

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

Interpretation of Sinus Node Atrial Dysrhythmias

Objectives

1. Terminal Learning Objective

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

2. Enabling Learning Objectives

a. Discuss the mechanisms for the genesis of cardiac dysrhythmias.

b. Identify, interpret and discuss specific dysrhythmias originating in SA node or atria to include rate, rhythm, combined rate and origin, treatment and nursing concerns.

NOTES

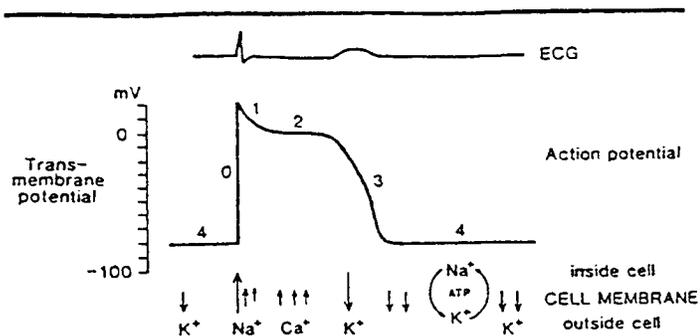
A. Mechanisms for Dysrhythmia Formation

1. Definition

a. Arrhythmias or dysrhythmias are defined as alterations in the heart's rate, rhythm, or conduction sequence

b. Results from disturbances in automaticity or conductivity

2. Altered automaticity is divided into two types - enhanced normal automaticity and abnormal automaticity



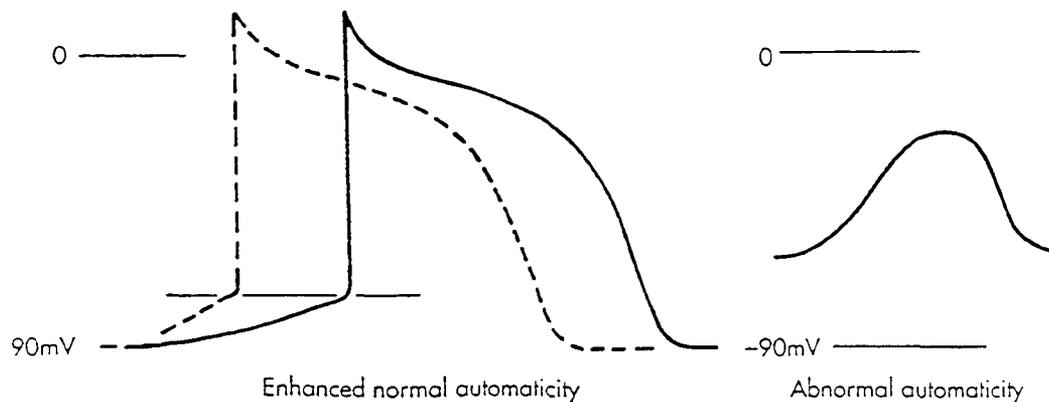
Schematic representation of ventricular myocardial working cell action potential. Arrows indicate times of major ionic movement across cell membrane.

a. Normal

- (1) Pacemaker cells possess property of self-excitation (automaticity)
- (2) Unstable membrane potential during phase 4
- (3) Disturbances in automaticity result from alterations in the slope of Phase 4 of pacemaker cells in SA node, atria, junction, or ventricles

b. Enhanced normal automaticity may occur in HIS- Purkinje fibers that are functioning normally

- (1) Increased slope of phase 4 secondary to catecholamines can cause increase of firing rate
  - (a) Increased automaticity of SA node produces sinus tachycardia
  - (b) Increased automaticity of ectopic pacemaker cells produce premature beats and tachycardias
- (2) Factors which increase the slope of phase 4 include
  - (a) Hypoxia
  - (b) Hyperthermia
  - (c) Drugs - atropine, isuprel and adrenalin
- (3) May be treated with overdrive pacing



c. Abnormal automaticity

(1) Spontaneous firing secondary and ischemia or electrolyte imbalance

(2) Occurs in myocardial fibers with abnormally reduced membrane potentials

(a) The result of increase in extracellular potassium or decrease in intracellular potassium

