EMERGENCY PHARMACOLOGY I & II

Advanced Cardiac Life Support

Seminole Community College

Based on the Guidelines 2000 for Cardiopulmonary Resuscitation & Emergency Cardiovascular Care

International Consensus on Science

Pharmacology I

Antiarrhythmic Drugs

- Mechanism of Action Adenosine is a endogenous purine nucleoside that depresses AV node and sinus node activity.
- Since most common forms of PSVT involve a reentry pathway including the AV node, Adenosine is effective in terminating these arrhythmias.

- Mechanism of Action cont.- if arrhythmia does not involve a reentry pathway including Atrial Fibrillation or Flutter, Atrial or Ventricular Tachycardias, Adenosine will not be effective in terminating these arrhythmias.
- If this is the case Adenosine may cause AV or Ventricular blocks.

 INDICATIONS - Used in the treatment of Paroxysmal Supraventricular Tachycardia (PSVT) including PSVT in WPW syndrome.

- PRECAUTIONS: Adenosine produces a short lived pharmacologic response because it is rapidly metabolized by enzymatic degradation in the blood and tissues.
- The half-life is approximately 5 seconds or less.

- PRECAUTIONS: Side effects are common but usually short lived; Flushing, dyspnea, chest pain and transient sinus bradycardia and ventricular ectopy after termination of PSVT.
- Should not be used diagnostically for stable, wide complex tachycardias of unknown type (this is a new guideline).

 DOSAGE - 6 mg rapid bolus over 1-3 seconds. Followed by 20 ml flush. A brief period of Asystole (up to 15 seconds) may occur after rapid administration.

 After 1-2 minutes give 12 mg, may repeat a second 12 mg after 1 - 2 minutes. IV should be in anticubital fossa. 92% conversion usually after the first 12 mg dose.

- Mechanisms of Action: effects on sodium, potassium, and calcium channels, also with alpha and beta blocking properties.
- Lengthens the cardiac action potential (antisympathetic action). Negative dromotropic effects on SA node and AV node.
- It has vasodilatory affects that decrease cardiac workload and myocardial oxygen consumption.

- Indications: preferred treatment for atrial and ventricular arrhythmias. Used prior to Lidocaine.
- Treatment and prophylaxis of frequently recurring ventricular fibrillation and hemodynamically unstable ventricular tachycardia in patients refractory to other agents.
- Ventricular rate control of rapid atrial arrhythmias in patients with severely impaired left ventricular function when digitalis is ineffective.

- Indications: ventricular rate control due to accessory pathway conduction in preexcited atrial arrhythmias.
- Adjunct to electrical cardioversion of refractory PSVT's and atrial tachycardia.

- Effective for the control of hemodynamically stable VT, polymorphic VT, and wide-complex tachycardia of uncertain origin.
 - Used after defibrillation and epinephrine in cardiac arrest due to persistent VF or VT.

- Contraindications: hypersensitivity, cardiogenic shock, sinus bradycardia, 2nd or 3rd degree block.
- Hypotension most common side effect.
- Treatment with fluid and temporary pacing can correct hypotension due to Amiodarone in the field!

- Dosage: in cardiac arrest due to pulseless VT or VF 300 mg rapid infusion diluted in 20 to 30 ml of saline or D⁵W. Followed by 150 mg every 3-5 mins until arrhythmia is suppressed.
- Arrhythmias with a pulse: 150 mg over ten minutes followed by 1mg/min infusion for 6 hours. Repeat 150 mg if necessary to a maximum daily (24 hrs)dose of 2.2 grams.
- Maintenance infusion: 0.5 mg/min maximum daily dose of 2.2 grams.

Administration difficulties:

- Contained in an ampule must use filtered needle to draw up.
- Must be drawn up slowly because bubbles will occur if drawn rapidly (soap-like solution).
- Must be given rapidly and only once.
- Many side effects.
- 150 mg/ampule, must draw up two ampules.
- Expensive approximately \$175.00 per 300 mg dose.

ATROPINE SULFATE

 Mechanism of Action: Parasympatholytic drug which enhances both sinus node automaticity, and AV node conduction by reversing cholinergic-mediated affects. Inhibits the release of acetylcholine from the vagus nerve.

ATROPINE SULFATE

 Indications: Ventricular Asystole, Conduction disturbances (Symptomatic 1st or 2nd degree type 1 AV blocks), Symptomatic bradycardia, slow pulseless electrical activity.

