

11 Type A WPW syndrome. Note the positive delta wave in all the chest leads and the abnormal Q waves (negative delta waves) in the inferior leads. (Courtesy Dr. Ara G. Tilkian, Van Nuys Calif.)

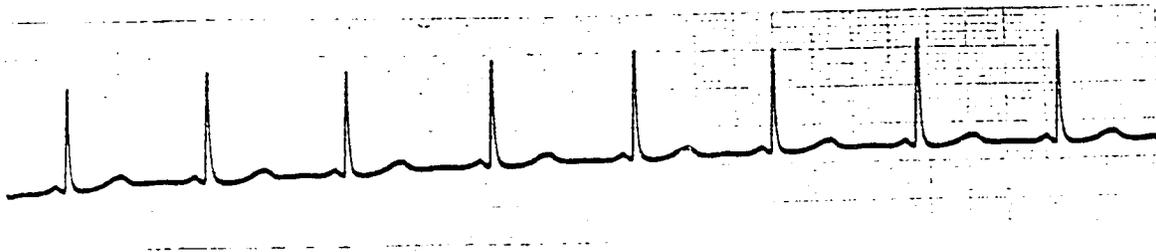
#### e. Arrhythmias in WPW syndrome

- (1) Orthodromic circus - movement tachycardia (CMT) - rapidly conducting accessory pathway
- (2) Incessant CMT
- (3) Antidromic CMT - opposite direction of normal current
- (4) Atrial fibrillation
  - (a) Fast
  - (b) Broad
  - (c) Irregular
- (5) Atrial flutter - not common
- (6) SVT

#### f. Other anomolous tracts/syndromes

- (1) Low-Ganong-Levine - short PR, > 0.11 second normal QRS)
- (2) No delta wave
- (3) AV node is bypassed

Short P-R Interval: Lown-Ganong-Levine S.



(4) James fibres - normal extensions of intranodal conduction pathways of the atria

g. Significance

- (1) Occurs in all age groups
- (2) Males 60-70%
- (3) Traceable to a congenital abnormality
- (4) Health care seeking is usually due to recurrent SVT, atrial fibrillation, syncope or other symptoms
  - (a) A-V recurrent tachycardia - 80%
  - (b) AF 15%-30%
  - (c) Atrial flutter 5%
  - (d) Ventricular tachycardia - rare
- (4) Frequently misdiagnosed as junctional and mistreated
- (5) May have low cardiac output symptoms

h. Treatment

- (1) Drug therapy in WPW syndrome
  - (a) Affects - A-V node
    - (1) Digitalis
    - (2) Propranolol

(3) Verapamil

(4) Vagal stimulation

(b) Affects - accessory pathway

(1) Quinidine (atrial flutter/fibrillation)

(2) Procainamide

(3) Disopyramide

(c) Affects both

(1) Flecainide

(2) Encainide

(3) Amiodarone

(2) Surgical ablation of the accessory pathway

i. Nursing concerns

(1) Monitor for tachydysrhythmias

NOTE: Sudden death episodes are possible.

(2) Patient/family education regarding medications, test procedures and surgery; CPR

NOTE: Prognosis - doesn't alter normal life expectancy.

E. Atrioventricular Block

i. First degree A-V block

a. Mechanism

(1) Prolongation of conduction rather than "block"; a delayed conduction through the A-V node

(2) A condition superimposed on another rhythm

EXAMPLE: Sinus rhythm with a first degree block.

First-Degree AV Heart Block



b. Clinical implications

- (1) Depends on level of the lesion
- (2) Clinical scenario
  - (a) Of patients with acute MI, 13% develop first degree heart block
  - (b) Of these 50% go on to develop high grade block within first 72 hours post MI
  - (c) May be seen in persons without cardiac disease

c. ECG recognition

- (1) Rate: varies with underlying rhythm
- (2) Rhythm: varies with underlying rhythm - usually regular
- (3) P waves uniform and each is followed by a QRS - 1:1 to the ventricles
- (4) PR interval > .20 second
- (5) PR interval does not change from beat to beat
- (6) Narrow QRS representative of conduction delay with A-V node

d. Etiology

- (1) Myocardial ischemia or infarct
- (2) Infectious diseases, rheumatic fever, myocarditis

