

## ACLS Emergency Drugs

DRUG	CLASSIFICATION	ACTION	INDICATION
ACE Inhibitors		Prevents conversion of angiotension I to II, a potent vasoconstrictor.	HTN & CHF
Adenosine	Antiarrhythmic	<ul style="list-style-type: none"> <li>-Restores Normal sinus rhythm by interrupting re-entrant pathways in the AV node.</li> <li>-Slows conduction time through the AV node.</li> <li>-Produces Coronary Artery Vasodilation.</li> </ul>	<ul style="list-style-type: none"> <li>- Conversion of Paroxysmal-Supraventricular Tachycardia (PSVT) to normal sinus rhythm when vagal maneuvers are unsuccessful.</li> <li>- As a diagnostic agent to asses myocardial perfusion defects.</li> </ul>
Albuterol (Ventolin)	Bronchodilator (sympatho- mimetics)	<ul style="list-style-type: none"> <li>-Binds to beta2-adrenergic receptors in airway smooth muscle, &amp; thus activate adenyl-cyclase &amp; increased level of cyclic-3, 5 adenosine monophosphate (cAMP).</li> <li>-Relaxation of airway smooth muscle with subsequent bronchodilation.</li> </ul>	<ul style="list-style-type: none"> <li>-Acute Bronchospasm &amp; for prevention of exercise induced asthma.</li> <li>- Reversible airway obstruction caused by asthma or COPD.</li> <li>- Long term control agent in patients with chronic /persistent bronchospasm.</li> </ul>
Alteplase (Activase,tPA)	Thrombolytic Agent	<ul style="list-style-type: none"> <li>-Directly Activate Plasminogen-Alteplase</li> <li>-Exerts its action on the endogenous fibrinolytic system to convert plasminogen to plasmin</li> <li>-This is done by directly hydrolyzing the arginine-valine bond in plasminogen</li> </ul>	<ul style="list-style-type: none"> <li>-Acute Myocardial Infarction</li> <li>-Coronary Artery</li> <li>-Thrombosis</li> <li>-Ischemic Stroke</li> <li>-Occluded IV Catheter</li> <li>-Pulmonary Embolism</li> </ul>

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DRUG	CLASSIFICATION	ACTION	INDICATION
Amiodarone (Cordarone, Pacerone)	Antiarrhythmic (Class III)	-Prolongs action potential & refractory period  -Inhibits adrenergic stimulation  -Slows the sinus rate, increases PR and QT intervals  -Decreases peripheral vascular resistance	-Ventricular Fibrillation  -Supra-ventricular Tachycardia
Aspirin (ASA)	-Platelet Inhibitor  -Salicylate	-Decreases Platelet Aggregation	-Acute Myocardial Infarction  -Angina  -Arterial Thromboembolism  -Prophylaxis  -Arthralgia  -Ischemic Stroke
Atropine	Antiarrhythmics  Anticholinergics (antimuscarinic)	-Accelerates the HR by blocking parasympathetic nervous system.  -Enhances sinus node automaticity and AV Conduction. Effects of Atropine on the heart is almost entirely to its electrical activity.	-Symptomatic Bradycardia; Bradycardia with PVC's; Asystole; PEA with rate < 60 after Epinephrine; May be useful in AV blocks – 1 <sup>st</sup> degree AVB, 2 <sup>nd</sup> Degree Type I (Wenckebach); Watch carefully for slowing.
Atropine Sulfate	Parasympatholytic	-Inhibits actions of acetylcholine at postganglionic parasympathetic neuroeffector sites, primarily at muscarinic receptors	-Hemodynamically Significant Bradycardia; Asystole; Organophosphate Poisoning; Pulseless Electrical Activity