ATROPINE

• Precautions:

- Tachydysrhythmias.
- Increased myocardial oxygen consumption
- Use with caution in the presence of acute myocardial ischemia associated with acute myocardial infarction.
- Do not push slowly as paradoxical bradycardia may occur.
- Less than 0.5 mg can cause a further slowing of the rate.

ATROPINE

- Precautions: do not use when bradycardia from AV block at the His-Purkinje level Mobitz (type) II or 3rd degree (complete) heart block with new wide-QRS complexes is suspected.
- Atropine can rarely accelerate sinus rate and AV node conduction.

ATROPINE

• Dosage:

- Asystole & Slow PEA 1 mg IVP and repeated every 3-5 mins if asystole persists.
- Bradycardia & AV Blocks 0.5 mg 1.0 mg every 3 5 mins.
- ET administration: 2.0 2.5 mg diluted in 10 ml NS.
- Maximum dose: 3 mg (0.04 mg/kg) complete vagolytic dose.
- Less than 0.5 mg causes paradoxical bradycardia.
- Maximum dose should be reserved for asystolic cardiac arrest only, this is due to the increase in myocardial oxygen demand!

PROPRANOLOL, METOPROLOL, ATENOLOL, ESMOLOL

- Mechanism of Action Beta blocking agents attentuate the effects of circulating catecholamines by blocking their ability to bind to beta receptors.
- Reduces heart rate, blood pressure, myocardial contractility and therefore myocardial oxygen consumption.
 Decreases (depresses) the pumping function of the heart.

PROPRANOLOL, METOPROLOL, ATENOLOL, ESMOLOL

- Mechanism of Action benefits in patients with acute coronary syndromes, including patients with non-Q wave MI and unstable angina.
- Therefore beta blockers should be administered in these patients under these conditions!

PROPRANOLOL, METOPROLOL, ATENOLOL, ESMOLOL

- Indications Primary indication is to control recurrent ventricular tachycardia ventricular fibrillation and supraventricular arrhythmias.
- **Precautions:** Hypotension, Congestive heart failure and bronchospasm.
- **Dosage** Varies with desired effect. Currently not used in the field!

BRETYLIUM TOSYLATE (Bretylol)

 Indications – no longer indicated for treatment of ventricular arrhythmias, due to the decrease in availability and it's documented ineffectiveness.

Has been removed from all algorhythms.

 Mechanism of Action - Both are calcium channel blocking agents that slow conduction and increase refractoriness in the AV node. These actions terminate reentrant arrhythmias that require AV nodal conduction.

 Verapamil is a negative inotropic agent that causes a reduction in myocardial oxygen requirement. May also control ventricular response in A-Fib, A-Flutter, or multifocal Atrial Tachycardia.

 Indications - Used in the treatment of Paroxysmal Supraventricular Tachycardia (PSVT) narrow complex and ventricular rate control in Atrial Fibrillation. However, Adenosine is the drug of choice.

 Precautions - Possible hemodynamic compromise. Should not be used in WPW syndrome or impaired heart function.

 Calcium is used for possible overdose of Verapamil or other Calcium channel blocker.

Dosage:

Verapamil: 2.5 - 5.0 mg IV over 2 minutes. Repeat doses of 5 - 10 mg every 15 to 30 minutes to a maximum of 30 mg. Diltiazem: 0.25 mg/kg over 2 minutes followed by 0.35 mg/kg. Produces less myocardial depression than Verapamil.

DISOPYRAMIDE

- Mechanism of Action: antiarrhythmic agent that acts to slow conduction velocity, and prolongs the effective refractory period (similar to Procainamide).
- Potent anticholinergic, negative inotropic, and hypotensive effects.
- Not used in the field!

FLECAINIDE

- Mechanism of Action: potent sodium channel blocker with significant conduction-slowing effects.
- Indications: ventricular arrhythmias and for patients in supraventricular arrhythmias with structural heart disease. IV version (not approved in U.S.) has corrected A-fib, Ectopic A-tachycardia, A-flutter.
- **Precautions:** must be administered slowly, bradycardia, hypotension, and neurological abnormalities.

IBUTILIDE

- Mechanism of Action: short-acting antiarrhythmic drug. Effective by prolonging the action potential duration and increasing the refractory period of cardiac tissue.
 - Indications: acute pharmacologic
 conversion of Atrial flutter or A-Fib
 when electrical cardioversion has failed.

