

(5) Etiology

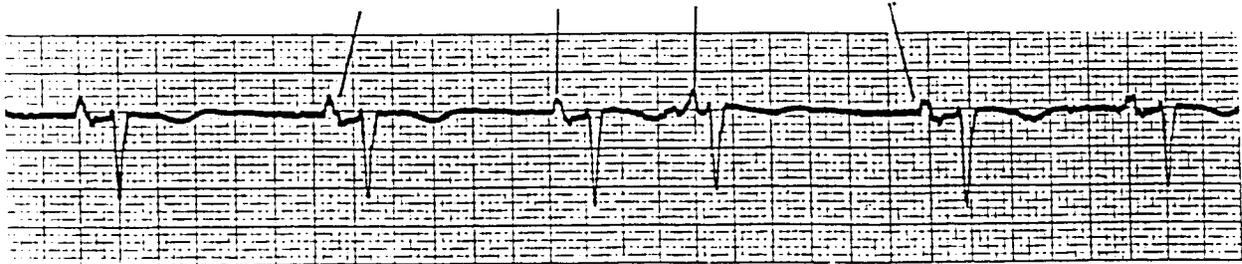
- (a) Ischemic heart disease
- (b) Inflammatory disease (rheumatic fever)

(6) Treatment - usually none - treatment same as in bradycardia. Give vagotonic drugs carefully

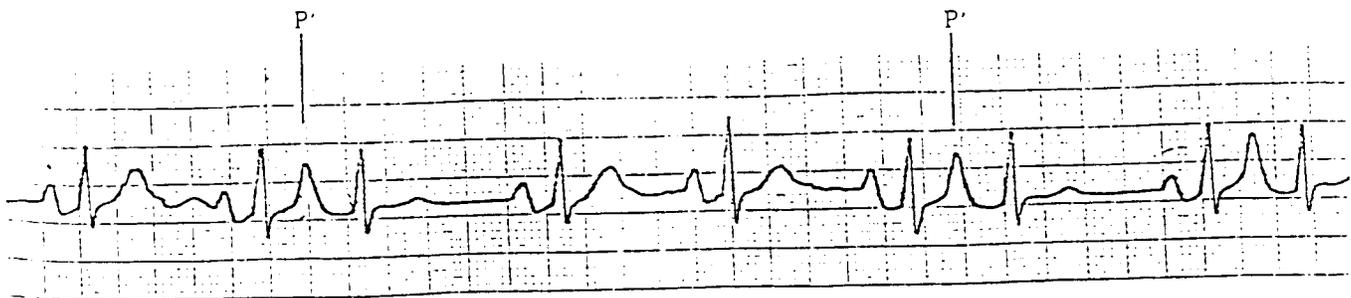
(7) Nursing concerns

- (a) Document with rhythm strip
- (b) Monitor to ascertain that sinus node is still working
- (c) Possibly digoxin induced - monitor digoxin levels

d. Premature atrial contraction - atrial impulse that is premature to next expected sinus beat



The fourth P wave is a PAC.

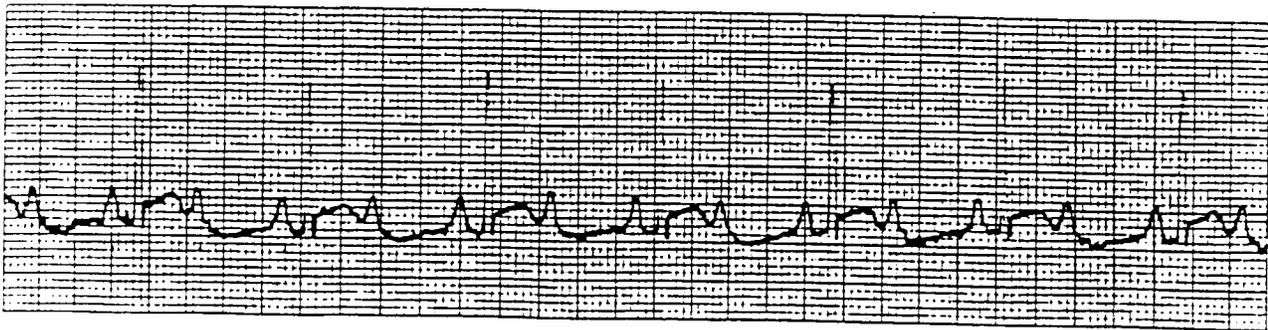


PACs hidden in T waves.

