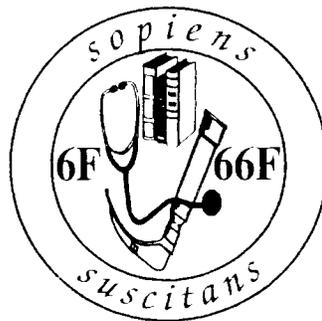


**U.S. Army Graduate Program in
Anesthesia Nursing
6F-66F**



**Critical Care Competencies
Precourse Work**

Critical Care Competencies
Packet Contents

1. Pre-enrollment Verification of Clinical Competencies Checklist
2. Interpretation and Treatment of Cardiac Dysrhythmias Originating in the Atrioventricular Node and Junction
3. Interpretation and Sinus Node Atrial Dysrhythmias
4. Interpretation and Treatment of Cardiac Dysrhythmias Originating in the Ventricle
5. Dysrhythmia Practice
6. Hemodynamic Monitoring
7. Blood Gas Interpretation
8. Mechanical Ventilation

ARMY NURSE CORPS
Pre-enrollment Verification of Clinical Competencies
Anesthesia Nursing Program

TO BE COMPLETED BY THE INDIVIDUAL'S RN CLINICAL SUPERVISOR: The individual named below has demonstrated the knowledge and ability to perform the following nursing activities in the bedside care of critically ill patients and has performed each at least once in the preceding two years. The remainder of the competencies may be evaluated by either observation of direct patient care, return demonstration in a skills lab, or case study analysis.

NAME:	RANK:	AOC:	COMPO: USA	ARNG	USAR
ACTIVITY			INITIALS	DATE	
<u>CARDIOVASCULAR/HEMODYNAMIC:</u>					
1. Able to provide immediate and continual assessment and intervention to stabilize and manage patients with:					
a. Cardiogenic, hypovolemic and septic shock.					
b. Actual or potential life-threatening cardiac dysrhythmias (ventricular tachycardia, ventricular fibrillation, asystole, and complete heart block).					
2. Able to troubleshoot and manage the care of patients requiring the following devices/ interventions:					
a. Continuous EKG monitoring.					
b. Cardiac pacemaker (external, transvenous or permanent).					
c. Invasive arterial pressure monitoring.					
d. Central venous pressure monitoring.					
e. Pulmonary artery pressure monitoring and cardiac output determination.					
f. Fluid resuscitation					
3. Able to describe the indications, expected effects, side effects/adverse effects and demonstrate appropriate administration of the following:					
a. Inotropics (for example, Dopamine and Dobutamine).					
b. Vasodilators (for example, Nitroglycerine and Nitroprusside).					
c. Vasopressors (for example, Levophed or Neosynephrine).					
d. Antiarrhythmics (for example, Lidocaine or Procainamide).					
e. Advanced cardiac Life Support Drugs.					
SIGNATURE				DATE	

(Adapted from 8A Competency Checklist)

CRITICAL CARE NURSING COURSE
BROOKE ARMY MEDICAL CENTER
FORT SAM HOUSTON, TEXAS 78234

Interpretation and Treatment of Cardiac Dysrhythmias
Originating in the Atrioventricular Node and Junction

Objectives

1. Terminal Learning Objective

Formulate a nursing treatment plan for the patient with cardiac dysrhythmias.

2. Enabling Learning Objectives

- a. Discuss the mechanisms which account for the genesis of cardiac dysrhythmias: disturbances of impulse formation, disturbances of impulse conduction; combined disturbances of impulse formation and conduction and fibrillation.
- b. Given the recommended format for interpreting dysrhythmias: identify the common dysrhythmias originating in the A-V Junction, and bundle branch.
- c. Discuss the impossible hemodynamic effects and treatment of the dysrhythmias.
- d. Discuss the nurse's role in continued assessment, monitoring and treatment of each dysrhythmia.