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Betablockers		-a – adrenergic receptor blockade; Anti-dysrhythmic	-Effective antiarrhythmic; Decreased incidence of VF in post MI patients; May decrease rate of nonfatal re-infarction; Slows HR & treat rhythm problems; Decreased myocardial ischemia by decreasing O <sub>2</sub> requirements; Decreased blood pressure.
Calcium Channel Blockers	Mineral and electrolyte replacements/supplements	-Act as an activator Inhibit the flow of Ca <sup>+</sup> across the cell membrane Relax arterial smooth muscle  -Slow rate of SA node & AV node conduction  -Prevents reentry arrhythmia's Slow HR & Decrease myocardial O <sub>2</sub> demand	-Emergency treatment of hyperkalemia and hypermagnesemia and adjunct in cardiac arrest.  -Hypocalcaemia.  -Treatment for essential hypertension; Prophylaxis of angina pectoris; Prevent/control SVT & Sinus Tachycardia; Prevent neurologic damage due to subarachnoid hemorrhage
Calcium Chloride	Electrolyte	-Maintain cell membrane and capillary systems; act as an activator in the transmission of nerve impulses and contraction of cardiac, skeletal, and smooth muscle	-Hypocalcemia -Hyperkalemia -Hypermagnesemia
Dextrose 50%	Nutrient	Glucose Replacement	-Hypoglycemia -Hyperkalemia

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Diltiazem (Cardizem)	<ul style="list-style-type: none"> <li>-Antiarrhythmic (class IV)</li> <li>-Antianginal</li> <li>-Antihypertensive Agent</li> </ul>	<ul style="list-style-type: none"> <li>-Inhibits the transport of calcium into myocardial and vascular smooth muscle cells; resulting in inhibition of excitation-contraction coupling and subsequent contraction</li> <li>- Systemic vasodilation resulting in decreased blood pressure.</li> <li>-Coronary vasodilation</li> <li>-Suppression of arrhythmias</li> </ul>	<ul style="list-style-type: none"> <li>-Angina pectoris and vasospastic (Prinzmetal's) angina.</li> <li>- Supraventricular tachycardia and rapid ventricular rates in atrial flutter or fibrillation.</li> <li>- Management of Raynaud's syndrome.</li> </ul>
Diphenhydramine (Benadryl)	<ul style="list-style-type: none"> <li>Antihistamine</li> <li>Allergy</li> <li>Cold Remedies</li> <li>Antitussives</li> </ul>	<ul style="list-style-type: none"> <li>-Antagonizes the effects of histamine at H1 receptor sites ; does not bind to or inactivate histamine</li> <li>-Significant CNS depressant and anticholinergic properties</li> </ul>	<ul style="list-style-type: none"> <li>-Anaphylaxis</li> <li>-Seasonal and perennial allergic rhinitis</li> <li>-Allergic dermatosis</li> <li>-Parkinson's disease and dystonic reactions from medications.</li> <li>-Mild nighttime sedation</li> <li>-Prevention of motion sickness</li> <li>-Antitussive (Syrup only).</li> </ul>
Digitalis	<ul style="list-style-type: none"> <li>Antiarrhythmic</li> <li>Inotropic</li> </ul>	<ul style="list-style-type: none"> <li>- Improves myocardial contraction by altering the calcium utilization by myocardial cells</li> <li>-Prolongs refractory period of the AV node</li> <li>-Decreases conduction through the SA and AV node</li> </ul>	<ul style="list-style-type: none"> <li>-Congestive Heart Failure (CHF)</li> <li>-atrial Fibrillation</li> <li>-atrial flutter</li> </ul>

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Digoxin (Lanoxin)	Antiarrhythmic  Inotrope	-Increases the force of myocardial contraction -Prolongs refractory period of the AV node -Decreases conduction through the SA and AV nodes	Atrial Fibrillation  Heart Failure
Dopamine (Intropics, Vasopressors)	Sympathomimetic  Inotropics  Vasopressor	-Stimulates dopaminergic and beta1-adrenergic receptors ; producing cardiac stimulation and renal vasodilation (Small Doses).  -Cardiac stimulation and renal vasodilation (Large doses).	-Adjunct to standard measures to improve: Blood pressure, Cardiogenic Shock, Cardiac output, Urine output in treatment of shock unresponsive to fluid replacement.  -Heart Failure  -Hypotension  -Septic Shock
Dobutamine	Sympathomimetic  Inotropics	-Stimulates beta-1 (myocardial) – adrenergic receptors with relatively minor effect on heart rate or peripheral blood vessels.  -dobutamine is mostly a beta-1 selective synthetic catecholamine with mild alpha-1 and beta-2 activity and therefore has mostly positive inotropic and chronotropic effects on the heart; dobutamine will often increase cardiac output with less tachycardia and arrhythmia than dopamine	-Short term (<48 hrs) management of heart failure caused by depressed contractility from organic heart disease or surgical procedure.  -Used for Pump problems with systolic blood pressure 90-100 CHF, Pulmonary Congestion & Cardiogenic-Pulmonary Edema  -Hypotension and Shock