(b) An increase in sodium permeability or decrease in potassium permeability

(3) Clinical condition/factors

(a) Ischemia

(b) Infarction

(c) Hypokalemia

(d) Hypocalcemia

(e) Cardiomyopathy

3. Conductivity

a. Ability of cardiac cells to transmit a stimulus from cell to cell

a. Impulses from SA node or atria can be delayed or completely blocked, allowing escape beats or escape rhythms to emerge

b. Slow ventricular rates and wide QRS complexes characterize many conduction disturbances

4. Re-entry

a. Repeated reactivation of fibers by the same impulse

b. Produces tachycardias and premature beats as the same electrical impulse depolarizes the same chamber more than once

c. Occurs most commonly in ischemic or injured myocardial tissue

d. Conditions necessary for re-entry

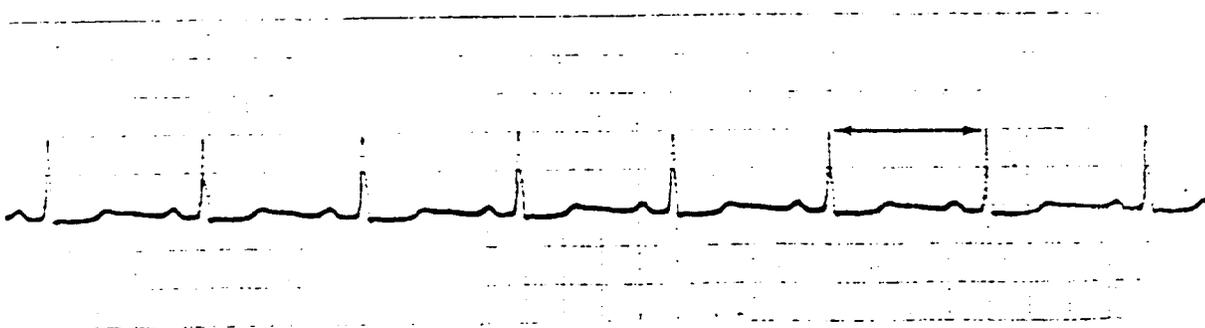
- (1) Initiating impulse, either normal sinus or ectopic
- (2) An area of slow conduction that is sufficiently long so that the impulse passing through is still active when the rest of the myocardium has become nonrefractory
- (3) One-way conduction, without which the impulse would cancel itself out without the area of slow conduction

e. Occurs in atrial, junctional and ventricular tissues

B. Identification and Interpretation of Dysrhythmias

I. Disorders of Rate

a. Normal sinus rhythm



- (1) Rate - 60 to 100 beats per minute (may have very slight variation and still qualify)
- (2) Rhythm - regular
- (3) Origin - sinus node; P wave configuration is normal
- (4) Conduction
  - (a) P wave precedes each QRS. Upright in Leads I, II, AV7, V4, V5, V6. Negative in lead AVR. In Leads III, AVL, V1, V2, V3 P waves can be positive, negative or biphasic
  - (b) PR interval 0.12-.20, QRS configuration is normal

(5) Etiology: normal

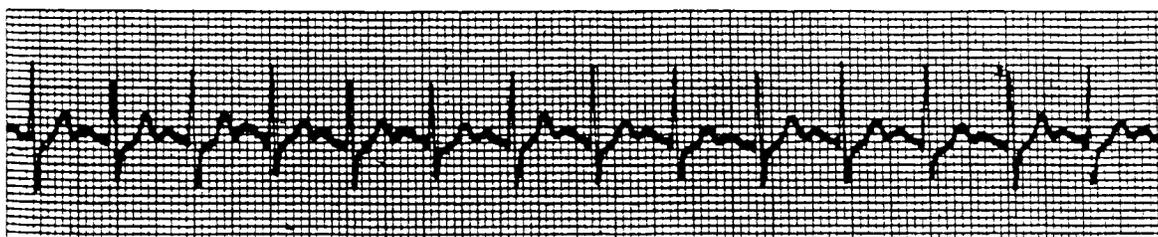
(6) Treatment: none

(7) Pediatrics

(a) Normal heart rate in the newborn - 110-200 bpm

(b) The younger the age the faster the rate

b. Sinus tachycardia - acceleration rhythm



(1) 100-180 beats per minute in adults; greater than 200 bpm in infant, 140-100 bpm in child