IBUTILIDE

- Dosage: Adults over 60 kg, IV 1 mg/10 ml over 10 minutes. Repeat if unsuccessful 1mg/10ml over 10 minutes.
- If under 60 kg administer 0.01 mg/kg and repeat in second dose.
- **Precautions:** minimal effects on the heart rate and BP. However. May cause ventricular proarrhymias especially when there is an impaired LV.

ISOPROTERENOL(Isuprel)

 Mechanism of Action - Isoproterenol is a pure beta adrenergic agent. Potent inotropic and chronotropic agent that increases cardiac output and myocardial oxygen demand.

Isoproterenol can increase myocardial **ischemia and exacerbates arrhythmias.**

ISOPROTERENOL(Isuprel)

• Indications - Refractory torsades de pointes (Chemical overdrive pacing) and immediate control of hemodynamically significant bradycardia especially in the denervated hearts of heart transplant patients. However, not the treatment of choice, used until a pacemaker, atropine, dobutamine is available and has failed.
ISOPROTERENOL(Isuprel)

 Precautions - ventricular ectopy and dysrhythmias. Electronic pacing preferred!

Dosage - 2 to 10 mcq/min titrated to effect. 1 mg/250 ml fluid infusion.

LIDOCAINE (Xylocaine)

 Mechanism of Action - Suppresses ventricular arrhythmias by decreasing the automaticity of phase 4 depolarization. Depresses conduction in reentrant pathways. Elevates fibrillation threshold. Decreases excitability of ischemic tissue.

- Indications: Used in the treatment of:
 - VF/ Pulseless VT that persists after defibrillation and epinephrine (Class Indeterminate).
 - Control of hemodynamically compromising PVC's (Class Indeterminate).
 - Hemodynamically stable VT (Class IIb)
- Lidocaine is the second choice medication behind other alternate agents: Amiodarone, Procainamide, Sotalol)

 Precautions - Routine prophylactic administration is no longer recommended in uncomplicated acute MI or ischemia without PVC's.

• Toxic - therapeutic balance is delicate.

- Dosage In cardiac Arrest
- Initial dose of 1.0 1.5 mg/Kg
- Additional boluses of 0.5 .75 mg/Kg repeat in 3 - 5 minutes; maximum total dose of 3 mg/Kg.
- The more aggressive dosing approach is recommended in cardiac arrest (1.5mg/kg).

- Dosage In V-Tach with a pulse or other ventricular ectopic beats 0.5 – 0.75 mg/Kg, 3 mg/Kg maximun dose.
- Infusion: 2-4 mg/min
- Mix 1 Gm in 250 ml NS or 2 Gm in 500 ml.

- In Patients older than 70 years old, and in those with Hepatic dysfunction should receive the initial normal loading dose.
- The maintenance infusion should be reduced by 50%.
- Infusion rate of 1 4 mg/minute (class indeterminate).

- Special Considerations:
- Endotracheal Dose: 2 4 mg/kg
- Reappearance of arrhythmias during a constant infusion of Lidocaine should be treated with a small bolus dose (0.5 mg/kg).

- Toxic reactions and Side effects:
 - Slurred speech
 - Altered consciousness
 - Muscle twitching
 - Seizures
 - Bradycardia

MAGNESIUM SULFATE

Mechanism of Action -

Magnesium deficiency is associated with cardiac arrhythmias, symptoms of cardiac insufficiency, and sudden cardiac arrest.

• Hypomagnesemia can precipitate refractory V-Fib.

MAGNESIUM SULFATE

 Indications - Used in the treatment of Ventricular Fibrillation / Ventricular Tachycardia after full doses of Amiodarone and Lidocaine have failed to convert rhythm. Treatment of choice for Torsades de pointes.

MAGNESIUM SULFATE

- Should only be used when arrhythmias may be caused by Magnesium deficiency or Torsades de Pointes.
- Dosage 1 to 2 grams loading dose mixed in 10 ml of solution and administered over 1 to 2 minutes.

 Mechanism of Action – supresses both atrial and ventricular arrhythmias. Acceptable for the pharmacological conversion of supraventricular arrhythmias (particularly A Fib and A Flutter) to sinus rhythm (class IIa).

 Supresses ventricular ectopy similar to Lidocaine. Recommended when Amiodarone and Lidocaine is contraindicated or it has failed to suppress ventricular ectopy.

 Increases ventricular fibrillation threshold. Shortens effective refractory period of the AV node, lengthens refractory period in bundle of his.

 Indications - Secondary to Amiodarone and Lidocaine in the field. Used when both medications have failed to suppress the life threatening ventricular arrhythmias. Suppresses PVC's and recurrent Vtach, V-fib.