(1) Rate - underlying rate may be normal, premature beat is early

- (2) Rhythm - irregular because of PAC's
- (3) Origin
 - (a) Atria excited and form impulses more rapidly than from SA node. Ectopic beat occurs early
 - (b) Significance
 - 1) May occur normally
 - 2) May precede more serious atrial dysrhythmias
- (4) Conduction
 - (a) The PAC has different morphology from sinus P wave. Different focus, different shape. May be inverted.
 - (b) Ectopic P wave is called P prime wave (P')
 - (c) P' R interval may be same as PR interval. May be prolonged due to prematurity of P wave.
 - (d) QRS and T normal or T peaked if P buried in it
 - (5) May not have full compensatory pause
 - (6) P may be conducted normally to ventricles (narrow QRS)
 - (7) PAC occurs very early - may not conduct - ventricles are still refractory and won't respond
- (5) Etiology
 - (a) Occur as a response to emotional disturbances, tobacco, tea, or coffee, stress, fatigue
 - (b) Digitalis produced
 - (c) Can result from organic heart disease, cor pulmonale
 - (d) Often a precursor to paroxysmal atrial tachycardia (PAT), atrial flutter or atrial fibrillation

- (e) Electrolyte imbalances (hypokalemia, hypomagnesium)
- (6) Treatment - treat cause - withdraw the tobacco, caffeine or upsetting circumstance
- (7) Nursing concerns
 - (a) Document PAC's and frequency
 - (b) Ascertain if other cause of irregularity present, i.e., A-fibrillation
 - (c) Monitor for serious arrhythmia that may develop, i.e., PAT, A-fibrillation
 - (d) Treat electrolyte deficit
- 3. Combined Rate and origin
 - a. Atrial tachycardia - combined rate/origin



Atrial tachycardia with 2:1 AV conduction.

- (1) Rate - 130-250 beats per minute.
 - (a) Usually less than 200 bpm if cause is digitalis toxicity
 - (b) May be paroxysmal or sustained
- (2) Rhythm - regular if no block is present
- (3) Origin - atria, may follow PAC's or begin abruptly with no identifiable cause
- (4) Conduction

- (a) Normal response of AV node to rapid atrial ectopic rhythm is to block
- (b) P waves aberrant. Morphology differs from sinus P wave except with digitalis toxicity.
- (c) QRS normal if no intraventricular conduction defect
- (d) 2:1: A-V conduction frequent

(5) Etiology

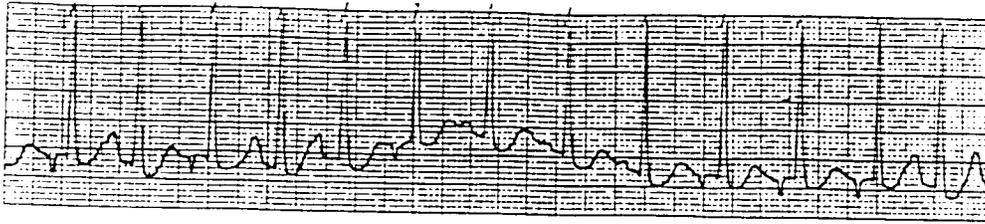
- (a) Abnormal automaticity for sustained atrial tachycardia
- (b) Pediatrics: More common in pediatric patients with cardiac tumors or aneurysms. Rates in children are usually slower than in adults.
- (c) Ischemia
- (d) Stretch
- (e) Electrolyte imbalance
- (f) Catecholamines
- (g) Hypoxia
- (h) Drugs - digitalis

(6) Treatment - symptomatic (see PAT) HIS bundle ablation may prevent death.

(7). Nursing concerns

- (a) Document arrhythmia
- (b) Monitor for signs/symptoms of ↓ CO
- (c) Monitor for evidence of A-V blocks
- (d) Monitor patients on digoxin - this is frequently related to digoxin toxicity

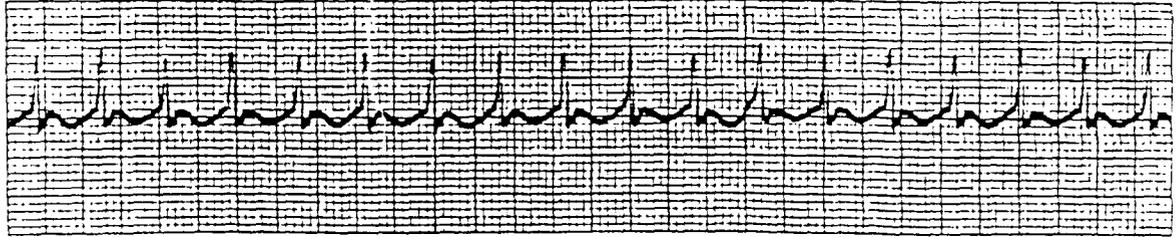
b. Chaotic Atrial Tachycardia (or multifocal atrial tachycardia)



Chaotic atrial tachycardia.