NOTES

A. Characteristics of the Junction

1. Consists of AV node and bundle of His, down to where it begins to branch
2. Sympathetic and parasympathetic innervation
3. Function - slow conduction velocity (1/10th atrial)
 - a. Conduction delay allows more complete atrial emptying
 - b. Protects ventricles from too rapid impulses (AF, A flutter)
4. Regions are divided according to cell types

5. Natural pattern of conduction in AV Junction

a. AN (atrionodal) region

- (1) Thin transitional nodal fibers join the AV node
- (2) Nodal fibers are four times thinner than the atrial fibers they join, which slows conduction
- (3) Found posteriorly (slow pathway) and anteriorly (fast pathway)

b. N (central node) region

- (1) Crisscrossing thin fibers
- (2) Slow A-V conduction

c. NH (nodal - His) region

- (1) Nodal cells join His bundle
- (2) In this region secondary pacing function (junctional rhythm) originates
 - (a) Thought to be two types of junctional escape pacemakers
 - (b) Thought to be focus of escape rhythm
 - (c) Inherent rate 40-60

6. Natural pattern and rate of conduction in A-V Junction can be altered

- a. Aberrant pathways - accelerated conduction in pre-excitation syndrome
- b. Depression of A-V node conduction velocity leads to A-V blocks
- c. Conduction velocity is continually influenced by autonomic nervous system

B. Reentry

1. Types

- a. A-V nodal reentry (45%): reentry involving a microreentry circuit within the A-V node

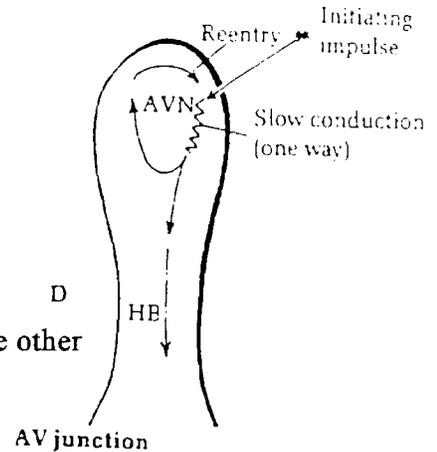
- b. Preexcitation: A-V reentry involving larger reentry circuit using A-V node and an accessory pathway

NOTE: Accessory pathways are abnormal tracts of extra cardiac muscular or conductive tissue between the atria and ventricles.

2. Prerequisites for reentry

- a. Initiating impulse
- b. Unidirectional block
- c. Area of slowed conduction

3. Two separate pathways - impulse travels up one and down the other



C. A-V Nodal Reentry

1. Antegrade conduction down rapid (beta) pathway

2. Retrograde conduction up slow (alpha) pathway

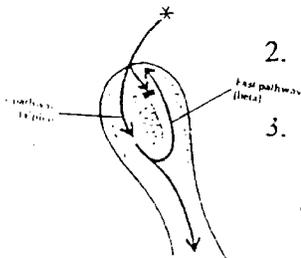
3. Conduction

- a. Early atrial beat --> impulse is conducted down the alpha pathway which has short refractory period to ventricles via normal pathways and back to atria via the beta pathway with long refractory period
- b. In A-V nodal reentry, QRS is narrow and aberrancy uncommon
- c. Initiating P-R is long (result of conduction down alpha pathway)
- d. P' wave during tachycardia buried in QRS or seen at end of QRS
- e. Retrograde P (negative in II, III, and aVF)
- f. Conduction ratio usually 1P:1 QRS

4. Treatment

- a. Drug therapy

(1) Digoxin



- (2) Beta-blockers (propranolol, loproressor, breviblock)
- (3) Calcium channel blockers
- (4) Procainamide
- (5) Quinidine
- (6) Other drugs
 - (a) Cholinergic (tensilon)
 - (b) Pressor drugs (Neosynephrine)
- b. Vagal stimulation - causes a lengthening of refractory period in A-V node and interrupts the circle of conduction
 - (1) Carotid sinus massage, gagging, coughing, valsalva
 - (2) Release of acetylcholine that blocks A-V node and terminates A-V nodal reentry and PSVT

NOTE: Vagal maneuvers should be reattempted after each drug therapy.

- c. Carotid sinus massage
 - (1) Manuever performed at the angle of the jaw
 - (2) Elevate BP in CS to achieve slowing of A-V conduction
 - (3) Cautions
 - (a) Carotid sinus pressure for longer than 3 seconds may be generous
 - (b) Take care in patients over 65 years of age; long sinus pauses may result (3 and even 6 seconds have been reported)
 - (c) Prior to the maneuver listen for carotid bruits, and take a history that will reveal any transient ischemic attacks
 - (d) Never perform carotid sinus stimulatín without ECG monitoring; the results must be observed on the ECG and responded to if necessary
- d. DC cardioversion

e. Atrial pacing

C. Rhythms That Originate in the A-V Junction

1. Mechanism

- a. Triggered activity (digitalis toxicity or catecholamine excess)
- b. Altered automaticity (myocardial stretch, ischemia or hypokalemia)
- c. Reentry

