

Northwest Community EMS System
Paramedic Training Program
ATRIAL DYSRHYTHMIAS
Connie J. Mattera, M.S., R.N., EMT-P

Reading assignment:

Aehlert Vol. 1 pp 770 – 784
SOP: Narrow QRS Complex Tachycardia
Drugs: Adenosine; Verapamil
Skills: Vagal maneuvers; synchronized cardioversion

OBJECTIVES:

Upon reading the text assignments, completion of the class and study questions, reviewing the SOPs, and working with their small group, each participant will independently do the following with a degree of accuracy that meets or exceeds the standards established for their scope of practice:

1. Identify on a 6-second strip the following rhythms:
 - a) Premature atrial contractions (PAC's)
 - b) Atrial tachycardia
 - c) AV Nodal Reentrant Tachycardia (AVNRT); AV Reentrant Tachycardia (AVRT)
 - d) Atrial flutter
 - e) Atrial fibrillation
 - f) Wandering atrial pacemaker (multiformed atrial rhythm)
2. Systematically evaluate each rhythm using the following criteria:
 - a) Rate / R-R interval, P-P interval
 - b) Rhythm: Regular or irregular
 - c) Presence/absence of P waves
 - c) P-QRS relationship
 - d) P-R interval
 - e) QRS duration
3. Correlate the cardiac rhythm with patient assessment findings to determine the emergency treatment for each rhythm according to NWC EMSS SOP's.
4. Discuss the action, prehospital indications, side effects, dose and contra-indications of the following:
 - a) Adenocard (adenosine)
 - b) Verapamil
5. Discuss the following treatments for atrial tachydysrhythmias, including indications, contraindications, side effects and procedure for completing:
 - a) Vagal maneuvers
 - b) Synchronized cardioversion

NWC EMSS Paramedic Training Program
ATRIAL DYSRHYTHMIAS

I. The atrium as a pacemaker

- A. Etiology of atrial dysrhythmias: Abnormal impulse formation and conduction through the atria.
- B. P waves that are present reflect atrial depolarization but are different from sinus P waves as the impulse follows a different conduction pathway to the AV node.
 - 1. In slower atrial rhythms, the P waves are often small, pointed, upright waveforms or they may be inverted if the impulse originates near the AV junction with retrograde conduction of the atria.
 - 2. In faster atrial rhythms P waves may be superimposed on the previous T wave so they are not readily apparent (Paroxysmal atrial tachycardia or PAT).
 - 3. In some atrial rhythms atrial depolarization is reflected as
 - a. a sawtooth pattern (atrial flutter); or
 - b. an irregular wavy baseline (atrial fibrillation).

C. Electrophysiologic mechanisms

1. Disorders of impulse formation

a. Altered automaticity

- (1) Sinus node is usually the fastest pacemaker, so normally controls the HR and rhythm
- (2) Secondary pacemakers can take over by accelerating their own automaticity or because the sinus node decreases its automaticity
- (3) Conditions that predispose cardiac cells to altered automaticity
 - (a) Ischemia (hypoxia)
 - (b) Drug toxicity
 - (c) Hypocalcemia
 - (d) Electrolyte imbalances
- (4) The resulting complexes may bear no resemblance to the underlying rhythm as they are discharged by sick or ischemic cells.
- (5) Dysrhythmias of altered automaticity: Premature atrial contractions (PACs), supraventricular tachycardia (SVT), atrial flutter and atrial fibrillation (A-fib or AF).

b. Triggered activity (after - depolarization)

- (1) Occurs when escape pacemaker and myocardial working cells depolarize more than once after stimulation by a single impulse.
- (2) Can result in atrial, junctional, or ventricular ectopic beats that occur in groups (paired or coupled beats) or in burst of 3 or more (paroxysms or tachycardia).
- (3) Abnormal electrical impulses occur during repolarization when cells are normally quiet or right after repolarization.
- (4) Causes
 - (a) Myocardial ischemia and injury
 - (b) Hypoxia
 - (c) Catecholamines
 - (d) Hypomagnesemia
 - (e) Long QT syndrome

- (f) Slow heart rates
- (g) Medications that prolong repolarization (quinidine)

2. **Disorder of conduction: Reentry or reactivation**

- a. An impulse can travel through the myocardium, depolarize it and then re-enter the same area to depolarize it again due to an imbalance of conduction and refractoriness.
- b. Reentry requires
 - (1) A potential conduction circuit or circular conduction pathway like a fork in the Purkinje fibers or an accessory AV conduction pathway.
 - (2) A block within part (one limb) of the circuit.
 - (3) Delayed conduction within the remainder of the circuit.
- c. Normally, an impulse spreads through the heart only once after it is initiated by pacemaker cells. In reentry, an electrical impulse is delayed or blocked (or both) in one or more divisions of the conduction system while being conducted normally through the rest of the system. This results in the delayed electrical impulse entering cardiac cells that have just been depolarized by the normally conducted impulses, producing a circular movement of the impulse that continues as long as it encounters receptive cells (Aehlert, 91).
- d. This usually produces a single premature beat or may result in repetitive electrical impulses creating rapid heart rates.
- e. Re-entrant arrhythmias do bear a relationship to the underlying rhythm
- f. Common causes
 - (1) Hyperkalemia
 - (2) AMI
 - (3) Some antidysrhythmic medications
- g. Reentrant dysrhythmias
 - (1) Premature atrial contractions (PACs)
 - (2) Paroxysmal supraventricular tachycardia (PSVT)

3. **P/QRS ratio imbalance:** When atrial rates are very rapid, the AV node blocks some of the impulses, protecting the ventricles from high rates. Atrial rates will be faster than ventricular rates with fewer QRS complexes than P, flutter or fibrillatory waves.

D. **Clinical implications**

- 1. Younger patients generally tolerate rapid rates better than older ones
- 2. If decreased cardiac reserves, decreased diastolic filling times may rapidly lead to heart failure, decreased cardiac output, and cardiogenic shock.
- 3. Treatment usually depends on the hemodynamic stability of the patient and the length of time they have experienced the rhythm.

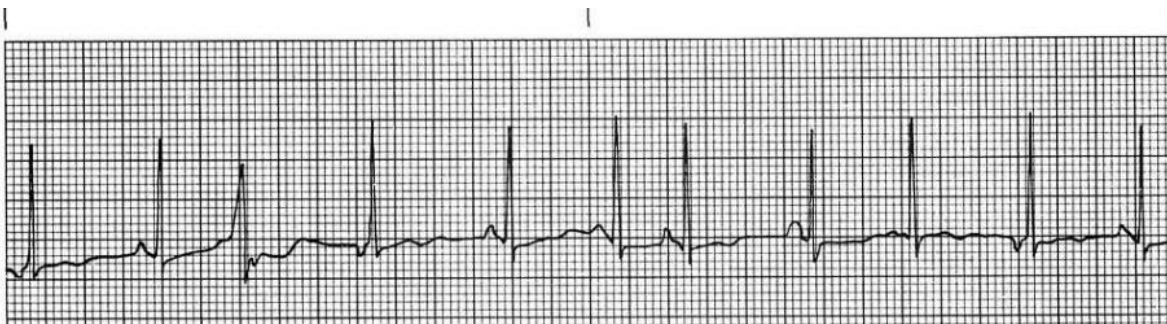
II. **Multiformed Atrial Rhythm – updated term for the rhythm formerly known as Wandering Atrial Pacemaker (WAP)**

- A. **Description:** Pacemaker site shifts back and forth from the sinus node to other pacemaker sites in the atria and sometimes the AV node producing a change in the size, shape, and direction of the P waves.
- B. Uncommon – do not focus on this rhythm!