(3) **Drugs**

- (a) Quinidine - rare
- (b) Digitalis
- (c) Calcium channel blockers
- (d) Beta blockers

(4) Increased parasympathetic tone will slow A-V conduction velocity

e. **Treatment**

- (1) Usually none
- (2) Withdrawal of precipitating drugs
- (3) Atropine may speed conduction and increase HR in symptomatic patient

f. **Nursing concerns**

- (1) If new, obtain 12 lead ECG and continuous cardiac monitor
- (2) Observe for progression
- (3) Assess for symptoms of ↓ CO and ↓ tissue perfusion

2. **Second degree block - Mobitz Type I (Wenckebach)**

a. **Mechanism**

- (1) A-V node normally has a slow-response action potential and slow conduction
- (2) Depressive influences compound the normal A-V action
- (3) Progressive lengthening of A-V conduction time

b. **Clinical implications**

- (1) Abnormal
  - Digoxin toxicity
  - Acute inferior wall MI - 50% will progress to high grade A-V block
  - Post-operative
  - Immediately following open heart surgery
- (2) Normal or benign
  - Chronic, as in patients without organic disease

**Wenckebach-Type Second-Degree AV Block**



c. ECG recognition

- (1) Rate: varies with underlying rhythm
- (2) Rhythm: irregular "grouped beating"
- (3) PR lengthens until one P wave is not conducted
- (4) Conduction ratio is counted P: QRS

EXAMPLE: 4:3 Wenckebach

- (5) If conduction is 2:1, there is no PR lengthening no grouped beating
- (6) R-R intervals shorten

NOTE: In atypical Wenckebach, the 1st R-R interval may lengthen rather than shorten.

- (7) R-P intervals shorten
- (8) QRS usually narrow < .12; if broad QRS there is probably both A-V nodal block and BBB

d. Etiology

- (1) Occurs with MI - more common with inferior MI

(2) Usually represents reversible ischemia in A-V node or common bundle

(3) Drug induced - particularly calcium channel blockers

e. Treatment

(1) Generally none

(2) Withdrawal of one or more drugs if drug-induced

(3) Observation

(4) Atropine, epinephrine or isoproterenol - to enhance conduction through A-V node if symptomatic

(5) Temporary pacemaker if hemodynamically significant - rarely needed

f. Nursing concerns

(1) If new obtain ECG and continuous monitor

(2) Observe for progression

(3) Document rhythm strips

(4) Assess patient for ↓ CO and ↓ tissue perfusion

3. Second degree heart block - Mobitz Type II

a. Mechanism

(1) Lesion usually within or below the bundle of His

(2) Usually associated with a RBBB

b. Clinical implications

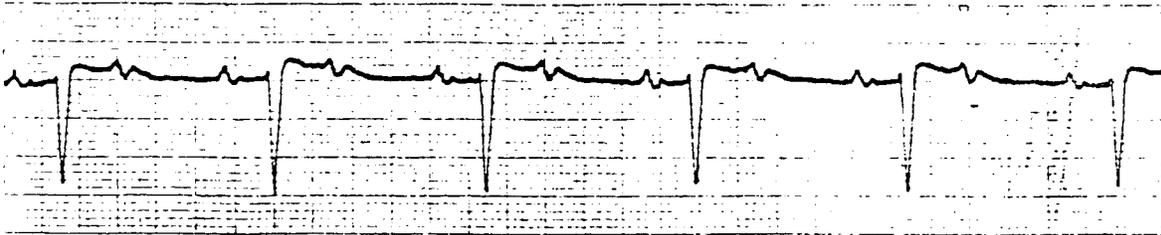
(1) Less common than Type I

(2) Often associated with anteriorseptal MI

(3) More frequently progresses to complete heart block with a ventricular escape rhythm - especially with MI - no warning and rapid progression

- (4) May cause CHF or angina

#### Second-Degree AV Block (2:1 Type)



#### c. ECG characteristics

- (1) Rate: Usually bradycardia, 1/2 to 1/3 normal rate
- (2) Rhythm: May be regular or irregular
- (3) P waves: Upright and uniform; more than one P wave for every QRS complex
- (4) "Dropped" beats
- (5) PR intervals remain the same on conducted beats and are not prolonged  $<.20$
- (6) QRS broad ( $>0.12$  sec) if associated with RBBB

#### d. Etiology

- (1) Anteroseptal MI
- (2) Vagal maneuvers (vomiting or valsalva after MI)
- (3) Induced by drugs: digitalis toxicity, quinidine, lidocaine, procainamide

#### e. Treatment

- (1) Withdrawal of drugs (if indicated)
- (2) Atropine has no effect if block is infranodal
- (3) Often temporary pacemaker immediately inserted, acute MI or symptomatic. Permanent demand pacemaker if block is still present 72-96 hours post MI.