2. Clinical implications depend on the rhythm and the individual

3. Premature junctional contraction/complex

- a. Mechanism: altered automaticity
- b. Originates in AV Junction usually in NH region.
- c. Atrial depolarization is due to retrograde conduction
- d. Clinical significance
 - (1) May be insignificant
 - (2) May be early warning of myocardial irritability

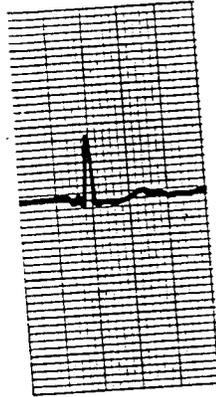
Premature Junctional Contraction (PJC)



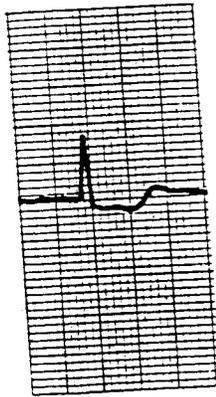
c. ECG characteristics

- (1) Rate - depends on underlying rhythm
- (2) Rhythm - irregular because of premature beat(s)
- (3) Conduction

- (a) Normally down to ventricles
- (b) Retrograde to atria
- (c) P waves may be inverted

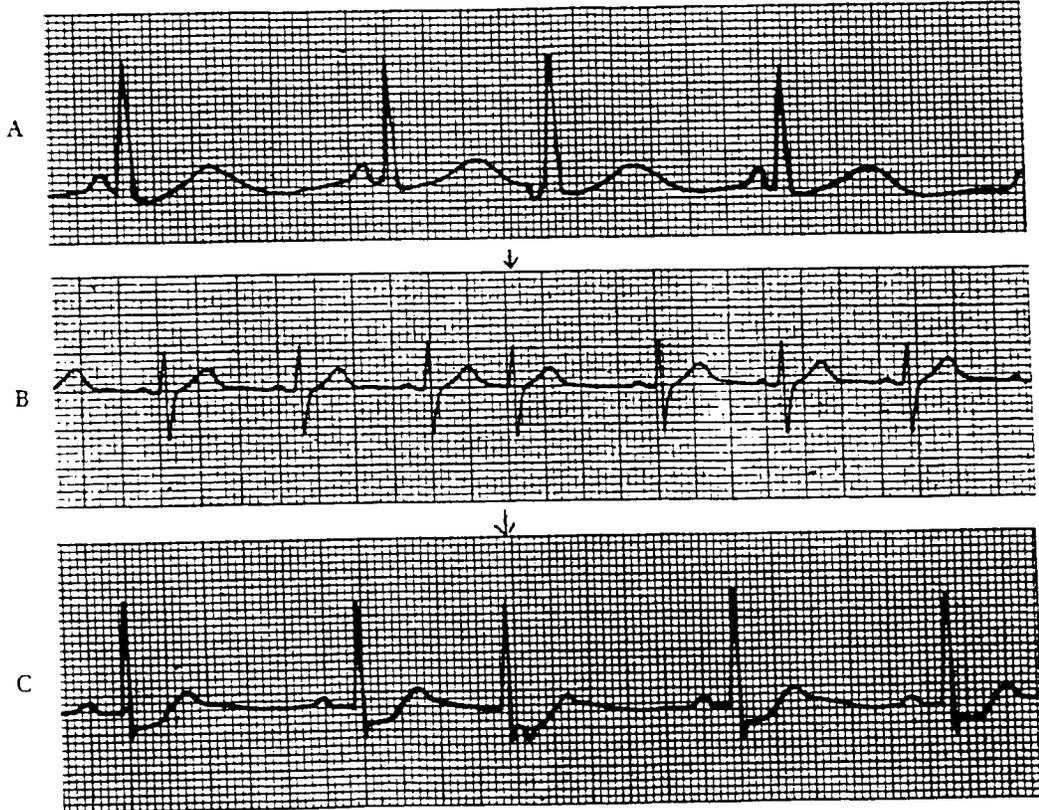
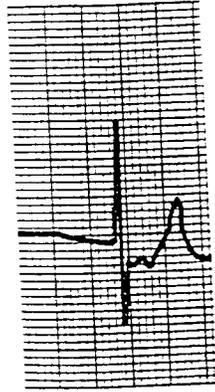


- 1) May precede QRS if atria depolarized before ventricles
PRI $< .12$ seconds
- 2) May be hidden in QRS if atria and ventricles are depolarized simultaneously. PRI not measured.