- (1) Rate - 100-130 bpm
 - (2) Rhythm - irregular
 - (3) Origin
 - (a) Atrial ectopic foci
 - (b) At least three different shapes of P waves identified. Multiple shapes of P waves in a single EKG lead
 - (c) Pediatrics: Associated with atrial septal defect in neonates and infants. Atrial dilatation can lead to CHF
 - (4) Etiology
 - (a) Frequently seen in patient with chronic lung disease
 - (b) Electrolyte imbalances, aminophylline therapy, hypoxia
 - (c) Often misdiagnosed as atrial fibrillation
 - (5) Treatment
 - (a) Treat the underlying disease and optimize cardiopulmonary status
 - (b) Treat symptomatically. Cardioversion is of no value.
 - (6) Nursing concerns
 - (a) Document arrhythmia
 - (b) Monitor hemodynamic response (signs of decreased CO)
 - (c) Monitor and document response to treatment
- c. Paroxysmal atrial tachycardia - intraatrial re-entry

c. Paroxysmal atrial tachycardia - intraatrial re-entry



- (1) Rate - 150-250 beats per minute
- (2) Rhythm
 - (a) Abrupt onset and sudden spontaneous termination
 - (b) Usually regular
- (3) Origin - atria
- (4) Conduction
 - (a) Ventricular response to each atrial beat may be normal QRS-T
 - (b) May conduct 1:1
 - (c) May see PAT with block

NOTE: Remember, the AV node will tend to block ectopic atrial rhythms.

- (d) P'R interval may be prolonged due to slow conduction through atria because of agents such as digitalis toxicity
- (5) Etiology
 - (a) Emotional response/trauma
 - (b) Organic heart disease (ischemic, valvular, myopathies)
 - (c) May be seen without evident cardiac disease

(6) Treatment

- (a) If patient is aware of palpitations and pounding, learn to yawn, hold breath, rub neck, perform valsalva maneuver to increase vagal tone and abolish their symptoms. Immerse face in cold H₂O.

NOTE: Carotid sinus massage should not be performed if patient has a cervical bruit or history of TIA

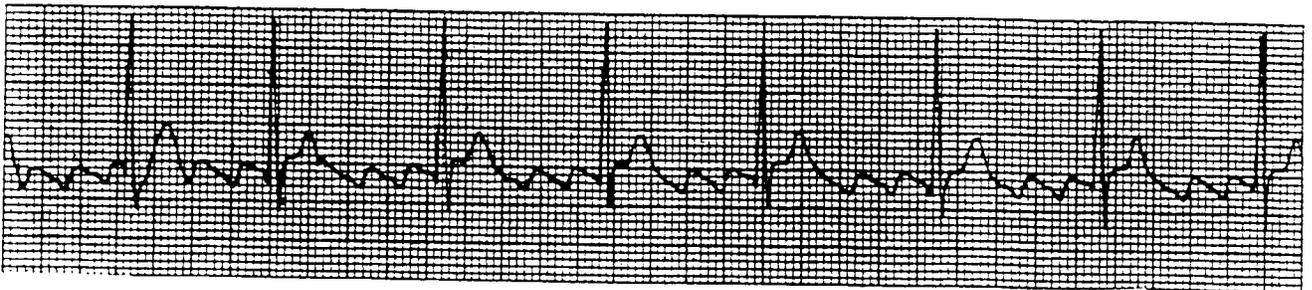
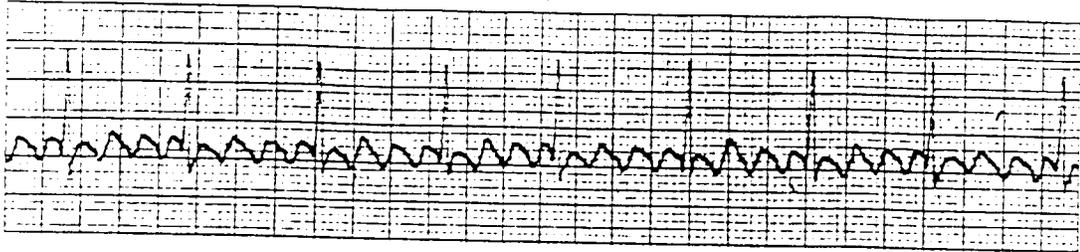
- (b) Decrease sympathetic stimulation at cardiac nerve endings (adrenergic blockers - Inderal)
- (c) Increase sympathetic tone (cholinergic stimulators - digoxin)
- (d) Decrease atrial automaticity (quinidine, pronestyl)
- (e) Cardioversion, pacing
- (f) Decrease sympathetic input from CNS (tranquilizers)
- (f) Verapamil or Diltiazem IV - calcium channel blockers; decreases conduction through SA and A-V nodes. If heart failure is evident administer digoxin instead of calcium channel blockers.
- (g) Adenosine - slows conduction through A-V node