- (4) Assess patient for angina, CHF, ↓ CO, ↓ tissue perfusion
- (5) Monitor for progression, monitor patient response to therapy
- (6) Norepinephrine, dobutamine if atropine is ineffective

f. Nursing concerns

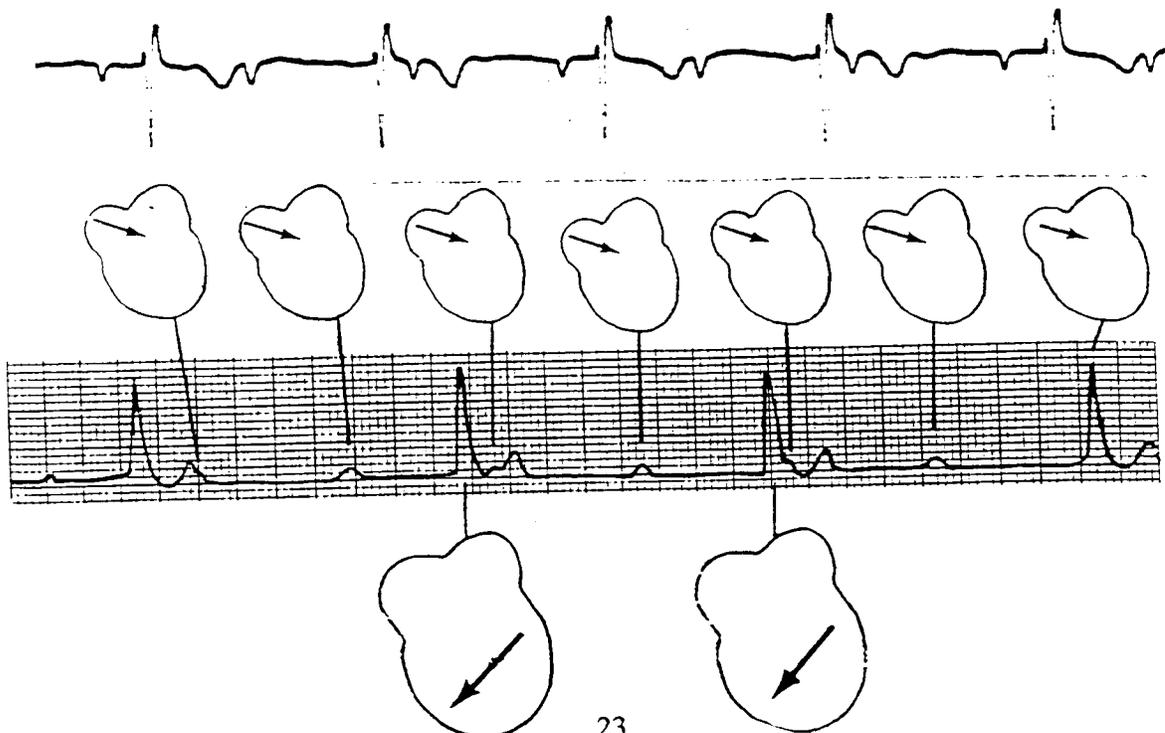
- (1) Rhythm requires immediate response and assessment of patient's symptoms
- (2) May require emergent temporary pacemaker especially in face of acute MI
- (3) Code, if symptoms severe enough
- (4) Monitor effects of sympathetic stimulating medication

3. Complete heart block (third degree block)

a. Mechanism - lesion may be A-V nodal or in the bundle of His

b. Clinical implications

- (1) Rate and dependability of escape pacemaker depends on the level of the lesion
- (2) Loss of adequate cardiac output



c. ECG recognition

(1) Rate

(a) Atrial rate usually normal and regular

(b) May have idiojunctional escape (40-60) or idioventricular escape (20-40)