(2) Rhythm - usually regular/may be slightly irregular

(3) Origin

(a) Sinus node

(b) P wave configuration - normal or may be peaked

(4) Conduction

(a) PR may shorten because AV node control is the same SA node with increased heart rate, will have shortening of AV conduction

(b) QRS normal or prolonged. IF QRS is prolonged, usually due to a refractory conduction pathway

(5) Etiology

(a) Physiologic response to stress

(b) Response to some underlying pathology or pharmacology

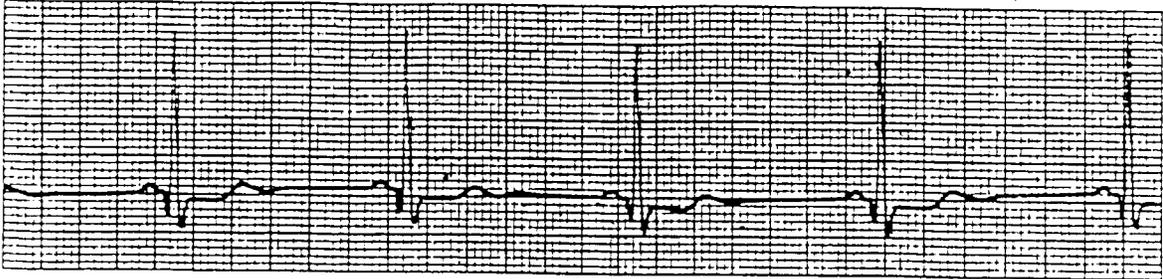
- (c) Increased metabolic demands (fever, hyperthyroidism, pain)
  - (d) Hyperflow state (anemia, heat)
  - (e) Diminished blood flow (shock, MI, CHF, pulmonary embolism)
  - (f) Hypoxia
  - (g) Emotional excitement
  - (h) Pharmacology - atropine, adrenergic drugs, exogenous thyroid, alcohol, nicotine and caffeine, aminophylline
- (6) Treatment - (symptomatic)
- (a) Directed toward underlying factor (i.e. hypovolemia, anemia, fever)
  - (b) Rest
  - (c) If heart rate itself is deleterious (i.e., angina, acute MI), beta blockers are usually the best form of therapy.
  - (d) Vagal stimulation - valsalva, cough, carotid massage

NOTE: If drug therapy is used to slow reflex tachycardia (hypovolemia) or compensatory tachycardia (LV dysfunction) it may precipitate profound hypotension or heart failure.

(7) Nursing concerns

- (a) Identify the arrhythmia - document with strip
- (b) Assess hemodynamic effect
- (c) Assess patient for possible cause of arrhythmia
- (d) Monitor treatment modalities for side effects
- (e) Alleviate anxiety of patient/family

c. Sinus bradycardia - deceleration rhythm



- (1) Rate - less than 60 beats per minute. Transient sinus bradycardia may be seen in normal premature infants. Rare in normal healthy children.
- (2) Rhythm - regular
- (3) Origin - sinus node, P waves are normal
- (4) Conduction - normal
  - (a) PR may be slightly lengthened. Greater than 0.12 seconds
  - (b) QRS normal
- (5) Etiology
  - (a) Excessive vagal or decreased sympathetic tone (vomiting, carotid massage, valsalva maneuver, orbital pressure, nose blowing)
  - (b) Parasympathetic center stimulation (increased intracranial pressure)
  - (c) Decreased metabolic demand (sleep, hypothermia)
  - (d) Physical conditioning (young adults, especially athletes)
  - (e) Pharmacology - beta blockers, digoxin, calcium channel blockers

NOTE: May be desirable - due to decrease in myocardial oxygen demand.

