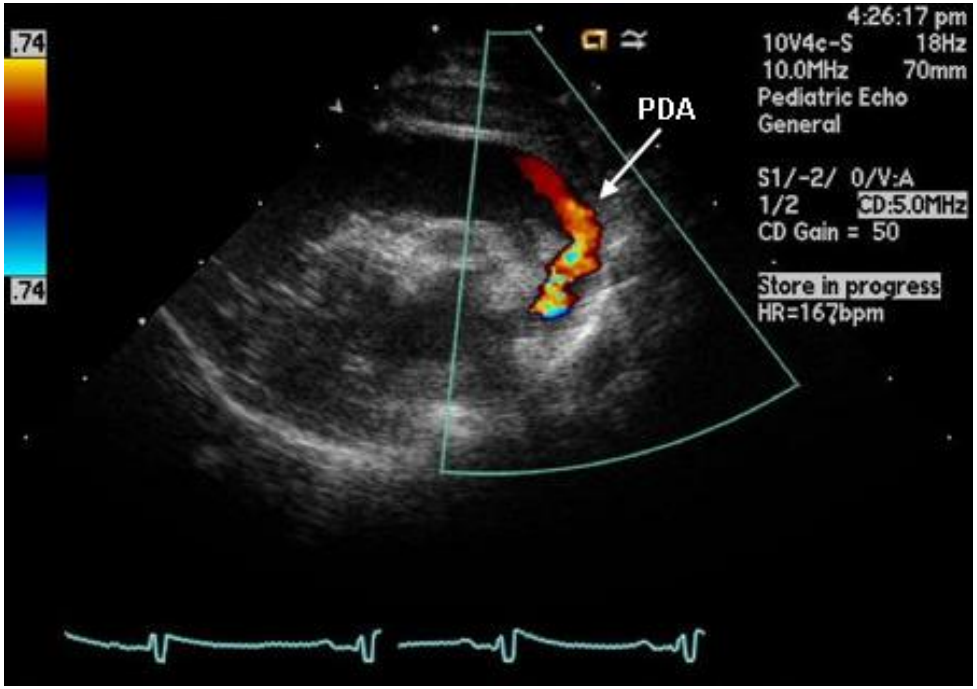
Muscular VSD
L-R Shunt



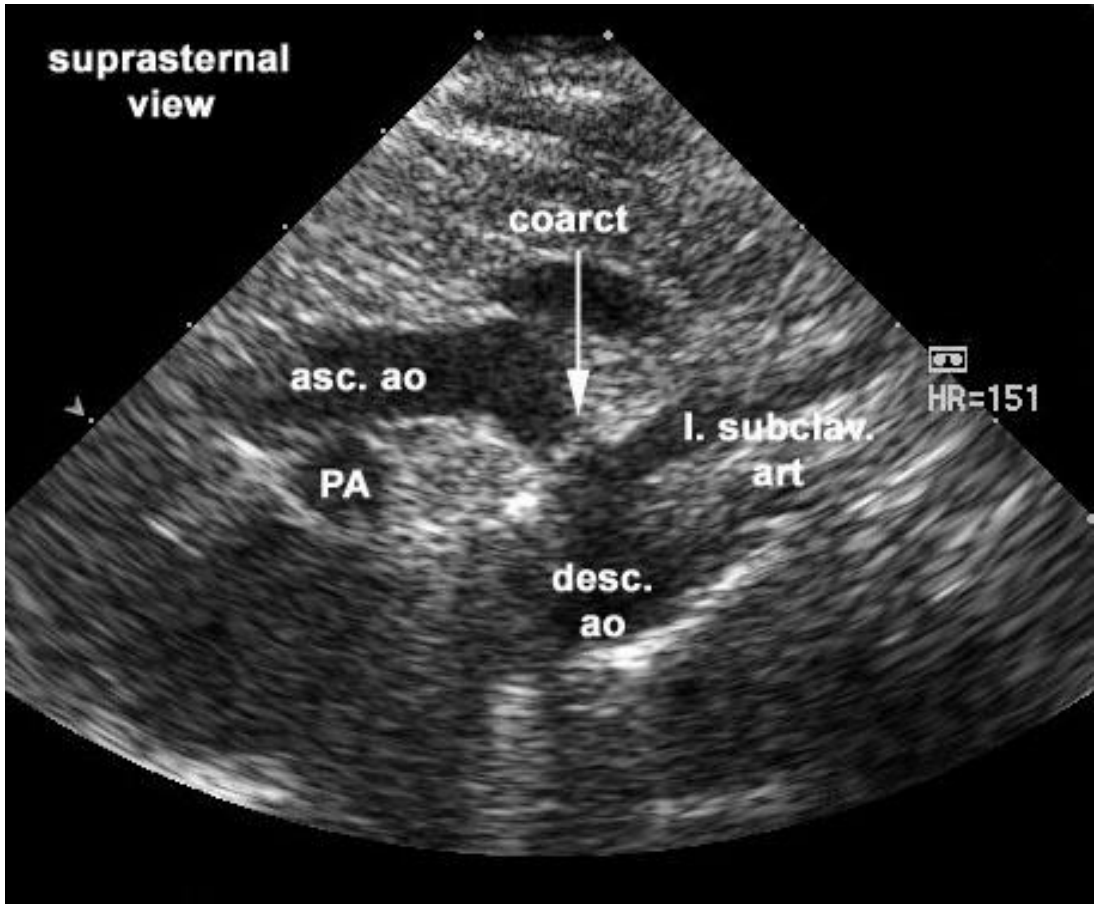
Peri membranous VSD, L – R shunt



PDA


.74
.74
PDA
4:26:17 pm
10V4c-S 18Hz
10.0MHz 70mm
Pediatric Echo
General
S1/-2/ 0/V:A
1/2 CD:5.0MHz
CD Gain = 50
Store in progress
HR=167bpm

Coarctation of aorta



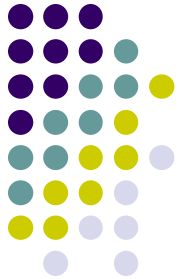
Cardiac catheterization

- Diagnosis of complex congenital heart disease when echo inconclusive, COA.
- Assess pulmonary pressures before correction of VSD, ASD
- Determine significance of shunting in ASD i.e pulmonary/systemic flow ratio
- Diagnosis of restrictive cardiomyopathy vs constrictive pericarditis
- Therapeutic; ASD closure, PBMV,
- Assess LV systolic function, presence of LV thrombus during coronary angiography

Coronary angiography

- Confirm, assess severity of CAD
- Plan treatment of CAD
- PTCA – percutaneous coronary angioplasty

LAB INVESTIGATIONS



- Cardiac enzymes – Key in diagnosis of ACS
  - Troponin I/T
  - Creatnine phosphokinase – CK-MB
- Others
  - U/E/C
  - RBS
  - Lipid profile