(2) Regularity: Usually regular

(3) P wave: Upright and uniform; more P waves than QRS

(4) PRI: No relationship between P waves and QRS complexes

(5) QRS < .12 second if junctional escape

> .12 second if ventricular escape

d. Etiology

(1) Acute MI

(2) Drug toxicity

(3) Degenerative changes of age

(4) Congenital complete A-V block

e. Clinical significance in cardiac output may result in

(1) Angina

(2) CHF

(3) Syncope (Stokes-Adams syndrome)

(a) Faintness, lightheadedness syncope

(b) Prognosis poor

(c) Requires CPR if symptomatic

(4) Death results from pump failure or shock

f. Treatment

- (1) Prevention - early treatment of warning rhythms
- (2) Atropine
- (3) Isuprel
- (4) Withhold drugs (digitalis, quinidine) which may contribute
- (5) Temporary pacemaker acutely; permanent pacemaker may be required long term

g. Nursing concerns

- (1) Requires immediate assessment of patient and appropriate emergent (ACLS) action

- (2) Code

4. A-V dissociation

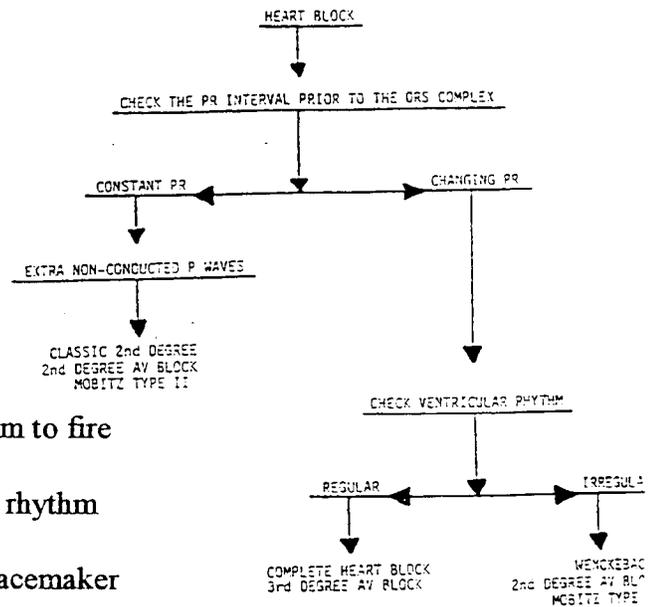
a. Independent beating of atria and ventricles

b. Used generically, doesn't tell much

c. Causes

- (1) Sinus bradycardia - allows another rhythm to fire
- (2) Triggered activity - competes with sinus rhythm
- (3) Abnormal automaticity of a subsidiary pacemaker
- (4) Complete A-V block - escape of subsidiary focus
- (5) Any combination of the above

d. Presence of absence of A-V dissociation depends on the rate and temporal relationships of the two pacemakers



## 5. Summary Table of heart blocks - conduction delays

AV block	Impulses	Rhythm	PR interval	ECG
First degree	All conducted (with delay)	Regular	Prolonged (>0.20 sec.); constant	
Second degree Mobitz I (Wenckebach)	Some conducted, some blocked	Irregular	Progressively longer until dropped beat; cyclic pattern	
Second degree Mobitz II	Some conducted, some blocked	Irregular	Constant for conducted beats; some Ps without QRSs; can appear 2:1, 3:1, or occasionally	
Third degree	None conducted	Regular QRS; independent regular P	Variable; no P-QRS relationship	

## F. Bundle Branch Block

1. Differential diagnosis of left or right bundle branch blocks require a 12 lead ECG with precordial leads
2. Characteristic widened QRS complexes
3. Mechanism
  - a. Conduction through one or more of the bundle branches is slowed or stopped
  - b. Affected area receives impulse through the myocardial muscle - slower