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Epinephrine (Adrenalin)	Antihistamics  Bronchodilators  Vasopressors	<ul style="list-style-type: none"> <li>-Results in accumulation of cAMP at beta adrenergic receptors.</li> <li>-Affects both beta (cardiac) adrenergic receptors and beta (pulmonary) adrenergic receptors.</li> <li>-Produces bronchodilation</li> <li>-Alpha-adrenergic agonist properties</li> <li>-Resulting in vasoconstriction</li> <li>-Inhibits the release of mediators of immediate hypersensitivity reactions from mast cells</li> </ul>	<ul style="list-style-type: none"> <li>-Management of reversible airway disease due to asthma or COPD, severe allergic reactions.</li> <li>-Acute Bronchospasm</li> <li>-Anaphylaxis</li> <li>-Angioedema</li> <li>-Asthma; Bronchospasm Prophylaxis</li> <li>-Cardiac Arrest; Glaucoma</li> <li>Pulseless Electrical Activity</li> <li>Ventricular Asystole</li> <li>Ventricular Fibrillation</li> <li>Surgical Bleeding</li> </ul>
Furosemide (Lasix)	Diuretic	<ul style="list-style-type: none"> <li>-Inhibit the reabsorption of sodium and chloride from the loop of Henle and distal renal tubule</li> <li>-Increase renal excretion of water, sodium, chloride, magnesium, hydrogen, and calcium</li> </ul>	<ul style="list-style-type: none"> <li>-Edema</li> <li>-Heart Failure</li> <li>-Hypertension</li> <li>-Nephrotic Syndrome</li> <li>-Pulmonary Edema</li> <li>-Renal Impairment</li> </ul>
Fibrinolytic Therapy		<ul style="list-style-type: none"> <li>-Thrombolytic Enzymes binds to fibrin, converts plasminogen to plasmin initiating local fibrinolysis.</li> </ul>	<ul style="list-style-type: none"> <li>-Lysis of thrombi obstructing coronary arteries in Acute MI; Acute Ischemic Stroke; Pulmonary Embolism</li> </ul>

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Fibrolitic Agents		<p>-Activate both soluble plasminogen and surface bound plasminogen to plasmin when generated close to fibrin clot digest fibrin and dissolves the clot.</p>	<p>-Lysis of thrombi obstructing coronary arteries in acute MI; Ideally within the first 6 hours of onset of symptoms; Re-establish blood flow to infarct related artery; Goal door to drug &lt; 30 min.; Acute Ischemic Stroke with 3 hours of onset of symptoms</p>
Heparin	Anticoagulant	<p>-In low doses, prevents the conversion of prothrombin to thrombin by its effect on factor Xa;</p> <p>-Higher doses neutralize thrombin, preventing the conversion of fibrinogen to fibrin</p>	<p>-Various thromboembolic disorders including: Venous thromboembolism, pulmonary emboli, atrial fibrillation with embolization, acute and chronic consumptive coagulopathies, peripheral arterial thromboembolism.</p> <p>-Acute Myocardial Infarction</p> <p>-Arterial thromboembolism</p> <p>-Deep Venous Thrombosis</p> <p>-Unstable Angina</p> <p>-Prosthetic Heart Valves</p>
Ibutilide	Antiarrhythmics ( Class III)	<p>-Decrease the heart rate and AV conduction by activating a slow inward Na Current</p> <p>-Mildly slows sinus rate and AV conduction.</p>	<p>-Short acting antiarrhythmic; Treatment for recent onset Atrial Fibrillation and Atrial Flutter – 3 hours to 90 days</p>