C. **Etiology**

1. Multiple pacemaker sites are competing for control of the heart
2. Increased vagal tone on the SA node
3. Increased automaticity of the atria or junctional pacemaker cells
4. May be associated with chronic lung disease, valvular heart disease
5. May be associated with administration of digitalis
6. Is normal during sleep and in young healthy hearts

D. **Characteristics**



1. Rate
 - a. Usually 60 - 100 per minute but may be slow
 - b. If rate is > 100 , the rhythm is termed multifocal (or chaotic) atrial tachycardia.
2. Rhythm: Regular to slightly irregular as the pacemaker site shifts from the SA node to ectopic atrial locations and the AV junction
3. P waves
 - a. Change as pacemaker site changes (“wanders”)
 - b. Can change from beat to beat and may disappear completely
 - c. Vary in size, shape, and direction
 - d. Must see 3 different P wave configurations within one lead to be classified as a WAP.
4. P-R interval
 - a. Varies based on location of impulse formation
 - b. May become less than 0.12 seconds
5. QRS complex: 0.10 seconds or less as there is normal conduction through the ventricles.

E. **Clinical significance:** No detrimental effects unless HR is very slow.

F. **Treatment:** None usually required; may ask patient to cough to decrease vagal tone.

III. **Premature Atrial Contractions or Complexes (PACs)**

A. **Description**

1. Single **early** electrical impulse originating in the atria outside the SA node
2. Usually caused by enhanced automaticity in atrial tissue
3. May originate from a single site or from multiple sites
4. Creates a **premature P wave** occurring before the next expected sinus beat followed by an identical or very similar QRS to the normally conducted impulses.
5. They may or may not be conducted to the ventricles depending on their degree of prematurity and the state of AV conduction. If the early P wave is followed by a QRS, it is called a conducted PAC (atrial impulse is also conducted to the

ventricles). If the early P wave is not followed by a QRS it is called a **non-conducted PAC**.

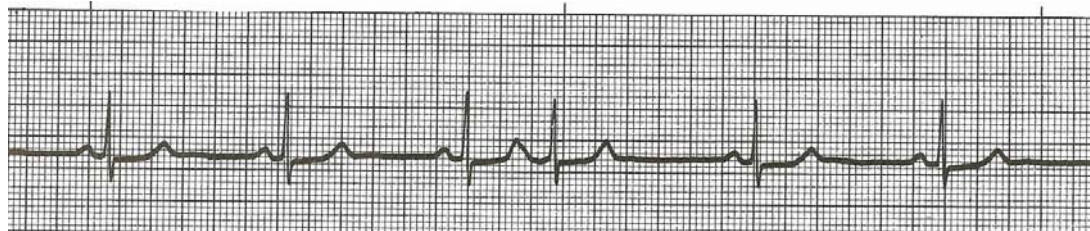
6. Followed by a **noncompensatory pause**: Measurement from R wave before PAC to R wave after PAC is less than 2 R-R intervals of the underlying rhythm

Premature depolarization of the atria by the PAC produces premature depolarization of the SA node. The SA node repolarizes earlier than normally expected, so the next sinus beat is a little earlier than normal.

B. Etiology

1. Use of caffeine, alcohol, nicotine
2. Sympathetic stimulant drugs - epinephrine
3. Emotional stress – increased sympathetic tone
4. Fatigue
5. Electrolyte disturbances (low K or Mg)
6. Hypoxia
7. Myocardial ischemia
8. Coronary artery disease (CAD)
9. COPD
10. Digitalis toxicity
11. Hyperthyroidism
12. Dilated or hypertrophied atria (mitral stenosis or atrial septal defect)
13. Organic heart disease
14. Can be due to a stretched atrium so may signal CHF
15. No apparent cause

C. Characteristics



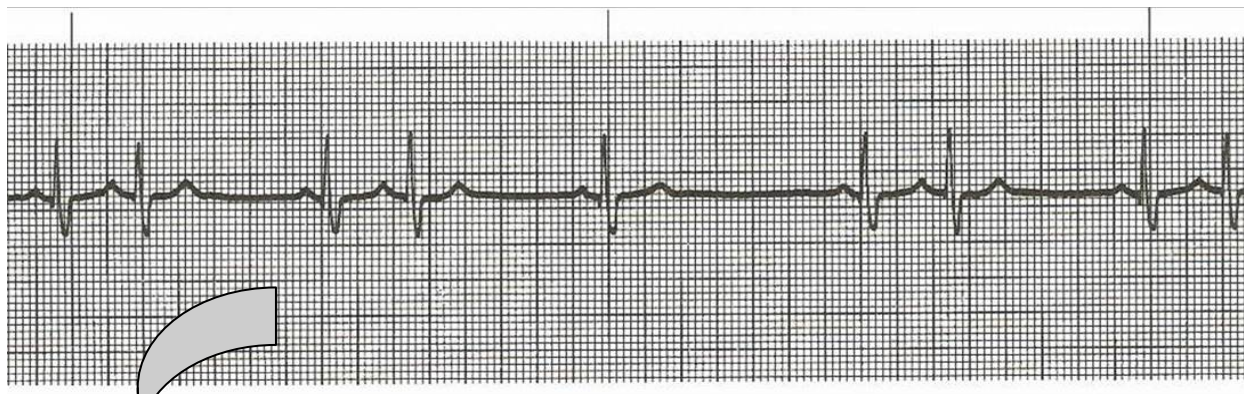
1. **Determine underlying rhythm first**
 - a. Generally occurs in a sinus rhythm or sinus bradycardia
 - b. Cannot occur in atrial fib
2. **Rate**: Usually within normal range but depends on underlying rhythm and number of premature beats
3. **Rhythmicity**
 - a. Depends on underlying rhythm: usually regular except for PACs
 - b. PAC will make the rhythm irregular
4. **P waves**
 - a. **Early P wave**
 - b. P wave of PAC may differ in shape from sinus P wave. Shape depends on the location of the ectopic pacemaker site. If close to SA node, may closely resemble sinus P waves.
 - (1) Lead II: Generally upright
 - (2) May be pointed, biphasic, flattened, or notched
 - (3) May be inverted if pacemaker site near AV node
 - (4) May be hidden in preceding T wave causing distortion of T wave
 - c. Precedes each QRS complex

5. **P-R interval of PAC**

- a. Usually normal or slightly shortened
- b. May be prolonged greater than 0.20 seconds depending on the prematurity of the complex
- c. Differs from underlying rhythm
- d. Not measurable if P is buried in previous T

6. **QRS complex of PAC**

- a. Usually 0.10 seconds or less but may be greater than 0.10 seconds if PAC is so early that the bundle branches (especially right side) may not have repolarized sufficiently to conduct the impulse normally (**aberrant or abnormal** conduction). The left bundle conducts the impulse fine but has delayed firing of the right bundle branch.
 - (1) Aberrant conduction results in a wide QRS (> 0.10 sec) – call it a PAC with aberrant ventricular conduction
 - (2) Need to distinguish between a PAC w/ aberrant conduction and a PVC. PVCs are not preceded by P waves.
- b. **QRS is absent if PAC is non-conducted** (meaning there is an early P wave with no QRS)– look carefully at the preceding T wave for a change in morphology. Look for an early buried P if there is a pause in the normal ventricular rate. This may be mistaken for sinus arrest if the early P wave is not seen and the QRS is non-conducted.