 Precautions - Contraindicated in Torsades de Pointes, hypotension after rapid injection. Adverse ECG effects. Use caution in acute MI.

- DOSAGE Infusion of 30 mg/min until one of the following is observed:
 - 1. Arrhythmia is suppressed
 - 2. Hypotension ensues
 - 3. QRS widens by 50%
 - 4. Total of 17 mg/kg administered (1.2 gm/70kg)
- Maintenance Infusion rate: 1 4 mg/minute

PROPAFENONE

- Mechanism of Action: Antiarrhythmic agent with significant conduction-slowing and negative inotropic effects with additional non-selective beta blocking properties.
 - Used in U.S. orally only for treatment of supraventricular and ventricular arrhythmias in patients without structural heart disease.

PROPAFENONE

- Indications: ventricular arrhythmias and for patients in supraventricular arrhythmias with structural heart disease. IV version (not approved in U.S.) has corrected A-fib, Ectopic Atachycardia, A-flutter.
- Dosage: No field dose

SOTALOL

- Mechanism of Action: prolongs action potential duration like Amiodarone, and increases cardiac tissue refractoryness.
- Used in U.S. orally only for treatment of supraventricular and ventricular arrhythmias in patients without structural heart disease.
- Dosage: No field dose

Pharmacology II

Agents to Optimize Cardiac Output and Blood Pressure

 Mechanisms of Action: Naturally occurring catecholamine with both Alpha and Beta adrenergic properties (Sympathomimetic agent).

• Greatest benefit from alpha adrenergic stimulating properties.

• Mechanisms of Action:

- Increases myocardial and cerebral blood flow during CPR
- Increased systemic vascular resistance
- Increased arterial blood pressure
- Increased heart rate (Chronotropic effects)
- Increased myocardial contraction (Inotropic effect). Increased automaticity
- Increased myocardial oxygen requirement

- Indications: "First Agent in All Forms of Cardiac Arrest"
 - Improves V-Fib conversion, PEA, May restore electrical activity in Asystole.
 - Vasopressor agent for symptomatic bradycardia (not first line drug).
 - Vasopressin may be substituted in the V-fib algorhythm.

- Precautions: May precipitate or excerbate myocardial ischemia. Do not mix with alkaline solutions. Ventricular ectopy in digitalized patients.
- Remember the increase in Myocardial Oxygen Demand!

• Dosage:

- 1 mg of 1: 10,000 solution IVP every 3-5 minutes followed by 20 ml flush.
- ET administration: 2 2.5 mg in 10 ml.
- Infusion: 1 mg in 250 ml, dose 1 mcq/min.

VASOPRESSIN

- Mechanism of Action: naturally occurring antidiuretic hormone that acts as a non-adrenergic peripheral vasoconstrictor.
- Indications: May be substituted for Epinephrine in the V-fib / V-tach without a pulse algorithm (class IIb).

VASOPRESSIN

- Mechanism of Action: during a short duration of V-Fib, during CPR increased coronary perfusion pressure, and vital organ blood flow.
- Vasopressin does not increase myocardial oxygen demand because of the lack of beta adrenergic stimulation.
- Vasopressin remains intact during acidosis.

VASOPRESSIN

- Half-life 10 20 minutes
- Dosage: 40 U (units) IVP
- May follow up with Epinephrine after 10

 20 minutes of initial administration.
- May later be used for PEA and Asystole.
- Inexpensive to purchase and easy to administer.

NOREPINEPHRINE (Levophed)

 Mechanism of Action - Naturally occurring catecholamine that is a potent alpha receptor agonist and vasoconstrictor. Causes a great increase in myocardial oxygen demand and will exacerbate myocardial ischemia.

NOREPINEPHRINE (Levophed)

 Indications – (None in the field) Treatment for hemodynamically significant hypotension that is refractory to other sympathomimetic amines. Should be considered as a last and

temporary measure.

NOREPINEPHRINE (Levophed)

- Precautions Increases myocardial oxygen requirement and will exacerbate ischemia. If drug infiltrates 5 to 10 mg of Phentolamine should also be infiltrated to prevent tissue necrosis and sloughing.
- Dosage 0.5 1.0 mcq/minute titrated to effect. 4 mg placed in 250 ml D⁵W.

DOPAMINE (Intropin)

 Mechanism of Action - A chemical precursor of norepinephrine that has both alpha and beta actions, and stimulates dopaminergic receptors in a dose dependant fashion.