- 3) May follow QRS if ventricles are depolarized before atria.
PRI not measured.

4) QRS - usually normal in shape if no BBB



junctional premature beats.

d. Etiology - irritable focus within nodal His region

e. Treatment

(1) Usually none

(2) Treat underlying process

(3) Remove stimulants - CNS medications, alcohol, caffeine, tobacco

f. Nursing concerns

(1) Observe for frequency progression

(2) Assess patient for symptoms

4. Junctional escape rhythm

a. Mechanism - failure of higher pacemaker (sinus or atrial) to produce impulse

b. Junctional escape beats that follow one after another

c. Clinical significance - may be lifesaving protection against excessive slowing

d. Underlying bradycardia or pauses present

e. ECG characteristics

(1) Rate: 35-60 beats per minute

(2) Rhythm: Regular

(3) Atrial conduction: P wave inverted in Lead II

(4) Ventricular conduction: QRS complex normal or same shape as sinus conducted beats

f. Etiology - may be secondary to ischemia, electrolyte or drug suppression of sinus rhythm

g. Treatment

(1) Withdraw the suppression of the sinus node and atria

(2) Atropine if symptomatic

NOTE: Atropine sulfate may cause conversion to sinus rhythm or may increase rate of junctional rhythm.

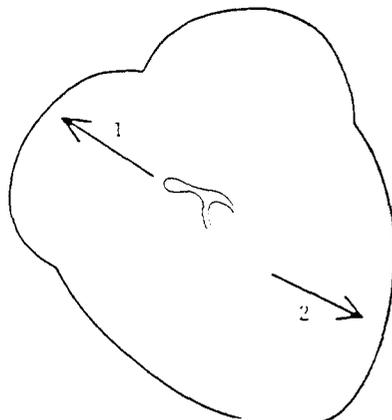
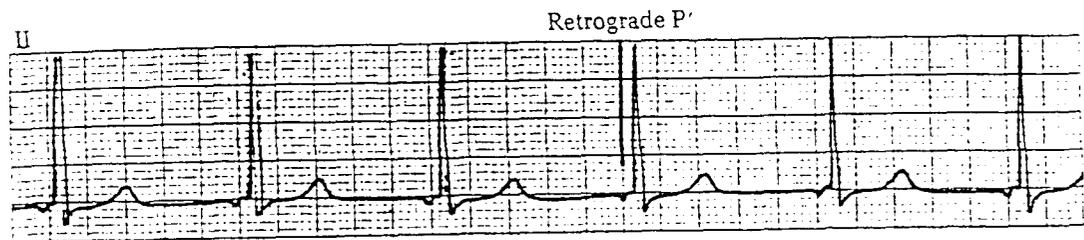
(3) Temporary pacemaker is symptomatic

(4) Permanent pacemaker if little chance of regaining normal rhythm at a normal rate

h. Nursing concerns

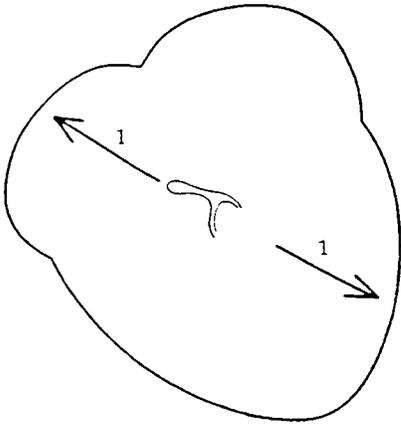
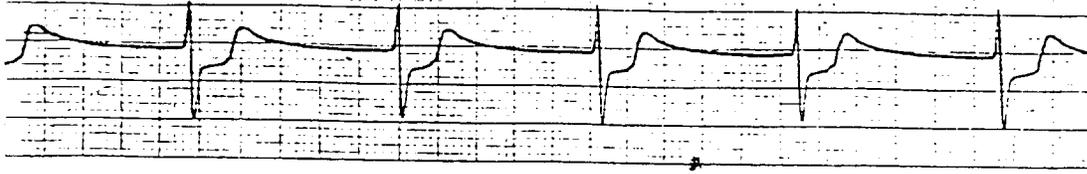
(1) Observe, document rhythm strip, 12 lead ECG

(2) Assess patient for symptoms of ↓ CO, ↓ tissue perfusion
(check BP, mentation, lung sounds)



Junctional escape rhythm (60 beats/min).
Atrial activation precedes ventricular activation.