7. **Pattern of PACs**

- a. Single beat
- b. Every other beat – bigeminal
- c. Every 3rd beat – trigeminal
- d. Every 4th beat – quadrigeminal
- e. In pairs: couplets
- f. In runs of 3 or more: atrial tachycardia
- g. May warn of more serious atrial arrhythmias

8. **A PAC is not an entire rhythm** – it is a single beat. It is important to identify the underlying rhythm plus the ectopic beats, i.e., sinus bradycardia at 46 beats/min with two PACs (Aehlert, 93).

D. **Clinical significance**

1. Isolated: Very common; minimal significance
2. Frequent
 - a. May suggest organic heart disease
 - b. May predispose to other atrial tachydysrhythmias

E. **Treatment:** Treat the patient and the underlying cause, not the PAC's

IV. **Supraventricular Tachycardia (SVT) in general**

A. **Description**

1. All the tachydysrhythmias whose site of impulse formation is above the Bundle of HIS. Rapid atrial or junctional depolarization overrides SA node.
2. By strict definition, this includes sinus, atrial, and junctional tachycardias as well as the tachycardias associated w/ atrial flutter and fibrillation. This term should not be used however, if P waves can be clearly seen and the ventricular rate is < 150.
3. This term can apply to a dysrhythmia with a rapid ventricular rate and narrow QRS complex, but no discernable P waves and the specific origin (atrial or junctional) is uncertain
4. Often occurs in paroxysms--lasting minutes to hours
5. Paroxysmal is the term used to describe the sudden onset or cessation of a dysrhythmia and identification of the rhythm that preceded it. An SVT that starts or ends suddenly is called **paroxysmal supraventricular tachycardia (PSVT)** (Aehlert, 96).

B. **Etiology**

1. Common in both normal and diseased hearts
2. Common in childhood and young adulthood
3. Can be due to automaticity, triggered activity or reentry

C. **Three primary types of PSVT**

1. Atrial tachycardia
2. Atrioventricular nodal reentrant tachycardia (AVNRT)
3. Atrioventricular reentrant tachycardia (AVRT)

D. **Classifications**

1. **AV nodal active:** Require the AV node to sustain the tachycardia. Regular, narrow QRS complex tachycardias are most commonly caused by AV Nodal Reentrant Tachycardia (AVNRT) and AV Reentrant Tachycardia (AVRT), both of which require the AV node as part of the reentry circuit that sustains the tachycardia. Most common type.
2. **AV nodal passive:** AV node does not play a part in the maintenance of the tachycardia. Passively conducts the supraventricular rhythm into the ventricles. **They do not need the AV node to sustain the dysrhythmia.**
 - a. Atrial tachycardia
 - b. Atrial flutter
 - c. Atrial fibrillation

V. **AV nodal Reentrant Tachycardia (AVNRT) - PSVT**

A. AVNRT is **usually precipitated by a PAC** that is discharged by the electrical circuit.

1. There is normally a reentry focus in the AV node. Two conduction pathways in the AV node conduct impulses at different speeds and recover at different rates.
 - a. Fast pathway – Conducts rapidly but has a long refractory period (slow recovery time).
 - b. Slow pathway – Conducts impulses slowly but has a short refractory period.
 - c. Conduction normally occurs down the fast pathway to activate the Bundle of HIS and the ventricles.
2. Under the right conditions, the fast and slow pathways can form an electrical loop or circuit. As one side is repolarizing, the other side is depolarizing. This allows the

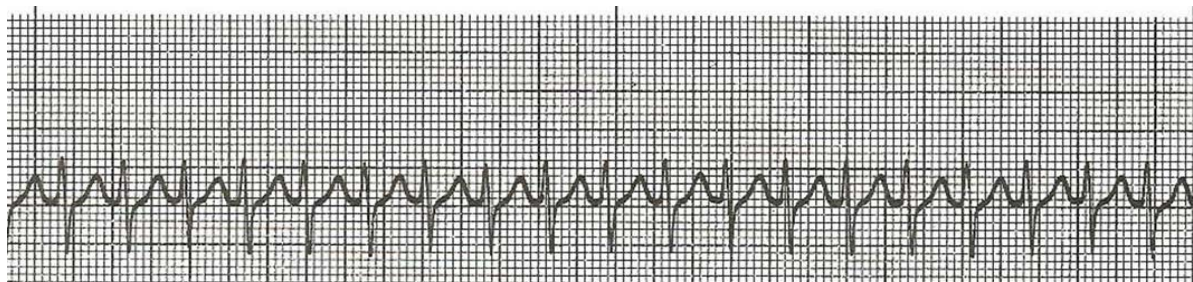
impulse to circle the pathway indefinitely, reentering the normal electrical pathway with each pass around the circuit. This results in a **very rapid and regular rhythm** that ranges from **150-250 beats/min (typically 170-250)**. (Three or more sequential PACs occurring at a rate of more than 100/minute is technically a paroxysmal atrial tachycardia.)

3. This reentry phenomenon overrides the sinus node and becomes the pacemaker. Can last minutes, hours, or days

B. Etiology – similar to PACs

1. Any age unassociated with heart disease; Common in young, healthy persons without no structural heart disease
2. Frequently associated with underlying atherosclerotic heart disease (ASHD) and rheumatic heart disease
3. May be precipitated by
 - a. stress;
 - b. overexertion; or
 - c. ingestion of alcohol, caffeine, or nicotine.
4. Pathological conditions that can cause AVNRT
 - a. Atherosclerotic heart disease; HTN
 - b. Rheumatic heart disease (mitral valve disease)
 - c. AMI
 - d. Digitalis intoxication
 - e. Chronic obstructive pulmonary disease

C. Characteristics



1. **Rate:** approximately 150-250 per minute; typically 170-250
2. **Rhythm:** Atrial is regular and ventricular is regular except at onset and termination
3. **P waves:**
 - a. Atrial P waves may be seen that differ from sinus P waves – often pointed.
 - b. Because AVNRT originates in the area of the AV node, the impulse spreads simultaneously to the atria and the ventricles. Thus P waves are **often hidden in the QRS**.
 - c. If the ventricles are depolarized first and then the atria, an inverted (negative) P wave will appear after the QRS (retrograde P wave) in Leads II, III, and aVF. This P wave often distorts the end of the QRS complex.
 - d. Conduction of atrial complexes to the ventricles is usually 1:1 unless atrial rates are very rapid. When the AV node selectively filters conduction of the some of the impulses to the ventricles it is called **PAT with block**. This rhythm is frequently associated with digitalis toxicity.
4. **PR interval:** Usually not measurable
5. **QRS complex:** 0.04-0.10 seconds unless a ventricular conduction delay exists.