c. Sequential depolarization or dyssynchronous depolarization

4. Causes of BBB

a. Scarring or edema adjacent to affected branch

b. Ischemia or hypoxia to myocardium from COPD, smoking, angina, or MI

5. Treatment

a. None if asymptomatic

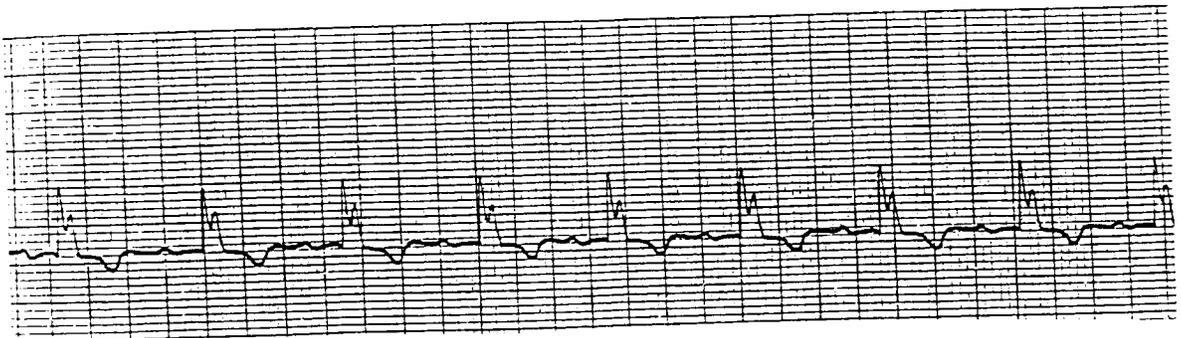
b. Identify and treat underlying cause

c. Possible temporary pacing in face of MI

d. Treat symptoms

6. Right bundle branch block

AN EXAMPLE OF A RIGHT BUNDLE BRANCH BLOCK PATTERN.



a. Mechanism

(1) Block at right bundle

(2) Conduction proceeds normally down left bundle

(3) Area normally depolarized by right bundle is depolarized through myocardium

b. Clinical complications

(1) Usually seen with anterior vs inferior MI

(2) In setting of acute MI RBBB has a 27% risk of CHB

(3) In young individual, right ventricular hypertrophy may produce RBBB

(4) Older person - probable CAD

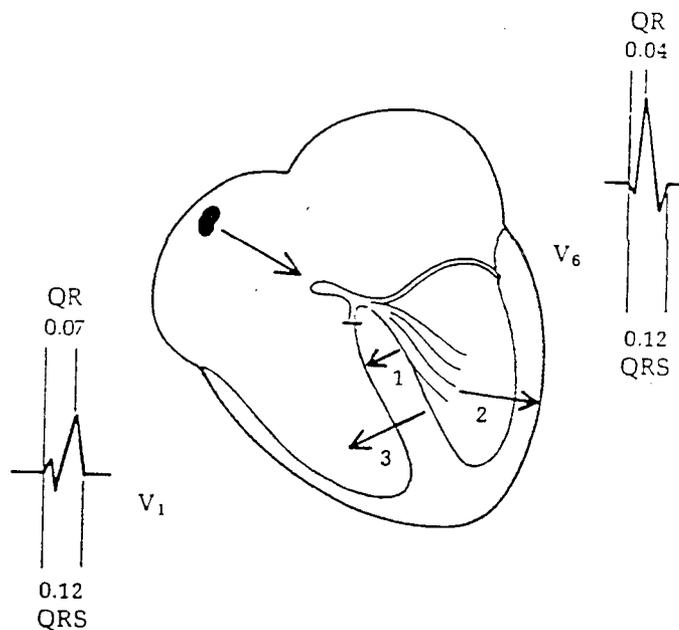
c. ECG recognition

(1) Prolonged QRS duration ( $> .12$  second)