(7) Nursing concerns

- (a) Document dysrhythmia
- (b) Verify rapid pulse, apical/radial
- (c) Assess patient for signs/symptoms CHF, decreased cardiac output, chest pain, cerebral function
- (d) Monitor vital signs as frequently as necessary
- (e) Determine previous experience with PAT - patient may be able to stop rhythm
- (f) Can lead to nodal tachycardia or atrial fibrillation
- (g) Can lead to CHF and coronary insufficiency if persistent

(h) Prepare patient for possible cardioversion

d. Atrial flutter



Atrial flutter with 4:1 AV conduction.

(1) Rate

- (a) Type I (reentry) atrial rate ranges from 250 to 350 beats per minute
- (b) Type II (mechanism unknown) atrial rate ranges from 350 to 450 beats per minute
- (c) Ventricular rate - depends on conduction ratio. Conduction ratio usually 2:1, 3:1, or 4:1

NOTE: Determination of Type I and Type II atrial flutter depends on the F wave morphology and polarity. Any narrow QRS tachycardia at rate of 150 bpm should be assumed to be atrial flutter.

(2) Rhythm

- (a) Atrial - very regular
- (b) Ventricular - usually regular but may vary with A-V conduction ratio

- (c) Common in neonate with atrial rate around 400 bpm. Outgrows dysrhythmia in 12-18 months of age
- (3) Origin
- (a) Multiple atrial foci, or single atrial focus
 - (b) F waves rather than P waves - characteristically saw-toothed, especially when atrial beats approach 300 per minute
- (4) Conduction
- (a) Usually 2:1, 3:1, 4:1
 - (b) A-V node triages impulses - some are not conducted
 - (c) Wenckebach conduction may cause irregular appearance
 - (d) QRS complexes normal
- (5) Etiology
- (a) Rarely found without pathology
 - (b) Usually underlying organic disease, such as coronary artery disease, valvular disease, hyperthyroidism
 - (c) Seen with quinidine treatment of atrial fibrillation
- (6) Treatment
- (a) Emergent treatment - DC cardioversion - 50-100 -200-260J, rapid atrial pacing
 - (b) Digitalis - cardioversion is contraindicated if digitalis toxic
 - (c) Quinidine, procainamide
 - (d) Synchronized cardioversion and transesophageal overdrive pacing in neonate
 - (e) Implanted antitachycardia pacemaker for recurrent atrial flutter
 - (f) Radiofrequency ablation