D. Clinical significance

1. Often sensed as palpitations or funny heart beats – this is a source of stress and anxiety for the patient.
2. Tolerated for a short time in young hearts with good cardiac reserve.
3. Rapid rates may cause significant decreases in cardiac output and coronary artery perfusion in patients with underlying heart disease.
 - a. Increased myocardial oxygen demand and cardiac workload may precipitate angina, hypotension, or HF in people with CAD
 - b. Lightheadedness, dizziness, weakness, nervousness
 - c. Dyspnea
 - d. Chest pain or pressure
 - e. Nausea, diaphoresis
 - f. Syncope, possible signs of shock
4. Recurrent episodes vary in frequency, duration, and severity from several times a day to every 2-3 years.

E. **Treatment**

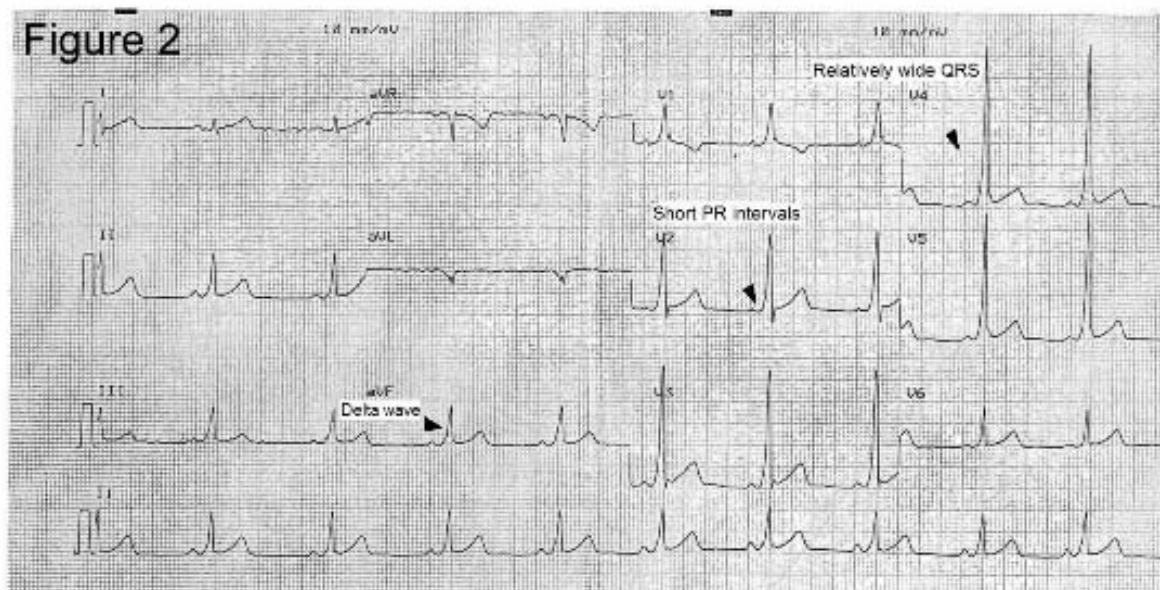
1. See NWC EMSS SOP: Narrow QRS Complex Tachycardia
2. Stable but symptomatic patients
 - a. Assess for cause
 - b. Eliminate /correct inciting cause if possible
 - c. Further treatment depends on patient's tolerance of the rhythm
3. Hemodynamically stable implies the following:
 - a. SBP 90 or above
 - b. Normal level of consciousness
 - c. Skin warm and dry
 - d. Free of chest pain, dyspnea, or signs of heart failure
4. IMC: Relieve anxiety
5. **Vagal maneuvers** – Slow the heart rate by increasing parasympathetic tone
Used to stimulate baroreceptors in the internal carotid arteries and the aortic arch. Stimulation of these receptors results in reflex stimulation of the vagus nerve and release of acetylcholine. Acetylcholine slows conduction through the AV node, resulting in slowing of the heart rate.
 - a. Bearing down (Valsalva maneuver)
 - b. Squatting
 - c. Breath-holding
 - d. **Blowing out through a very small straw** (like a coffee stirrer). Need positive intrathoracic pressure in chest for 15 seconds.
 - e. Stimulation of the gag reflex (not recommended for the field)
 - f. **Carotid sinus pressure** (Carotid massage): No longer done in the NWC EMSS after the 2007 SOPs. The carotid sinus is located at the bifurcation of the carotid artery at the angle of the jaw. The right carotid sinus has more vagal fibers to the SA node and atrial fibers. The left carotid has more vagal fibers to the AV node and some to ventricular muscle. The right carotid body is massaged for best results.
 - g. Immersion of face in ice water activates Mammalian Diving Receptors to slow HR (also not recommended in the field)
6. Adenocard (adenosine) – See drug profile
7. Verapamil - See drug profile

8. Amiodarone may be ordered by medical control: It has the best balance between side effects and effectiveness in patients with heart failure. Should not be used in combination with Verapamil due to additive hypotensive effects.
9. **S&S of hemodynamic compromise – Synchronized cardioversion**
 - a. Shock, chest pain, hypotension
 - b. Severe shortness of breath, pulmonary congestion, HF
 - c. Decreased level of consciousness
10. Carefully measure QRS (what appears to be SVT may be VT)

VI. **AV Reentrant Tachycardia (AVRT)**

- A. **Preexcitation** is used to describe rhythms that originate from above the ventricles but the impulse travels via a pathway other than the AV node and the bundle of HIS.
- B. The supraventricular impulse excites the ventricles earlier than would be expected if the impulse traveled through normal pathways in the AV node.
- C. Patients with preexcitation syndromes are prone to AVRT
- D. The abnormal pathways are formed during fetal development and persist as congenital malformations of working myocardial tissue in some patients. Because they bypass part or all of the normal conduction system they are called **accessory pathways**.
- E. The term **bypass tract** is used when one end of an accessory pathway is attached to normal conductive tissue. This pathway may connect the right atrial and ventricular walls, the left atrial and ventricular walls, or connect the atrial and ventricular septa on either the right or left side.
- F. **Three major forms of preexcitation syndromes**
 1. **Wolff-Parkinson-White (WPW) Syndrome:** Accessory pathway is called the **Bundle of Kent:** Directly connects the atrial to the ventricles, completely bypassing the normal conduction system in the AV node.
 2. **Lown-Ganong-Levine (LGL) Syndrome:** Accessory pathway is called the James bundle. This connects the atria directly to the lower portion of the AV node, thus partially bypassing the AV node.
 3. The third type involves Mahaim fibers that do not bypass the AV node but originate below the AV node and insert into the ventricular wall, bypassing part of all of the ventricular conduction system.
- G. **Wolff-Parkinson-White (WPW) Syndrome - most common form**
 1. P waves are present and of normal shape
 2. PR interval is short because AV node is bypassed
 3. QRS is typically prolonged because part of the ventricle receives the impulse early through the accessory pathway and starts to depolarize in a cell-to-cell fashion before the rest of the ventricle is activated by the normal pathways
 4. This preexcitation of the ventricles distorts the initial portion of the QRS giving it a slurred uptake in some leads called a **delta wave**.
 5. More common in males than females. About $\frac{2}{3}$ of patients have no heart disease. Rare: 4/100,000 people. One of the most common causes of tachydysrhythmias in infants and children. Symptoms may not appear until young adulthood.
 6. Prone to tachydysrhythmias due to loss of the protective blocking mechanism of the AV node and because the accessory pathway provides a mechanism for reentry.
 7. Clinical significance during tachycardia
 - a. Palpitations, anxiety, weakness, dizziness

- b. Chest pain, shortness of breath
- c. Shock



- H. PSVT and AF are two of the most common tachydysrhythmias seen in WPW. AF and A-flutter that occur in the presence of an accessory pathway are particularly dangerous because of the extremely rapid ventricular rate that can result from conduction of atrial impulses directly to the ventricles. The ventricular rate can be 250-300 beats/min and can deteriorate into VF and sudden death.