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Isoproterenol (Isuprel)	Sympathomimetic Inotrope  Adrenergic Agonist	-Potent Agonist of both Beta1- and Beta2- adrenergic receptors -Intracellularly, the actions are mediated by cyclic adenosine monophosphate  -The production of which is augmented by beta stimulation.	-Acute Bronchospasm -Asthma -AV Block -Bradycardia -Cardiac Arrest -Adams-Stokes Syndrome -Torsade De Pointes
Lidocaine (Xylocaine)	Antiarrhythmic (Class IB) Anesthetic	-Suppresses automaticity and spontaneous depolarization of the ventricles during diastole by altering the flux of sodium ions across cell membranes with little or no effect on heart rate  -Inhibiting transport of ions across neuronal membranes	-Ventricular arrhythmias -Anesthesia, Pain -Sympathetic Nerve Block -Ventricular Fibrillation -Ventricular Tachycardia
Magnesium		-Plays important role in neurotransmission and muscular excitability.	-Torsades de Pointes – Drug Induced; Suspected hypomagnesium; Sever recurrent or refractory V-fib
Magnesium Sulfate	Electrolyte	-Essential for the activity of many enzymes -Plays an important role in neurotransmission and muscular excitability	-Hypomagnesemia -Eclampsia -Preeclampsia



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Midazolam	Antianxiety agents, sedative/hypnotics	-Depressant action at all levels of CNS	-Conscious sedation for cardioversion  - Relieve anxiety and produce amnesia
Morphine	Opioid Analgesics	-Causes Vasodilation which reduces myocardial oxygen consumption  -Analgesia Sedation	-Severe pain  -Pulmonary Edema due to CHF  -Pain associated with AMI (f NTG not working)  -Dilate blood vessels  -Emergency treatment of Cardiogenic-Pulmonary Edema
Morphine Sulfate	Analgesic	-Binds to opiate receptors in the CNS  -Alters the perception of and responds to painful stimuli while producing generalized CNS depression	-Severe Pain  -Pulmonary Edema  -Pain associated with Myocardial Infarction
Naloxone (Narcan)	Opiate Antagonist	-Competitively blocks the effects of opioids, including CNS and respiratory depression ,without producing any agonist effects.	-Reversal of CNS depression and respiratory depression because of suspected opioid overdose i.e. Opiate Agonist Overdose  -Opiate Agonist-Induced respiratory Depression

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Nitroglycerin	Antianginals	<ul style="list-style-type: none"> <li>-Increase coronary blood flow by dilating coronary arteries and improving collateral flow to ischemic regions.</li> <li>-Vasodilator</li> <li>-Reduces myocardial oxygen consumptions</li> <li>-Decreases Preload (end-diastolic volume), &amp; left ventricular end-diastolic pressure.</li> </ul>	<ul style="list-style-type: none"> <li>-Acute and long term management of angina pectoris.</li> <li>-Adjunct treatment of CHF Associated with acute Myocardial Infarction</li> <li>-Heart Failure</li> <li>-Hypertension Pulmonary Edema Unstable Angina</li> </ul>
Nitroprusside	Antihypertensives	<ul style="list-style-type: none"> <li>-Potent rapid acting peripheral vasodilator reduces peripheral arterial pressure (preload &amp; afterload)</li> </ul>	<ul style="list-style-type: none"> <li>-Hypertension crises</li> <li>-Controlled hypotension during anesthesia</li> <li>-Cardiac pump failure or Cardiogenic Shock</li> <li>-AMI, if Nitroglycerin not effective</li> </ul>
Norepinephrine (Levophed, Noradrenaline)	<ul style="list-style-type: none"> <li>Sympathomimetic</li> <li>Inotrope</li> <li>Vasopressor</li> </ul>	<ul style="list-style-type: none"> <li>-It is an endogenous catecholamine synthesized in the adrenal medulla</li> <li>-It is the biochemical precursor of epinephrine</li> <li>-Norepinephrine is the transmitter of most sympathetic post ganglionic fibers of the CNS.</li> </ul>	<ul style="list-style-type: none"> <li>-Cardiogenic Shock</li> <li>-Hypotension</li> <li>-Septic Shock</li> </ul>