II



Junctional escape rhythm (57 beats/min).
Atrial and ventricular activations occur simultaneously.

5. Junctional tachycardia

a. Mechanism

(1) Triggered automaticity

(2) Usurps sinus rhythm

b. Clinical significance - may cause hemodynamic compromise
(\downarrow CO, \downarrow tissue perfusion) angina, CHF

c. ECG characteristics

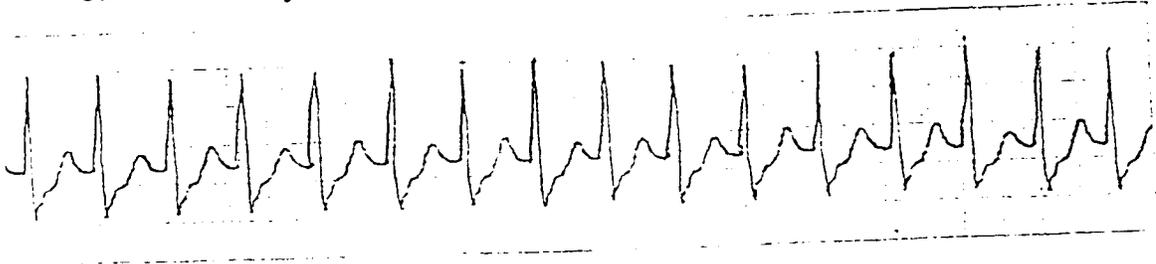
(1) Rate: Exceeds 100 beats per minute

(2) Rhythm: Regular

(3) Origin: NH region

(4) Conduction: Same as junctional rhythms

Junctional Tachycardia



d. Etiology

- (1) Occurs most commonly in patients who have underlying heart disease (inferior MI, rheumatic myocarditis, following open heart surgery)
- (2) Digitalis toxicity

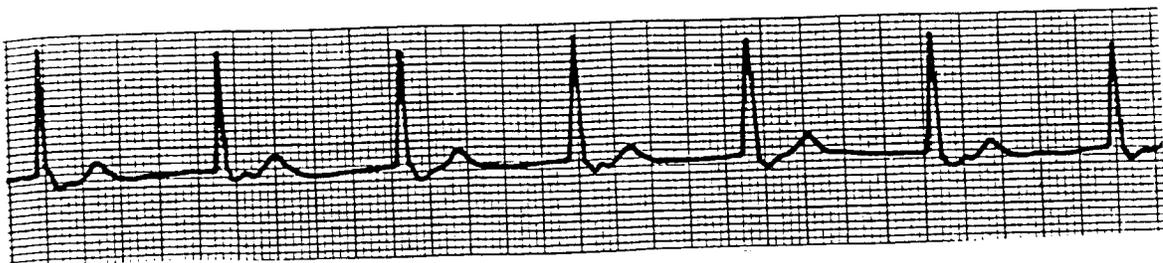
e. Treatment

- (1) Withhold digoxin if digitalis toxic
- (2) Calcium channel blockers - most successful
- (3) Digoxin - if patient is not on digoxin and is not digitalis toxic
- (4) Adenosine
- (5) DC cardioversion
- (6) Atrial pacemaker

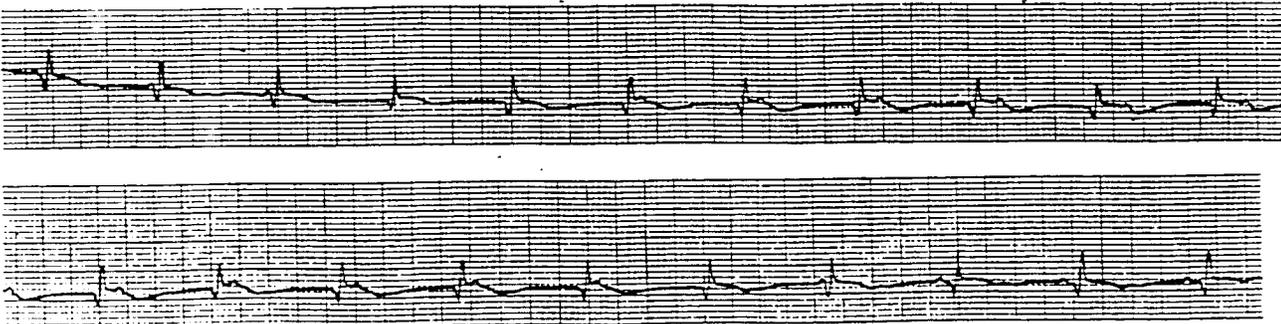
f. Nursing concerns

- (1) Assess patient response to rhythm
- (2) Monitor for CO, tissue perfusion (CHF, BP, angina)
- (3) Assess patient response to treatment