DO NOT give calcium blockers like Verapamil to WPW – they may accelerate the speed of conduction through the accessory pathway, resulting in a further increase in ventricular rate. The hospital may order amiodarone or cardioversion.

VII. Atrial Flutter

A. Description

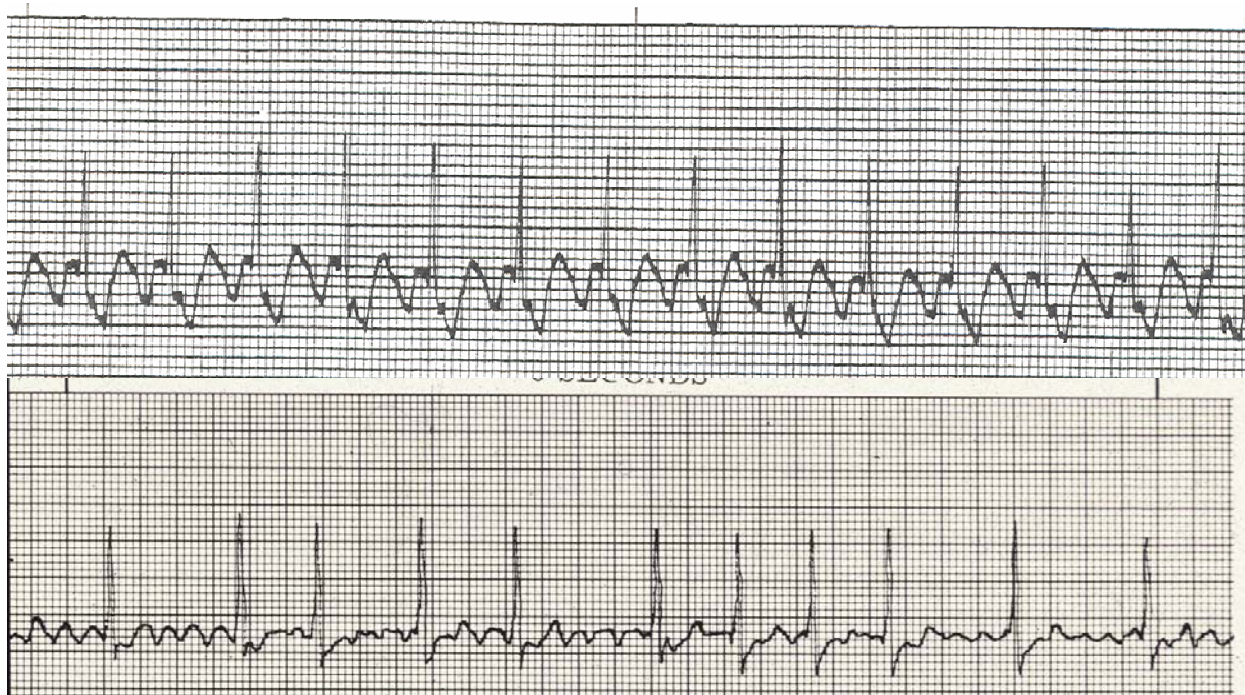
1. 2 theories: Rapidly firing single ectopic site in the atria caused by accelerated automaticity or rapid re-entry circuit in the right atrium (Type I – also called *typical* or *classical*). Type II atrial flutter is called *atypical* or *very rapid atrial flutter*. They often develop atrial fibrillation. The exact mechanism for this type has not been defined.
2. Atria are depolarized at rates of 250-400 times/minute
3. Atria demonstrate wave deflections called **flutter (F) waves**. They have an initial negative component followed by a positive component producing V-shaped waveforms that have a **sawtooth or picket fence appearance**. They alter the baseline completely so there is no isoelectric line. The T wave is often obscured by the flutter waves.
4. The AV node cannot conduct all the impulses to the ventricles. Physiologic refractoriness of the AV node, not pathologic block prevents the 1:1 ventricular response. Normally, the AV node cannot conduct faster than 180/minute. At an atrial rate of 300, every other impulse arrives at the AV node while it is still refractory. Thus the common ventricular rate would be 150 and is called 2:1 conduction.
5. The AV junction may conduct impulses in various ratios: 1:1, 2:1, 3:1, 4:1 or more resulting in a discrepancy between atrial and ventricular rates. Even ratios are more common than odd. Accessory pathway conduction may be 1:1 as the AV node is bypassed.

6. AV blockade of impulses may be consistent or variable. If consistent, the R-R is regular; if inconsistent, the R-R is irregular. **If inconsistent report the rhythm as atrial flutter with variable AV conduction.**
7. QRS are usually narrow as long as ventricular conduction is normal.

B. Etiology

1. Usually a paroxysmal rhythm precipitated by a PAC that may last for seconds to hours and sometimes 24 hours or longer
2. Valvular (mitral or tricuspid) heart disease
3. Ischemic heart disease, hypoxia
4. Hypertensive heart disease
5. Pulmonary disease; pulmonary emboli
6. HF
7. Pericarditis; Myocarditis; cardiomyopathy
8. Digitalis or quinidine toxicity
9. After cardiac surgery
10. Thyrotoxicosis
11. Alcohol intoxication

C. Interpretation



1. Rate
 - a. Atrial: approximately 250-450 per minute; typically 300
 - b. Ventricular: variable, depending on conduction through AV junction – will be less than the atrial rate. Will usually not exceed 180 due to intrinsic conduction rate of the AV node.
2. Rhythm
 - a. Atrial rhythm: Regular
 - b. Ventricular: Usually regular but can be irregular if variable block
3. P waves
 - a. V-shaped waveforms resemble "**sawtooth**" or "picket fence" pattern called **flutter waves**
 - b. May be difficult to identify in 2:1 flutter
 - c. Suspect 2:1 flutter when rhythm is regular and ventricular rate is 150

4. P-R interval: Not measurable
5. QRS complex: Normal (0.10 seconds or less) unless ventricular conduction disturbance

D. **Clinical significance**

1. Depends on the rate and regularity of the ventricular response. Normal ventricular rates are usually well tolerated.
2. Rapid ventricular rates
 - a. May compromise cardiac output by ↓ atrial "kick"
 - b. Promotes stasis of blood in atria that may form mural thrombi leading to a risk of pulmonary emboli, arterial emboli or stroke
 - c. Precipitate associated symptoms

E. **Treatment**

1. Indicated only for rapid ventricular rates with hemodynamic compromise
2. See NWC EMSS SOP: Narrow QRS Complex Tachycardia
3. Ventricular rate control is indicated for most patients.
4. Response to vagal stimulation may be characteristic and helpful diagnostically. The ventricular rate may exactly halve (from 160-80) and then double back after the stimulation ceases.
5. Go directly to Verapamil for stable patients.
6. In patient with HF, OLMC may order amiodarone instead if dysrhythmia has lasted less than 48 hours. If longer than 48 hours will need anticoagulation before conversion of the rhythm.
7. Synchronized cardioversion – unstable patients – will often respond to 50 J.