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Oxygen		<ul style="list-style-type: none"> <li>-Improves tissue oxygenation and hemoglobin saturation when circulation is maintained.</li> </ul>	<ul style="list-style-type: none"> <li>-Acute Chest Pain</li> <li>-Hypoxia</li> <li>-Cardiopulmonary Arrest</li> <li>-Pulmonary Edema</li> </ul>
Phenytoin (Dilantin, DPH)	Anticonvulsant Antiarrhythmic (Class IB)	<ul style="list-style-type: none"> <li>-Antiarrhythmic properties as a result of improvement in AV conduction</li> <li>-Limit seizure propagation by altering ion transport</li> <li>-Decrease synaptic transmission</li> </ul>	<ul style="list-style-type: none"> <li>-Seizure ( Tonic- clonic – grand-mal seizures &amp; complex partial seizures).</li> <li>-Status Epilepticus</li> </ul>
Procainamide (Pronestyl)	Antiarrhythmic ( Class IA)	<ul style="list-style-type: none"> <li>-Decreases myocardial excitability</li> <li>-Slows conduction velocity</li> <li>-May depress myocardial contractility</li> </ul>	<ul style="list-style-type: none"> <li>-Treatment of wide variety of ventricular and atrial arrhythmias, including; PVC's, Ventricular tachycardia, Paroxysmal atrial tachycardia</li> <li>- Maintenance of normal sinus rhythm after conversion from atrial fibrillation or flutter.</li> <li>-Cardiopulmonary Resuscitation</li> </ul>
Quinidine	Antiarrhythmic Class IA	<ul style="list-style-type: none"> <li>-Decreases myocardial excitability</li> <li>-Slows conduction velocity (Prolongs the QT interval)</li> </ul>	<ul style="list-style-type: none"> <li>Atrial pre-mature contraction</li> <li>-Premature ventricular contraction</li> <li>-Paroxysmal atrial tachycardia</li> <li>-Venricular tachycardia</li> </ul>

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Reteplase (Retavase)	Thrombolytic Agent	-Directly Activate Plasminogen Alteplase exerts its action on the endogenous fibrinolytic system -To convert plasminogen to plasmin by directly hydrolyzing the arginine-valine bond in plasminogen.	-Acute Myocardial Infarction  -Coronary Artery Thrombosis
Sodium Bicarbonate	-Alkalinizing Agent  -Electrolyte Replacement	-Acts as an alkalinizing agent by releasing bicarbonate ions  -Following oral administration releases bicarbonate, which is capable of neutralizing gastric acid	-Cardiac Arrest  -Cardiopulmonary Resuscitation  -Dyspepsia  -Hyperkalemia  -Metabolic Acidosis  -Pyrosis  -Urinary Alkalinization
Sotalol	Antiarrhythmic (Class II and III)	Blocks stimulation of beta1 (myocardial) and beta2 (pulmonary and vascular)	Life-threatening ventricular arrhythmias  -Maintenance of normal sinus rhythm in patients with highly symptomatic atrial fibrillation/atrial flutter

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Streptokinase (Streptase)	Thrombolytic Agent	<ul style="list-style-type: none"> <li>-Directly Activate Plasminogen</li> <li>-Alteplase exerts its action on the endogenous fibrinolytic system</li> <li>-To convert plasminogen to plasmin by directly hydrolyzing the arginine-valine bond in plasminogen</li> </ul>	<ul style="list-style-type: none"> <li>-Acute management of coronary thrombosis (MI)</li> <li>-Arterial thrombo-embolism</li> <li>-Deep Venous Thrombosis</li> <li>-Pulmonary Embolism</li> <li>-Occluded Arteriovenous Cannula</li> </ul>
Tenecteplase (TNKase)	Thrombolytic Agent	<ul style="list-style-type: none"> <li>-Directly Activate Plasminogen;</li> <li>-Alteplase exerts its action on the endogenous fibrinolytic system</li> <li>-To convert plasminogen to plasmin by directly hydrolyzing the arginine-valine bond in plasminogen</li> </ul>	<ul style="list-style-type: none"> <li>-Acute Myocardial Infarction</li> <li>-Coronary Artery</li> <li>-Thrombosis</li> </ul>
Vasopressin (Pitressin, ADH)	Pituitary Hormones (antidiuretic hormones)	<ul style="list-style-type: none"> <li>-Alters the permeability of the renal collecting ducts, allowing reabsorptions of water</li> <li>-Directly stimulates musculature of the GI tract</li> <li>-Nonadrenergic peripheral Vasoconstrictor</li> </ul>	<ul style="list-style-type: none"> <li>-Central diabetes insipidus due to deficient AHD</li> </ul>