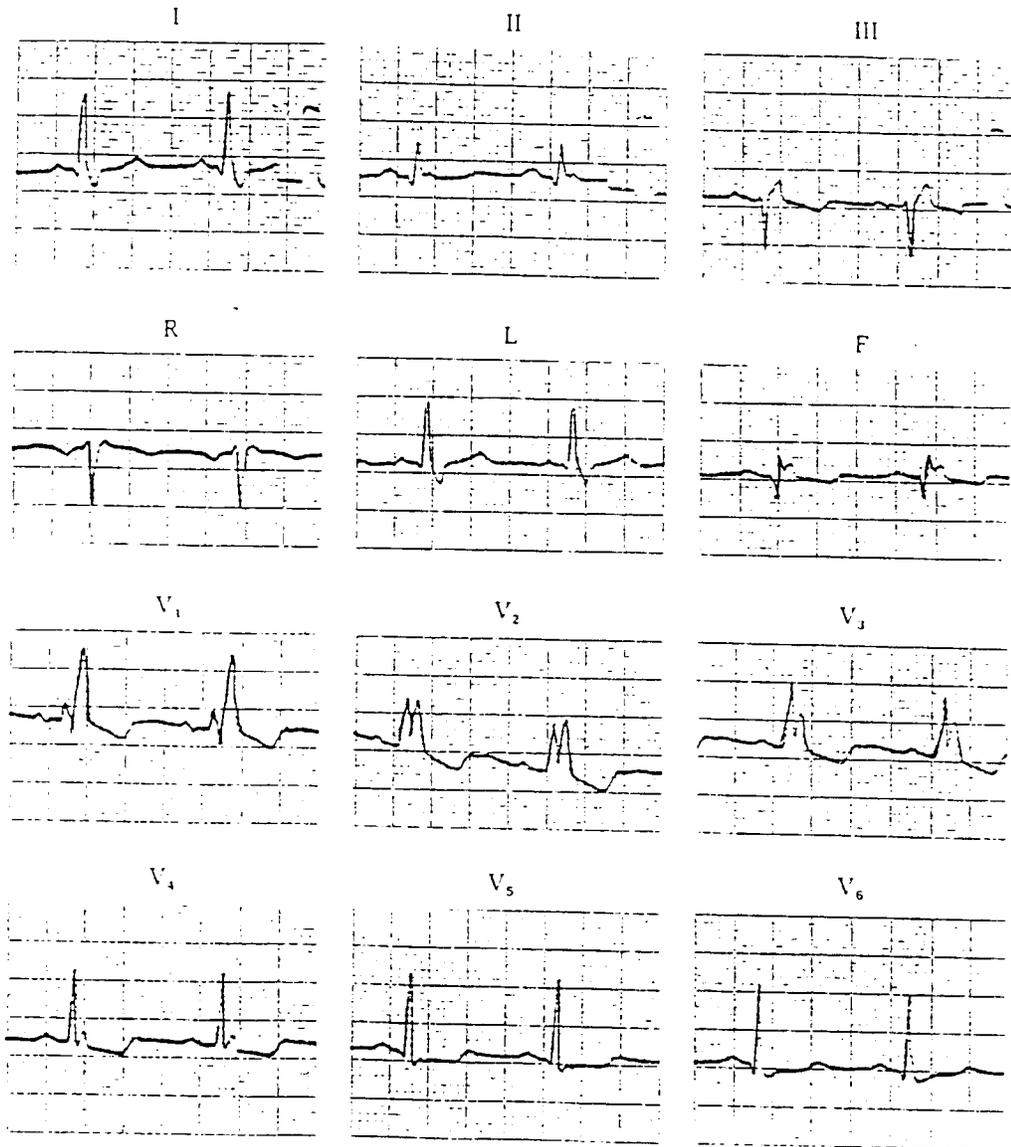
(2) Triphasic complex in  $V_1$  ( $rSR'$ ) and in  $V_6$  (QRS)

(3) T wave changes

(4) Small q and broad S in leads I, aVL,  $V_6$



RBBB as reflected in  $V_1$  and  $V_6$ .



**RBBB.** Note the classical rSR' pattern in V<sub>1</sub>, and the normal delta q waves and broad S waves in I, aVL, and V<sub>6</sub>.

## 7. Left Bundle Branch Block

### a. Mechanism

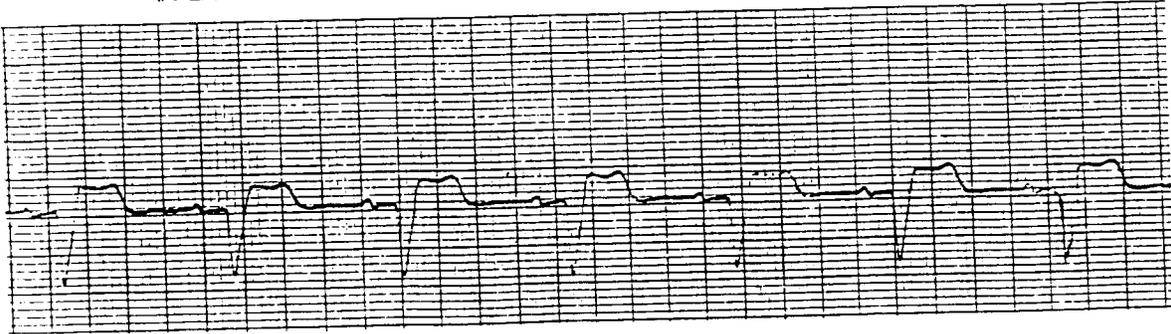
- (1) Block at left bundle, left anterior fascicle, left posterior fascicle
- (2) Conduction proceeds normally down right bundle
- (3) Area normally depolarized by left bundle is depolarized through myocardium