6. Accelerated junctional rhythm



- a. Mechanism: Same as junctional tachycardia
- b. Clinical significance - junctional pacemaker taking over from SA node
- c. ECG characteristics
 - (1) Rate: 60 to 100 beats per minute
 - (2) Rhythm: Regular
 - (3) Origin: NH region
 - (4) Conduction: Same as junctional rhythm
 - (a) If retrograde conduction to atria is blocked, rhythm is termed accelerated idiojunctional in which case there is A-V dissociation; may compete with sinus rhythm
 - (b) Narrow QRS

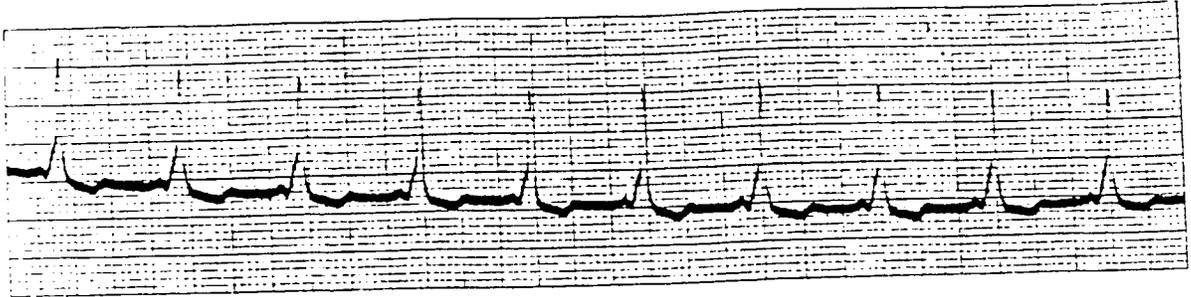


Accelerated junctional rhythm (74 beats/min) with AV dissociation.

- d. Etiology - frequently seen with digitalis toxicity; MI
- e. Treatment - essentially same as junctional tachycardia
 - (1) May increase sinus rate if loss of atrial kick is hemodynamically significant
 - (2) Removal of precipitating factors (digitalis or catecholamines)
- f. Nursing concerns - essentially same as junctional tachycardia

D. Preexcitation Syndromes

I. Wolff-Parkinson-White syndrome - accessory A-V connection (Kent bundles)



NOTE: The bundles of Kent are conduction pathways located along the sides of the heart and connect the atria directly to the ventricles. Usually becomes non functional 24 hours after birth.

a. Accessory A-V connection (Kent bundles)

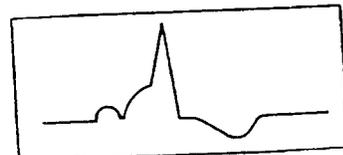
- (1) Single or multiple pathways
- (2) Active or inactive
- (3) May conduct anterograde and retrograde or only retrograde
- (4) WPW Type A - left bundle is functional. QRS predominantly upright
- (5) WPW Type B - right bundle remains functional QRS negative

b. Diagnosis from ECG

- (1) Surface ECG in sinus rhythm 60-70% (overt WPW syndrome)
- (2) Surface ECG only during tachycardia (latent or nonevident WPW)

c. ECG in overt WPW

- (1) Short PR interval $< .12$ second-result of accelerated conduction across an accessory pathway
- (2) Delta wave - appearance of broad QRS $> .10$ second; initial slurring of the QRS complex - result of conduction arriving at the ventricles via accessory pathway



(3) S-T wave changes. Depolarization does not follow normal sequence
repolarization process may also be out of sequence

d. ECG in concealed WPW - no ECG signs during sinus rhythm when
the accessory pathway conducts only in a retrograde direction

(1) ECG normal during sinus rhythm

(2) Tendency to develop PSVT because of retrograde conduction in
accessory pathway

NOTE: In 24% of patients with PSVT and no ECG signs of preexcitation, EP
(electrophysiologic) studies demonstrate concealed accessory A-V
connection forming the retrograde limb of reentry pathway.