VIII. **Atrial fibrillation**

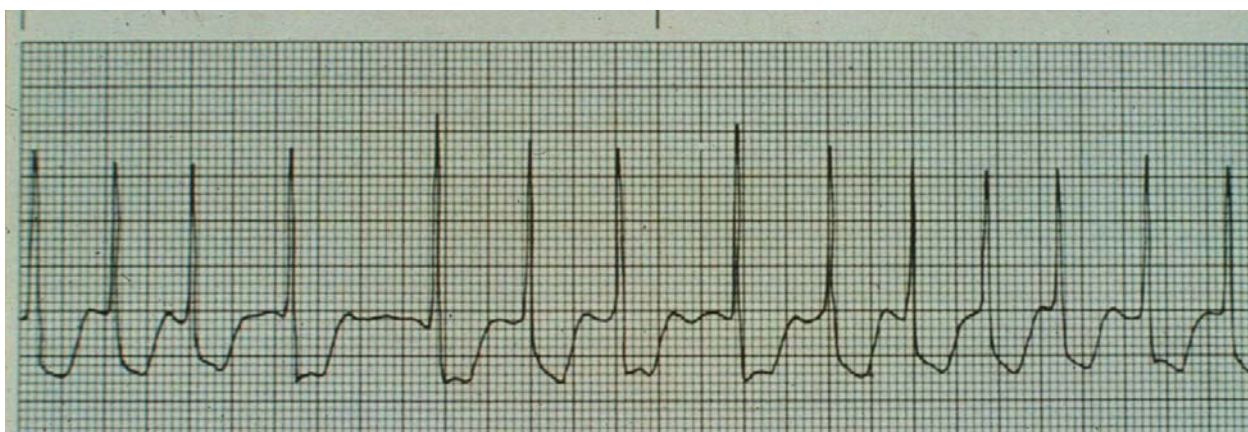
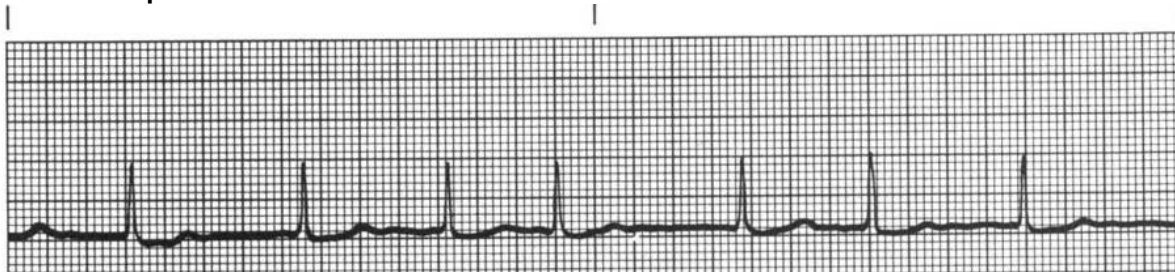
A. **Description**

1. Atrial fibrillation (AF) is the most common arrhythmia seen in clinical practice and is a potentially life-threatening disease increasing the risk for hospitalization, stroke, and mortality, and worsening the prognosis of patients with cardiovascular risk factors. It affects nearly 7 million people in the United States and Europe.
2. Multiple ectopic pacemaker sites or sites of rapid reentry circuits in the atria discharging impulses at rates of 400 or more. Atria quiver (fibrillate) rather than contract resulting in an ineffective atrial contraction.
3. May occur acutely (lasting < 48 hours), paroxysmally (intermittent) or chronically (lasting at least 1 month).
4. Produces rapid, small, irregular, **wavy deflections not resembling each other called fibrillatory (f) waves**
 - a. Large waves are called coarse.
 - b. Small waves are referred to as fine.
 - c. Sometimes they are so small they appear to be an almost flat baseline.
 - d. The fibrillation waves affect the whole baseline – none is normal.
 - e. Flutter waves may be mixed with fibrillatory waves (fib-flutter).
5. The AV node is bombarded and cannot handle (is refractory to) all the impulses from the atria. AV conduction is random and ventricular response is slower than atrial firing and is **highly irregular**.

B. Etiology

1. Normal people – temporary (paroxysmal); lasts hours to days associated w/ stress or excessive ingestion of alcohol or caffeine
2. Cardiac causes/conditions
 - a. Valvular disease: – mitral valve stenosis, mitral regurgitation, rheumatic heart disease
 - b. Left ventricular hypertrophy
 - c. Pericarditis; cardiomyopathy; myocarditis
 - d. Coronary artery disease; Myocardial infarction; Heart failure
 - e. Sick sinus syndrome
 - f. Post-cardiac surgery
 - g. HTN, congenital heart disease
3. Non-cardiac causes
 - a. Drugs or intoxicants: alcohol (“Holiday heart syndrome”), carbon monoxide
 - b. Acute or chronic pulmonary disease
 - c. Hyperthyroidism
 - d. Pneumonia; pulmonary embolism
 - e. Surgery: Common after heart surgery
 - f. Increased age; male gender
 - g. Electrolyte imbalance; hypokalemia
 - h. Electrocutation
 - i. Other pulmonary diseases such as COPD (Cottrell & Mack, 2008)
4. AF can cause pulmonary emboli or a stroke

C. Interpretation



1. Rate
 - a. Atrial: 350 or more/minute – not measurable on ECG
 - b. Ventricular: Varies greatly
 - c. If ventricular rate < 100: **Controlled A-fib**
 - d. If ventricular rate > 100: **Uncontrolled A-fib**
2. **Rhythm:** Irregularly irregular unless very fast. If ventricular rates are very fast the R-R may appear almost regular. Measure carefully!

3. P waves
 - a. No discernible P waves – fibrillatory waves
 - b. Chaotic atrial activity
 - c. Can look like a flat isoelectric line or a fine or coarse wavy line
4. P-R interval: Not measurable
5. QRS complex: 0.10 seconds or less.
 - a. QRS complexes are usually normal in shape and duration as long as ventricular conduction is normal – they just occur irregularly. This **irregular irregularity** is the most typical finding of A-fib.
 - b. AF can occur simultaneously with complete AV block. There will be no distinguishable P waves (just atrial fibrillatory waves) and the resulting ventricular rhythm will be slow and regular as the ventricles are pacing themselves.
 - c. Aberrant ventricular conduction does occur with some frequency and is difficult to identify since there is not a definite preceding atrial complex. This can be of clinical significance if the rhythm is identified as PVCs. Aberration is likely when a lengthening of the ventricular cycle is immediately followed by a short cycle – the beat ending the short cycle is aberrantly conducted. The long-short cycle is called “Ashman’s phenomenon”. Runs of aberrant beats can look like a run of V-tach.