- (f) If slow enough, another pacemaker at another site might take over

(6) Treatment

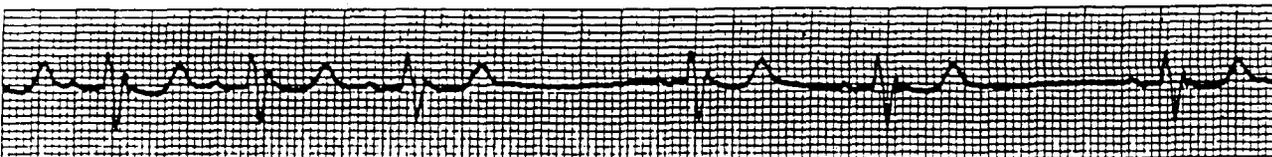
- (a) Usually none. Treatment should be associated with symptoms (angina, syncope, heart failure, hypotension, ventricular arrhythmias)
- (b) If cardiac output low or other dysrhythmias occur, atropine 0.6-2.0 mcg IV or 0.04 mg/kg/body weight, Isoproterenol 2-20 mcg/min IV.
- (c) Atrial pacing is preferred over ventricular pacing in the absence of AV block to preserve AV synchrony
- (d) Permanent pacemaker for patients with CHF or with chronic sinus bradycardia associated with low cardiac output

(7) Nursing concerns

- (a) Document arrhythmia as sinus bradycardia - assure it is not block or nodal rhythm
- (b) Frequent vital signs, especially pulse, BP
- (c) Assess for signs/symptoms LV function
- (d) Monitor for PVC's, escape beats

NOTE: Sinus arrhythmia (P-P interval varying over 0.16 sec) may co-exist with sinus bradycardia.

d. Sick sinus syndrome (tachy-brady syndrome) or sinus nodal dysfunction



- (1). Rate: Characteristic is inappropriate rate response
  - (a) Broad range of conduction abnormalities and arrhythmias which may occur alone or in combination.
  - (b) Pediatrics: seen in children post-op cardiac surgery
  - (c) Marked, symptomatic or inappropriate bradycardia due to a junctional escape rhythm, sinus arrest or SA block. Can result in syncope.

- (d) Marked, symptomatic or inappropriate sinus arrhythmia
- (e) Recurring atrial fibrillation with slow ventricular response not due to drugs
- (f) Classic bradycardia - tachycardia syndrome. Tachycardia phase may be paroxysmal A-fibrillation, atrial flutter
- (g) Transition show long pauses due to suppressed SA node

NOTE: SA nodal reentry can be sustained in either SA or AV node.

- (2) Rhythm - irregular
- (3) Origin - sinus node
- (4) Conduction - normal or variations related to fast rate
- (5) Etiology - due to diseases (ischemic, inflammatory, metabolic) which cause interruption or degeneration of sinoatrial and AV conduction system. SA node may be partially or totally destroyed.
  - (a) Abnormalities in automaticity or conduction
  - (b) Autonomic nervous system dysfunction
  - (c) Endocrine dysfunction
  - (d) Decreased blood supply to conduction system
  - (e) Myocardial muscle diseases/dysfunction
  - (f) Pericardial disease
  - (g) Connective tissue diseases
  - (h) Other: Amyloidosis, diffuse fibrosis, infection
  - (i) Drugs - digitalis toxicity, amiodarone