• **Dopamine** stimulates the heart through beta receptors.

DOPAMINE (Intropin)

- Indications: usually reserved for hypotension that occurs with symptomatic bradycardia or post cardiac arrest. Goal BP 90 mm Hg.
- Precautions: dose dependant, higher doses may have a profound negative impact on the heart. Do not use in the same IV lines as Bicarbonate, may inactivate. Extreme tissue destruction if IV infiltrates.

DOPAMINE (Intropin)

 Other Precautions - SVT or ventricular arrhythmias. MAO inhibitors may potentiate the effects of dopamine, use 1/10th the normal dose. Alkaline solutions inactivate dopamine. Must be tapered off, use central line due to risk of infiltration causing severe damage.
DOPAMINE (Intropin)

- Dosage: in low doses 1-2 mcq/Kg/min produces vasodilation of renal, mesenteric, and cerebral arteries by stimulation dopaminergic receptors.
- At midrange doses 2 10 mcq/Kg/min produces cardiovascular effects (Beta).

DOPAMINE (Intropin)

 At higher doses 20 mcq/Kg/min produce hemodynamic effects similar to norepinephrine (peripheral arterial vasoconstriction).

DOPAMINE

• Field Dosage -

5 mcq/Kg/min titrated to effect. Final recommended dose dosage range is 5 - 20 mcq/kg/min. **Dopaminergic effect:** 1 – 2 mcq/kg **Beta effect:** 2 –10 mcq/kg

Alpha effect:10 – 20 mcq/kg

DOBUTAMINE (Dobutrex)

- Mechanism of Action synthetic sympathomimetic amine that exerts its potent inotropic effects by stimulating beta-1 and alpha adrenergic receptors in the myocardium and blood vessels.
- Dobutamine has beneficial hemodynamic effects and its lack of norepinephrine release minimize its effects on myocardial oxygen demand. DOES NOT PRODUCE RENAL AND MESENTERIC VASODILATION LIKE DOPAMINE.

DOBUTAMINE (Dobutrex)

- Indications Treatment of pulmonary congestion and severe systolic heart failure.
- **Precautions** High doses may cause myocardial ischemia, SVT and V-tach.
- **Dosage** 2 to 20 mcq/Kg/min.

AMRINONE (Inocor)& MILRINONE

- Mechanism of Action Rapid-acting inotropic agent who net effects are similar to dobutamine. Cardiac output increases and peripheral vascular resistance and preload are diminished.
- Indications Severe congestive heart failure refractory to diuretics or cardiogenic shock.

AMRINONE (Inocor)& MILRINONE

- Precautions May induce or worsen myocardial ischemia, and ventricular ectopy.
- **Dosage** 0.75 mg/Kg every 2 3 minutes followed by 5 to 15 mcq/Kg/min infusion.

CALCIUM CHLORIDE

 Mechanism of Action - Although Calcium ions play a critical role in myocardial contractile performance and impulse formation, however studies have not shown benefit from the use of calcium and in fact may be detrimental.

CALCIUM CHLORIDE

 Indications - Hyperkalemia, Hypocalcemia and channel blocker toxicity (verapamil) class IIb.

Dosage - 2 mg - 4 mg/Kg repeat if necessary in 10 minute intervals.

DIGITALIS

- Mechanism of Action Rapid-acting inotropic agent who net effects are similar to dobutamine.
- Cardiac output increases and peripheral vascular resistance and preload are diminished.
- Extremely limited use in Emergency Cardiac Care.

DIGITALIS

- Indications decreases ventricular rate in Atrial flutter and Atrial fibrillation. No longer preferred method.
- Precautions May induce or worsen myocardial ischemia, and ventricular ectopy (all lethal forms of dysrhythmias have occurred).
- Dosage 10 15 mcq/Kg every 2 3 minutes followed by 5 to 15 mcq/Kg/min infusion.

NITROGLYCERINE

- Mechanism of Action Organic nitrate that relaxes vascular smooth muscle
- Indications Primary indication is to relieve Angina Pectoris, initial treatment of choice for ischemic-type pain or chest discomfort.
- Parenteral choice for the treatment of congestive heart failure, management of uncomplicated MI.

NITROGLYCERINE

- Precautions: Hypotension, Headache.
- Dosage 0.3 mg 0.4 mg every 3 5 minute intervals if discomfort is not relieved.
- Infusion 50 or 100 mg / 250 ml, rate 10 to 20 mcg/min increased by 5 – 10 mcg every 5 - 10 mins until desired clinical response.