### b. Clinical implications

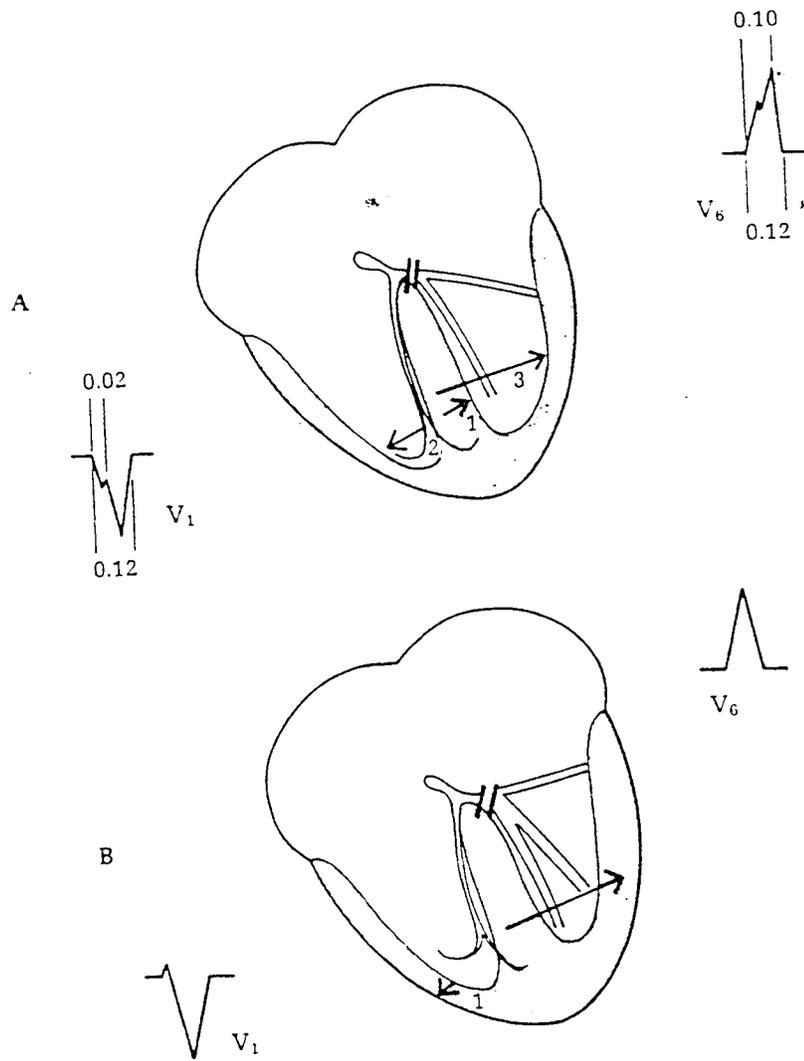
- (1) Usually associated with serious heart disease: hypertensive, ischemic or primary myocardial disease
- (2) Correlates significantly with cardiomegaly and suggests a more serious prognosis