D. Clinical significance – similar to A-flutter

1. No contraction of the atria as a whole
 - a. No atrial "kick" to finish ventricular filling
 - b. Increased myocardial oxygen demand and workload
 - c. Cardiac output decreased by 20-25%
 - d. Blood in atria tends to collect rather than be ejected which causes microthrombi to form on interior atrial walls. If one of these clots breaks loose, it can cause a pulmonary embolism (right heart) or stroke (left heart).
2. Frequently produces a pulse deficit
3. If rate of ventricular response is controlled: Usually well tolerated
4. Ventricular response less than 60 per minute
 - a. Suspect digitalis toxicity
 - b. May compromise cardiac output
5. Rapid ventricular response coupled with loss of "atrial kick" may cause cardiovascular decompensation
 - a. Angina, ACS
 - b. Heart failure
 - c. Cardiogenic shock

E. Treatment – Same as atrial flutter

1. The available pharmacologic interventions for AF have been the subject of intense investigation, especially in regards to endless debate of rhythm vs rate control strategies in AF. If dysrhythmia has a recognizable cause and is not creating hemodynamic instability, treat the underlying problem, i.e., pulmonary edema.
2. **With uncontrolled A-fib, need to control ventricular rate – goal is not to convert the rhythm in the field**
3. **Stable:** Go directly to Verapamil 5 mg IVP

- a. The benefits to be gained by controlling the ventricular rate, especially in those with systolic dysfunction and accompanying failure clearly outweigh the risk of negative inotropic effects of the calcium blocker.
 - b. Patients with systolic dysfunction also, more often than not, have a component of diastolic dysfunction of varying severity. Those with stiff ventricles and decreased diastolic filling rely heavily on the contribution of the "atrial kick" to ventricular filling. With the loss of the atrial kick and shortened ventricular filling time in uncontrolled AF, these patients suffer a "double wammie". The best interim treatment before attempts to convert them to NSR, is rate control to lengthen the diastolic filling time. Slowing the HR will lengthen the diastolic filling time, which will increase the cardiac output (Starling's law).
4. **Unstable:** Synchronized cardioversion

References

Cottrell, D.B. & Mack, K. (2008). Atrial fibrillation: An emergency nurse's rapid response. *JEN*, 34(3), 207-210.

ADENOSINE (Adenocard)

Classifications:	<ul style="list-style-type: none">➤ Pharmacologic: Naturally occurring nucleoside➤ Therapeutic: Antiarrhythmic
Actions:	Used to depress SA node activity and to slow conduction through the AV node and interrupt reentry pathways in the AV node.
Therapeutic benefit:	Can terminate reentry arrhythmias and restore NSR in patients experiencing PSVT including those associated with WPW
Indications:	PSVT and WPW Syndrome after vagal maneuvers have proven unsuccessful
Ineffective:	A-flutter, AF, atrial or ventricular tachycardia (dysrhythmias not due to reentry through the SA/AV node). In these rhythms, adenosine may produce transient AV or retrograde block (ventriculoatrial) that may clarify the diagnosis, but has been reported to cause death. Using adenosine to distinguish SVT from VT is discouraged. Use only if an SVT is suspected.
Contraindications:	<ul style="list-style-type: none">➤ 2° or 3° AV block➤ Known atrial fibrillation➤ Sinus pause/arrest/sick sinus syndrome (except in pts w/ a pacemaker)➤ Allergy
Dose & route	<ul style="list-style-type: none">➤ The half life of this drug is < 5 seconds. THE FASTER IT IS GIVEN THE MORE EFFECTIVE THE DRUG WILL BE➤ Adults: Initial dose: 6 mg rapid IVP over 1-3 seconds followed immediately by a 20 ml NS flush➤ Repeat dose if no results after 1-2 min.: 12 mg rapid IVP over 1-3 seconds followed immediately by a 20 ml NS flush If persistent/recurrent PSVT, consider a Ca blocker➤ Peds: 0.1 mg/kg IVP (max. first dose 6 mg). May double and repeat X 1 (max second dose 12 mg)
Side effects:	<ul style="list-style-type: none">➤ CNS: Headache, light headedness➤ CV: Chest pain, palpitations, hypotension, transient dysrhythmias (SB, heart block, asystole, PVCs and PACs) occur in 50-60% of all patients during conversion to sinus rhythm➤ Resp: Dyspnea, may cause bronchoconstriction in asthma patients➤ GI: nausea, metallic taste➤ Skin: Facial flushing <p>A major advantage of Adenosine is that adverse effects last less than 1-2 min due to the rapid elimination of the drug from the extracellular compartment.</p>
Precautions:	<ul style="list-style-type: none">➤ Start IV in a proximal vein➤ Assess VS frequently while giving the drug➤ If pt develops a high degree AV block, do NOT give additional doses➤ Use with caution in patients with preexisting bradycardias or conduction defects who do not have a functioning pacemaker➤ Use with caution in those with a denervated, transplanted heart, action will be prolonged. Adjust the dose or use alternative therapies.
Drug interactions:	<ul style="list-style-type: none">➤ Adenosine does not interact with Verapamil, Lidocaine, digitalis, quinidine, or other cardiac drugs.➤ Theophylline or related methylxanthines (caffeine, theobromide) prevent adenosine from binding to receptors sites. Asthma or COPD patients may be taking these drugs and may need larger doses.

VERAPAMIL (Isoptin, Calan)

Classifications:	<ul style="list-style-type: none">➤ Pharmacologic: Calcium channel blocker➤ Therapeutic: Antianginal, antiarrhythmic, antihypertensive
Actions:	<ul style="list-style-type: none">➤ Inhibits Ca movement into cardiac and vascular smooth muscle cells by blocking slow Ca channels in cell membranes. This action inhibits smooth muscle cell contractions and causes coronary vasodilation, ↓ myocardial O₂ consumption (effective anti-ischemic agent), reduces PVR and decreases systolic and diastolic pressures.➤ Potent direct negative chronotropic effect (decreases rate)➤ Direct negative inotropic effect (decreases contractile force)➤ Slows conduction time and prolongs refractory period in AV node➤ Interrupts reentry circuit in AV nodal re-entrant tachycardias
Therapeutic benefit:	Slows ventricular response. The coronary vasodilation effects ↓ the frequency and severity of cardiac chest pain.
Indications:	<ul style="list-style-type: none">➤ Paroxysmal Supraventricular tachycardias (PSVT) - Class I dysrhythmias with narrow complex not requiring cardioversion and not responsive to adenosine. 90% effective.➤ AF and flutter w/ rapid ventricular response: effectively slows ventricular response, converts to SR 1/3 of the time.
Dose & route:	<p>Adult: 5 mg slow IVP over 2 minutes; slower (over 3 min) in elderly patients. If necessary, may repeat 5 mg IVP in 15 min. over a 5 min. period. Maximum total dose not to exceed 10 mg in the field. In elderly or those w/ hypotension may cut dose by 50%.</p> <p>Peds per medical control (8-15 years): 0.1-0.3 mg/kg (2-5 mg) IVP over 2 min. May repeat dose: 0.1-0.2 mg/kg slow IVP 15 minutes after initial dose.</p>
Special considerations:	<ul style="list-style-type: none">➤ Usually works within minutes, often gradual slowing of tachydysrhythmias then sudden conversion to SR.➤ CaCl may be effective in reversing ↓ BP that may occur after Verapamil administration, but will not usually affect heart rate.
General precautions:	<ul style="list-style-type: none">➤ Obtain 12-lead ECG prior to administration to R/O AV accessory pathway dysfunction➤ Constant ECG & BP monitoring necessary➤ In stable patient, may attempt vagal maneuvers before using drug➤ Use cautiously in patients on digitalis
Side Effects: Significant!	<ul style="list-style-type: none">➤ CNS: Dizziness, fatigue, headache➤ CV: Sinus arrest, heart blocks, nodal escape rhythms, hypotension; rarely bradycardia & asystole, claudication, peripheral edema➤ Resp: Pulmonary edema, dyspnea, wheezing➤ GI: N/V, abdominal cramps, constipation
Contraindications:	<ul style="list-style-type: none">➤ Existing sick sinus syndrome or other bradyarrhythmias➤ Wide QRS complex tachycardias➤ WPW syndrome; may accelerate ventricular response → VF➤ Impaired ventricular function or heart failure (due to neg inotropic effects) unless secondary to SVT that responds to verapamil➤ Patient on β-blockers (negative inotropic & chronotropic effects)➤ Hypotension; cardiogenic shock➤ High degree heart block unless artificial pacemaker is in place
Drug interactions:	<ul style="list-style-type: none">➤ There may be ↓ BP if used with antihypertensives, nitrates, or quinidine➤ Do not administer to patients suffering from acute hemorrhage➤ Solutions with pH above 6 cause precipitation.