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Verapamil (Isoptin)	<ul style="list-style-type: none"> <li>-Antiarrhythmic</li> <li>-Antianginal</li> <li>- Antihypertensive Agent</li> <li>-Vascular headache suppressants</li> </ul>	<ul style="list-style-type: none"> <li>-Inhibits the transport of calcium into myocardial and vascular smooth muscle cells</li> <li>-Resulting in inhibition of excitation-contraction coupling and subsequent contraction</li> </ul>	<ul style="list-style-type: none"> <li>-Management of: Angina pectoris or vasospastic (Prinzmetal's) angina, Atrial Fibrillation, Atrial Flutter, Hypertension, Paroxysmal Supraventricular Tachycardia (PSVT)</li> </ul>
Disopyramide	Antiarrhythmic Class I	<ul style="list-style-type: none"> <li>-Decreases myocardial excitability</li> <li>-Slows conduction velocity</li> <li>-Little effect on heart rate but has a direct negative inotropic effect</li> </ul>	<ul style="list-style-type: none"> <li>Unifocal or multifocal PVCs</li> <li>-Paired PVCs</li> <li>-Ventricular tachycardia</li> <li>-Supraventricular tachyarrhythmia</li> </ul>

## DEFINITIONS

<i>Adrenergic</i>	Activated by, characteristic of, or secreting epinephrine or related substances, particularly the sympathetic nerve fibers that liberate norepinephrine at a synapse when a nerve impulse passes.
<i>Adrenergic Agonist</i>	is a drug that has an affinity for and stimulates physiologic activity at cell receptors normally stimulated by naturally occurring substances
<i>Cholinergic</i>	means related to the neurotransmitter acetylcholine, and is typically used in a neurological perspective. The parasympathetic nervous system is entirely <i>cholinergic</i> . Neuromuscular junctions, preganglionic neurons of the sympathetic nervous system, the basal forebrain, and brain stem complexes are also <i>cholinergic</i> .
<i>Diabetes Insipidus</i>	is a disease of the pituitary gland or kidney. Most people show some of the same signs as someone with diabetes mellitus-they have to urinate often, are very thirsty and hungry, and feel weak
<i>Embolism</i>	An embolism is an obstruction in a blood vessel due to a blood clot or other foreign matter that gets stuck while traveling through the bloodstream. The plural of embolism is emboli.
<i>Embolus</i>	An <b>embolus</b> is most often a piece of a thrombus that has broken free and is carried toward the brain by the bloodstream. The term thromboembolus is used a lot because it turns out that most emboli arise from thrombi. However, bits of plaque, fat, air bubbles, and other material also qualify as emboli. Presumably an embolus floats along with the flowing blood until it encounters a narrowing in an artery through which it cannot pass. When the embolus gets stuck, it blocks the artery. This reduces blood flow to downstream tissues and causes them to become ischemic.
<i>Hyperkalemia</i>	higher than normal levels of potassium in the circulating blood; associated with kidney failure or sometimes with the use of diuretic drugs
<i>Inotrope</i>	an agent that alters force or energy of muscular

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	contractions. Negatively <i>inotropic</i> agents weaken the force of muscles
<i>A parasympatholytic element, also referred to as anticholinergics,</i>	reduces the activity of the parasympathetic nervous system. (The parasympathetic nervous system is often colloquially described as the "Feed and Breed" or "Rest and Digest" portion of the autonomic nervous system. The parasympathetic nervous system becomes strongly engaged during or after a meal and during times when the body is at rest.)
<i>Sympathomimetic drugs</i>	are substances that mimic the effects of the catecholamines, epinephrine or adrenaline, norepinephrine (noradrenaline), and/or dopamine.
<i>Sympatholytic</i>	drug inhibits the postganglionic functioning of the sympathetic nervous system. They can all be used as an antihypertensive.
<i>Thrombolytic agent</i>	A drug that is able to dissolve a clot (thrombus) and reopen an artery or vein. Thrombolytic agents may be used to treat heart attack, stroke, deep vein thrombosis, pulmonary embolism, and occlusion of a peripheral artery or indwelling catheter.
<i>Thrombolytic agents</i>	Serine proteases (they digest protein) Convert plasminogen to plasmin This breaks down the fibrinogen and fibrin and dissolves the clot.
<i>Thrombus</i>	A thrombus is a solid mass of platelets and/or fibrin (and other components of blood) that forms locally in a vessel. Thrombi form when the clotting mechanism is activated. This is supposed to happen when you are injured. However, it can also occur at the site of an ulcerated atherosclerotic plaque or wherever the endothelial cells lining the inner surface of an artery have been damaged.
<i>Vasopressor</i>	Producing constriction of the blood vessels and a consequent rise in blood pressure