### c. ECG recognition

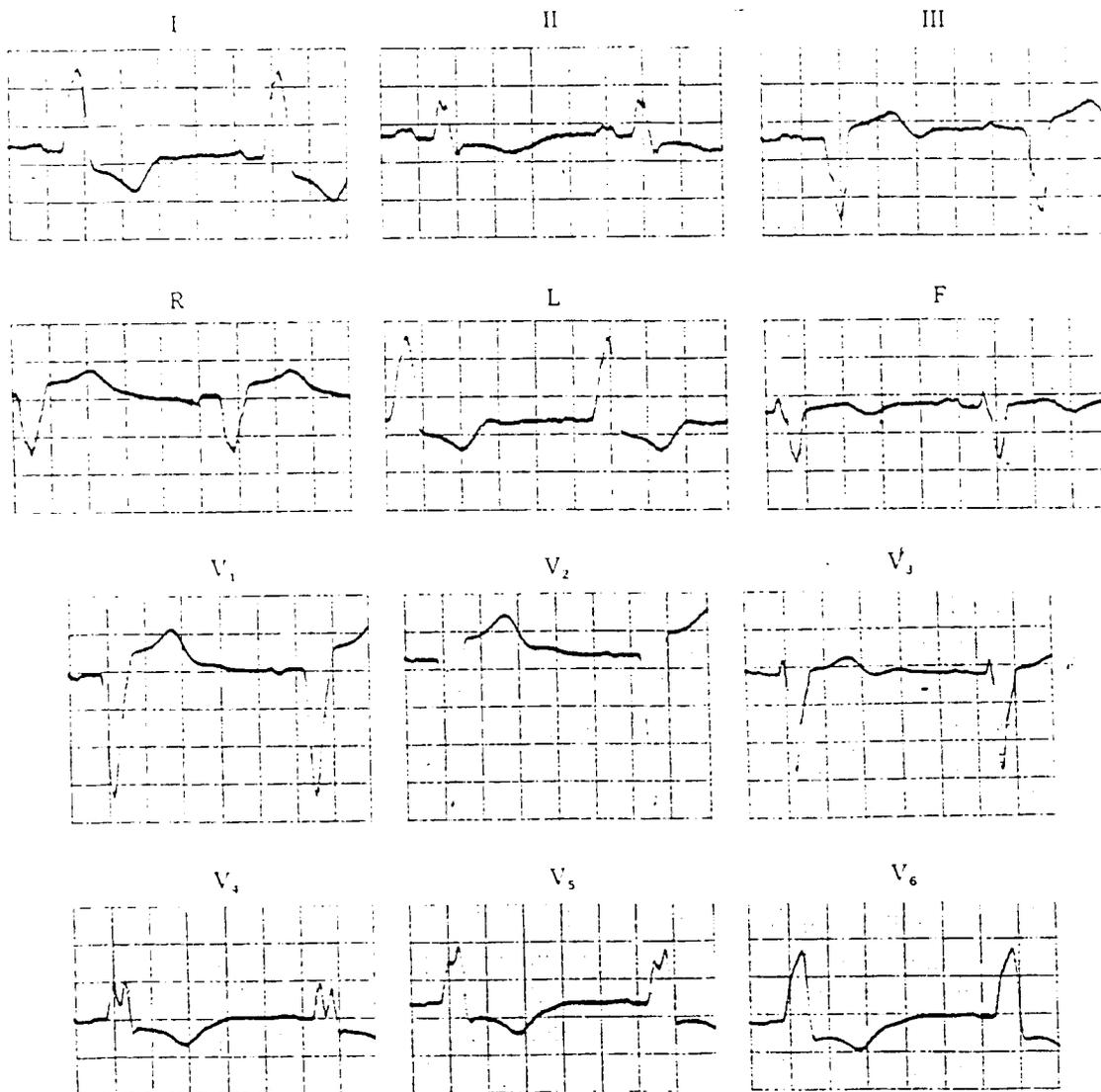
AN EXAMPLE OF LEFT BUNDLE BRANCH BLOCK PATTERN.



- (1) Prolonged QRS duration ( $>0.12$  second)
- (2) Mainly negative QRS complex (QS or rS) in  $V_1$  and always totally positive in  $V_6$



LBBB . Note that  $V_1$  is mainly negative and  $V_6$  totally positive. Neither a q nor an S is present in  $V_6$ . At the more common form of LBBB, without an r wave in  $V_1$ . LBBB with an r wave in  $V_1$ .



C. LBBB. Note that  $V_1$  is mainly negative and  $V_6$  totally positive. Neither a q nor an S is present in  $V_6$ . A, The more common form of LBBB, without an r wave in  $V_1$ . B, LBBB with an r wave in  $V_1$ .

**LBBB.** Note the classical, totally positive patterns in I,  $AV_L$  and  $V_6$ . The T wave has appropriate secondary changes.