SODIUM NITROPRUSSIDE

 Mechanism of Action - Sodium Nitroprusside is a potent peripheral vasodilator, effects are seen immediately and cease within minutes after infusion is discontinued.

SODIUM NITROPRUSSIDE

- Indications Parenteral treatment of choice for severe heart failure and hypertensive emergencies when immediate reduction of peripheral resistance is necessary.
- Nitroglycerine is preferred because it is less likely to lower coronary perfusion pressure, and likely to increase perfusion to the myocardium.

SODIUM NITROPRUSSIDE

- Precautions Hypotension is the most common adverse reaction seen with nitroprusside. Hypotension may precipitate myocardial ischemia, infarction or stroke.
- Dosage 0.1 to 5 mcq/Kg/min titrated to effect. Mix 50 100 mg/250 ml D5W.

 Mechanism of Action - Sodium Bicarbonate reacts with the hydrogen ions in the blood to form carbon dioxide and water to buffer metabolic acidosis.

- Indications Used during cardiac resuscitation only after defibrillation, effective CPR, Endotracheal intubation, hyperventilation with 100% oxygen and more than one dose of epinephrine.
- May be used with the following preexisting conditions:metabolic acidosis, hyperkalemia, or tricyclic or phenobarbital overdose.

- Precautions The major problem is that NaHCO3 has a high carbon dioxide content, the CO2 crosses rapidly into the cells causing an increase in intracellular hypercarbia and acidosis especially in myocardial and cerebral cells. Bicarbonate crosses much more slowly.
 - Other problems include: Hypernatremia and a shift in the oxyhemoglobin saturation curve preventing O2 release to the tissues. Metabolic Alkalosis. Do not mix with catecholamines.

- **Dosage 1 mEq/Kg initially, 0.5 mEq/Kg dose every 10 minutes thereafter.**
- Should be guided by blood gases if possible. Use after the first ten minutes in cardiac arrest, however not recommended by the AHA.

DIURETICS (Furosemide)

 Mechanism of Action - Rapidly acting potent diuretic that inhibits the reabsorption of sodium in the renal loop of henle. Has a direct venodilating effect in patients with pulmonary edema onset approximately 5 minutes. Diuresis occurs later.

DIURETICS (Furosemide)

- Indications Treatment of pulmonary edema associated with left ventricular failure.
- Precautions Dehydration, hypotension, eletrolyte depletion in coronary heart disease.
- Dosage: 0.5 1.0 mg/Kg initially IV slowly over 1 2 minutes.

MORPHINE

Mechanism of Action - Narcotic analgesic

 manifests analgesic and hemodynamic
 effects, increasing venous capacitance
 and reduces systemic vascular resistance,
 reduces preload, relieving pulmonary
 congestion. Reduces intramyocardial wall
 tension, which decreases myocardial
 oxygen requirements. Reduces anxiety.

MORPHINE

- Indications Drug of choice in treatment of pain and anxiety associated with AMI and in the Treatment of acute pulmonary edema.
- Precautions Respiratory depression, hypotension. Overdose can be corrected with IV Naloxone (Narcan) 0.4 -0.8 mg.

MORPHINE

 Dosage - 1 mg - 3 mg IV slowly over 1 to 5 minutes until the desired effect is achieved.

THROMBOLYTIC AGENTS (Anistreplase, Streptokinase, Alteplase)

 Mechanism of Actions - Activate both soluble plasminogen and surface bound plasminogen to plasmin. Pharmacolgic thrombolysis occurs when surface-bound plasminogen is converted to surface bound plasmin which digests fibrin and dissolves the clot.

THROMBOLYTIC AGENTS (Anistreplase, Streptokinase, Alteplase)

- Indications Should be initiated immediate after the onset of chest pain (within 6-12 hours, ideally within 6 hours).
- **Precautions** Bleeding is the major complication as a result of thrombolytic therapy. Various contraindications for this type of therapy.
- **Dosage** Varies with type of drug used.
- New Fibrolytic Agents now used: Ativase.

SYMPTOMATIC BRADYCARDIA

• Remember the following sequence:

1st line -2nd line -3rd line -4th line - Atropine (except high block) Pacemaker Dopamine Epinephrine

The End

- This Microsoft PowerPoint presentation was prepared by Rob Holborn Ed.D, EMT-P, Seminole Community College.
- The presentation was prepared by using the textbook: Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: International Consensus on Science and the 1998 National EMT-Paramedic Curriculum