(6) Treatment

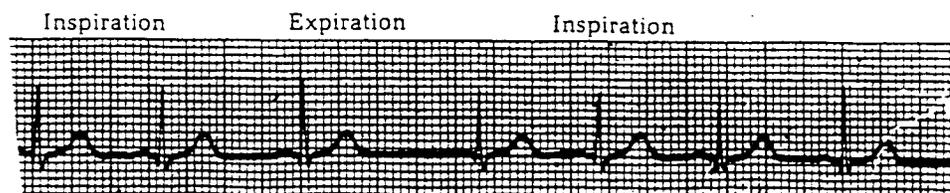
- (a) Permanent demand pacemaker - dual chamber or atrial pacing is preferred to ventricular pacing alone (cause lower incidence of stroke, A-fibrillation and death)
- (b) Chronotropic support with exertional intolerance
- (c) AV nodal blocking agents for atrial arrhythmias (i.e., Digoxin, Verapamil). Drug therapy for atrial tachycardias may exaggerate underlying conduction abnormalities, precipitate symptomatic bradyarrhythmias and necessitate permanent pacing.
- (d) Consider long term anticoagulation

(7) Nursing concerns

- (a) Document dysrhythmia
- (b) Patient may have cerebral dysfunction secondary to sinus node dysfunction and failure of escape rhythms-document
- (c) Document hemodynamic effects/symptoms: frequent vital signs
- (d) Check for drugs which may be causing dysrhythmias
- (e) Teaching for probable permanent pacemaker
- (f) Temporary pacemaker if markedly symptomatic

2. Disorders of Rhythm

a. Sinus arrhythmia



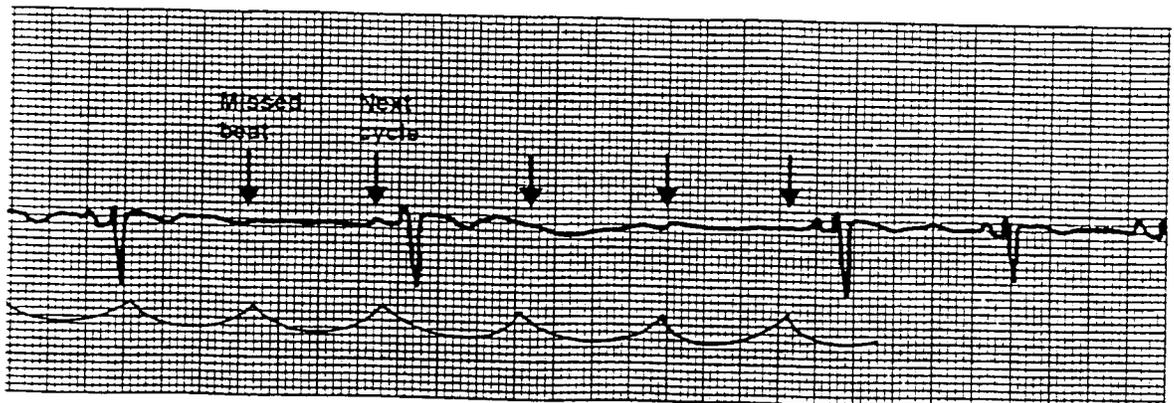
(1) Rate

- (a) Variation in R-R interval
- (b) Alternately increasing with inspiration and decreasing with expiration

(c) Pediatric: Rare in young infant, common in children and adolescents.  
Difference between the longest and shortest PP interval is 0.26 seconds.

- (2) Rhythm - irregular 2° acceleration/deceleration of HR
- (3) Origin - sinus node
- (4) Conduction - normal. Present when the difference between the shortest PP interval and longest PP interval is greater than 0.12 seconds
- (5) Etiology
  - (a) Most frequent dysrhythmia but a normal phenomenon, asymptomatic
  - (b) Respiratory effect on sinus rhythm is an autonomic response
  - (c) Sinus arrhythmia indicative of ↓ sympathetic tone, (change in abdominal tone changes vagus stimulation) relaxation, occurs in sleep since parasympathetic activity predominates
- (6) Treatment - only if symptomatic in bradycardic phase
- (7) Nursing concerns
  - (a) Document as sinus arrhythmia when the difference between the shortest PP interval and the longest PP interval is greater than 0.12 sec
  - (b) Be sure it's not a more serious arrhythmia
  - (c) Monitor

b. Sinus pause and arrest - failure of impulse formation in the SA node



In sinoatrial arrest, there is a missed beat and the next beat returns out of cycle. The rate after the missed beat is different from the rate of the pattern before the missed beat.