(g) Anticoagulation therapy on individual basis

(7) Nursing concerns

(a) Identify and document rhythm

(b) Assess patient - hemodynamic effects of rhythm, mentation, perfusion

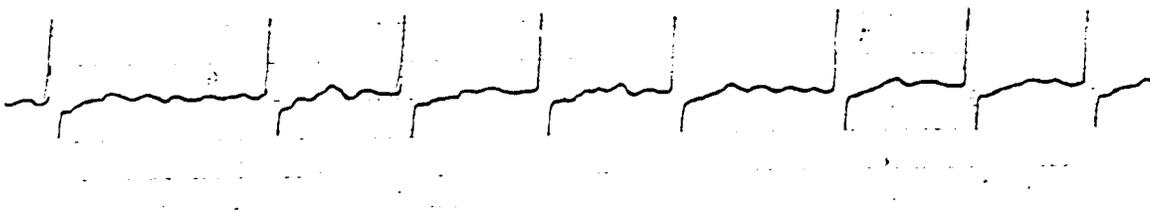
(c) Monitor vital signs

(d) Patient/significant other teaching regarding possible DC cardioversion

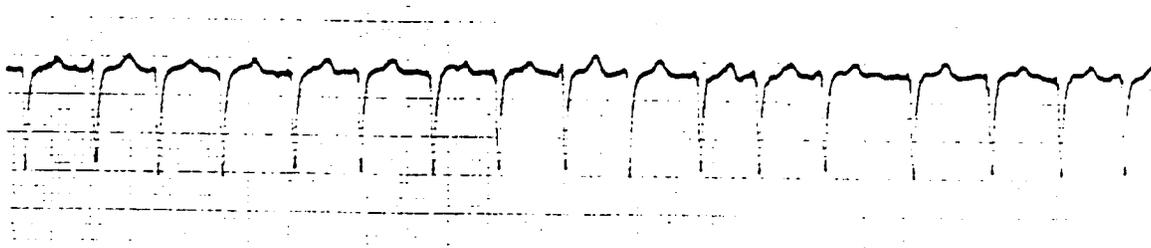
(e) Record response to drug therapy

(f) Hold digoxin 24° prior to cardioversion - if ordered.
Check digitalis level.

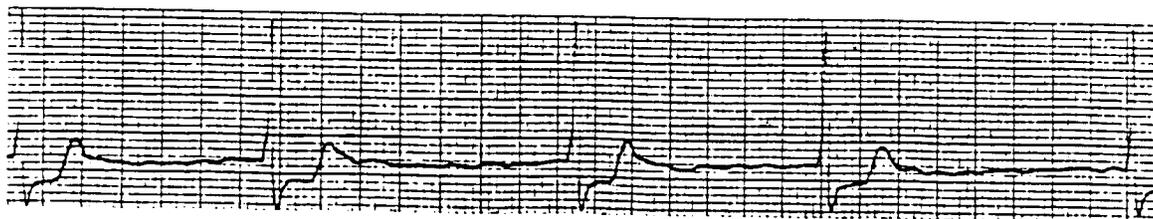
e. Atrial fibrillation



Rapid Atrial Fibrillation



Slow Atrial Fibrillation



(1) Rate

- (a) Atrial - absence of P waves due to chaotic electrical impulses. Indeterminate rate.
- (b) Rare in pediatric age group. Atrial rate around 300 bpm.

NOTE: The atrial can respond only to a certain rate - generally less than 400 beats per minute.

(2) Rhythm

- (a) Atrial - fibrillatory waves are chaotic. P waves absent
- (b) Ventricular - irregular due to weak atrial impulses

(3) Origin

- (a) Multiple chaotic atrial foci
- (b) Frequently results from the atria being stimulated by a PAC during their vulnerable period

(4) Conduction

- (a) No discernible P waves - small irregular undulations on baseline from 350-600 waves per minute. Ventricular response is usually 100-180 bpm in untreated patients
- (b) Currents bombard the AV node
 - 1) May not conduct through A-V node
 - 2) May conduct through AV but not through His Purkinje system
 - 3) Varying refractoriness
- (c) QRS may be normal - but grossly irregular rhythm
- (d) QRS may be widened (WPW, BBB)

(5) Etiology

- (a) May occur without discernible cardiac disease

(b) Coronary heart disease, mitral valve disease, AMI long standing
HTN, left atrial stretch due to mitral stenosis resulting from
rheumatic heart disease

(c) Post cardiac surgery especially in elderly

(d) COPD, hypoxia, WPW, Sick Sinus Syndrome

(6) Treatment

(a) Procainamide, digitalis, quinidine, propranolol (withhold digitalis if
cardioverting to forestall possibility of asystole or ventricular fibrillation)

NOTE: Digitalis and calcium channel blockers are not given if WPW is present. Shortens
refractory period of accessory pathways and causes ventricular rate to increase.

(b) Search for precipitating factor and remove it

(c) Anticoagulation for those with embolic risk

(d) Cardioversion - (if in A-fibrillation for >2 days, start on coumadin for
3-4 weeks before cardioversion)

(e) Radiofrequency ablation

(f) Aflutter surgery remains investigational

(7) Nursing concerns

(a) Document rhythm. Prepare for cardioversion (100-200-360 J)

(b) Assess patient for signs/symptoms of hemodynamic compromise, LV
failure (mentation, perfusion)

NOTE: Atrial kick is lost and cardiac output is decreased by 15-30%.

(c) Assess for chest pain, dyspnea, rales

(d) Monitor response to drug therapy. Ventricular rate should be 60-90 bpm

(e) Monitor signs/symptoms of digoxin or quinidine toxicity

(f) Formation of atrial thrombi

- (g) Monitor signs/symptoms of embolization: peripheral, pulmonary, cerebral (may occur up to 30%)
- (h) Chronic atrial fibrillation may see cardiac dilatation and hypertrophy
- (i) Prepare patient for elective cardioversion; assure informed consent signed