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Study Questions

1. What is the pathophysiology that results in a multi-formed atrial rhythm (wandering atrial pacemaker)?

2. List two causes of WAP:

3. In what type of patient is the presence of a WAP a normal phenomenon?

4. List the ECG features of a WAP:

Rate: _____

Rhythm (Reg or irreg) _____

Pacemaker site: _____

P waves present? _____

P wave configuration: _____

PR interval: _____

QRS present? _____

QRS duration: _____

P/QRS ratio: _____

5. What is the clinical significance of a WAP? _____

6. What is the prehospital treatment for WAP? _____

7. A PAC is known as an _____ beat.

8. From what part of the heart do PACs originate? _____

9. List three causes of PACs

Do PACs always originate from the same site? _____

10. List the ECG characteristics of a sinus rhythm with a PAC:

Rate: _____

Rhythm (Reg or irreg) _____

Pacemaker site: _____

P wave configuration: _____

PR interval: _____

QRS present? _____

QRS duration: _____

P/QRS ratio: _____

11. What would be the configuration of a T wave if a premature P wave was buried in it?

12. What is a PAC w/ aberrant ventricular conduction?

13. What pattern is present is a PAC appears every other beat? _____
Every third beat? _____
If PACs appear in pairs, they are said to be: _____
If three or more PACs occur in a consecutive run, one calls it _____

14. What is the clinical significance of isolated PACs?
What is the clinical significance of frequent PACs?

15. Is there an SOP for PACs?
What is the prehospital management of PACs?

16. What is a non-conducted PAC?

17. What dysrhythmia can be confused with a non-conducted PAC if you do not see the early P wave?

A conscious and alert adult is complaining of a rapid heart rate and palpitations. The patient is aware of what's happening and has a history of PSVT, AF, and A-flutter. History reveals a ventricular septal defect repair and the need for cardioversion last year. Meds: Vasotec, metoprolol, and Vytarin. Vagal maneuvers worked for a time, but the HR increased when the patient stopped. VS: BP 102/82; P 196; R 16; glucose 87; SpO₂ 96%; ECG: atrial tachycardia.

18. Where does atrial tachycardia originate?

19. If atrial tachycardia begins or ends abruptly, occurring in paroxysms, what is it called?

20. AVNRT (PSVT) is often initiated by a _____.

21. List three causes of AVNRT:

22. List the ECG characteristics of an atrial tachycardia:

Rate: _____
Rhythm (Reg or irreg) _____
Pacemaker site: _____
P waves present? _____
P wave configuration: _____
PR interval: _____
QRS present? _____
QRS duration: _____
P/QRS ratio: _____

23. Which of the above patient's medications are usually prescribed to treat cardiovascular disease and what is the classification and action of each?

<u>Drug</u>	<u>Classification</u>	<u>Action</u>
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24. Is this patient's pulse rate surprising in light of his medication history? Why?

25. What is the clinical significance of a PSVT?

26. How should oxygen be given to this patient? _____

27. What is the purpose of vagal maneuvers? _____

What vagal maneuvers should be attempted on this patient? _____

28. What is the first drug indicated for this patient per SOP? _____

How does this drug work? _____

Where should the IV be started? _____

What is the first dose of this drug? _____

Why must it be immediately followed with a saline flush? _____

29. What rhythms may a patient go into transiently following administration of this drug?

List two other side effects of this drug

30. List two contraindications for this drug

31. If the patient fails to respond to the first dose, what is the second dose and how long should an EMT-P wait before giving the 2nd dose?

32. If the patient fails to respond to this drug, what alternative drug is indicated?

An alert and oriented elderly adult is found sitting on the couch c/o of weakness, dizziness and a galloping heart. The patient has a history of cardiac disease and anxiety. Meds: Cardizem (diltiazem), Prilosec, Xanax, and aspirin. VS: BP 108/70; P irregular at 170; ECG: atrial fibrillation; R 18; SpO₂ 94%. Lung sounds are clear bilaterally, skin is pale, cool and dry. The patient weighs 130 lbs.

33. What is the pathogenesis of atrial fibrillation?

34. In what type of patients should AF be anticipated?

35. List the ECG characteristics of atrial fibrillation:

Atrial rate: _____

Ventricular rate controlled: _____

Ventricular rate uncontrolled: _____

Rhythm (Reg or irreg) _____

Pacemaker site: _____

P waves present? _____

P wave configuration: _____

PR interval: _____

QRS present? _____

QRS duration: _____

P/QRS ratio: _____

36. What is the clinical significance of AF?

37. For what major CV complication is a patient with AF at risk?

38. Which of her medications are usually prescribed to treat cardiovascular disease and what is the classification and action of each?

<u>Drug</u>	<u>Classification</u>	<u>Action</u>
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39. What is your paramedic impression? (From what part of the SOP should you begin treatment?)

40. How should oxygen be given to this patient? _____

41. What drug is indicated first for this patient? _____

What is the classification of this drug? _____

What is its expected action? _____

42. How is this medication prepared for administration?

43. What is the specific dose this patient should receive? _____

44. Based on the drug's classification, list two anticipated side effects

45. What treatment is indicated for a patient in PSVT with no chest pain, SOB, or cardiorespiratory compromise?

- A. Transport; support ABCs and reassess cardiorespiratory status enroute
- B. Adenocard 6 mg rapid IV push
- C. Verapamil 5 mg IVP
- D. Right side carotid massage

46. What intervention is indicated in a hemodynamically unstable patient with PSVT?

47. What S&S indicate hemodynamic instability?

48. If a patient is awake and aware, what medication (dose and route) should be given prior to synchronized cardioversion?

49. What should be the initial joule setting for synchronized cardioversion of a narrow QRS complex tachycardia?

Why is it necessary for the monitor to sense the patient's native R waves?

50. At what point in the cardiac cycle will the monitor discharge its current?

51. What is the joule sequencing in a monophasic defibrillator if the patient remains in PSVT?

52. If successful, what treatment should be provided enroute?

If unsuccessful, what treatment should be considered?

53. What is the pathogenesis of atrial flutter?

54. List the ECG characteristics of atrial flutter:

Atrial rate: _____

Ventricular rate _____

Rhythm (Reg or irreg) _____

Pacemaker site: _____

P waves present? _____

P wave configuration: _____

PR interval: _____

QRS present? _____

QRS duration: _____

P/QRS ratio: _____

55. What is the clinical significance of A-Flutter?

56. In what types of patients is A-flutter more likely to occur?