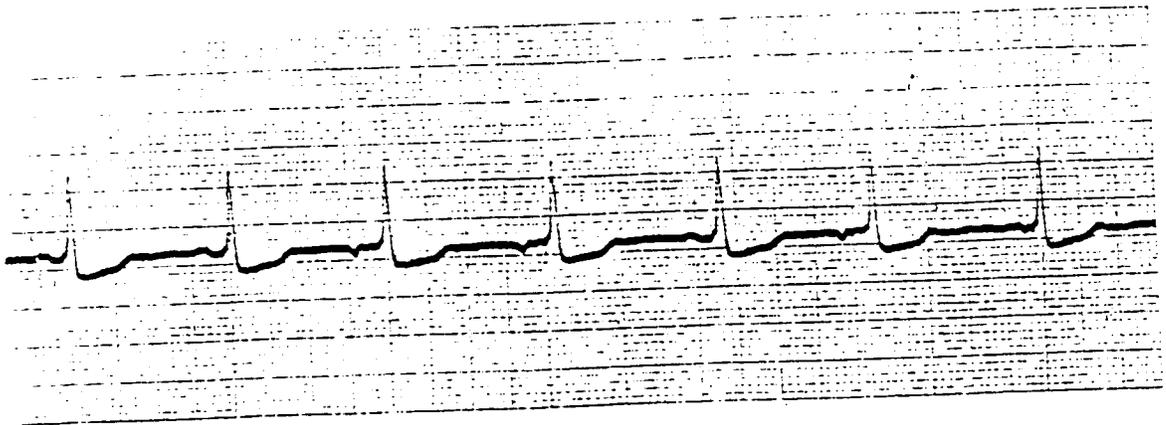
- (1) Rate - varies - may be marked bradycardia because of long pauses because back up pacemakers did not take over pacing functions
- (2) Rhythm
  - (a) Pause - momentary cessation of impulse formation with renewed normal cadence
  - (b) Arrest - prolonged failure of sinus node to initiate impulse greater than 3 seconds. Arrest may produce pauses 3-9 seconds or longer.

NOTE: The rhythm is - irregular, with periods of "asystole", pause length unpredictable.

- (c) Other latent pacemakers may take over to prevent ventricular asystole (escape beats or rhythms)
- (3) Origin - sinus node. PP interval may be fixed before and after the pauses,
- (4) Conduction - PR interval normal and fixed unless AV conduction problem is present
- (5) Etiology
  - (a) Myocardial infarction (especially with SA node or SA node artery involvement)
  - (b) Excessive vagal tone/stimulation
  - (c) Drugs: digitalis toxicity (↓ conduction), quinidine, procainamide
- (6) Treatment - symptomatic
  - (a) Increase sympathetic tone - isoproterenol
  - (b) Decrease parasympathetic tone - atropine
  - (c) Atrial or ventricular pacing
- (7) Nursing concerns
  - (a) Document with rhythm strip

- (b) Hold digoxin or quinidine when patients on these two drugs show sinus arrest - notify MD. Check digoxin level.
- (c) Monitor for escape beats
- (d) Prepare patient for possible pacemaker

c. Wandering pacemaker



- (1) Rate - may vary, each pacemaker will have its own inherent rate. Rate less than 100 bpm
- (2) Rhythm - slightly irregular based on pacemaker
- (3) Origin
  - (a) Impulse formation shifts from focus to focus within atria, SA node, or A-V node
  - (b) Shift of dominant pacemaker from sinus node to atrial or AV junctional sites may be normal
- (4) Conduction
  - (a) P wave morphology changes with site of origin. Have sinus and nonsinus P wave
  - (b) P-R may change, can be less than 0.12 seconds
  - (c) QRS normal