Drug Guide

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Verapamil

List of Home Medications
ACETAMINOPHEN

Classes: Analgesic; antipyretic

Trade Names: Abenal (Can), Aceta, Acephein, Actamen, Actimol (Can), Anacin-3, Anuphen, Apacet, APAP, Atasol (Can), Banesin, Dapa, Datril, Dolanex, Dorcol, Dymadon (Aus), Exdol (Aus), Feverall, Genapap (Can), Genebs, Halenol, Liquiprim, Mapap, Medacap, Neopap, Oraphen, Panadol, Panamax (Aus), Panex, Paralgin (Aus), Pedric, Redutemp, Robigesic (Can), Rounox (Can), Snaplets, St. Joseph, Supap, Tapanol, Tempra, Tenol, Tylenol, Typap, Valadol, Valorin

Therapeutic Action/Pharmacodynamics: Acetaminophen is a clinically proven analgesic/antipyretic. Acetaminophen produces analgesia by elevation of the pain threshold and antipyresis through action on the hypothalamic heat-regulating center. Acetaminophen is equivalent to aspirin in analgesic and antipyretic effectiveness. Unlike aspirin, acetaminophen has little effect on platelet function, does not affect bleeding time, and generally produces no gastric bleeding. It is unlikely to produce many of the side effects associated with aspirin and aspirin-containing products.

Emergency Uses: Acetaminophen is used as substitute for aspirin, when the latter is not tolerated or is contraindicated, to reduce fever and/or to temporarily relieve mild to moderate pain. Adult dose: 325–650 mg PO every 4–6 hr (maximum 4 g/day). 650 mg PR every 4–6 hr (maximum 4 g/day). Pediatric dose: 15 mg/kg every 4–6 hr.

Pharmacokinetics

Absorption: Rapid and almost complete absorption (60–70%) from GI tract; less complete absorption (30–40%) from rectal suppository; peak effect in 1–2 hr; duration is 3–4 hr; half-life is 1–3 hr.

Distribution: Well distributed in all body fluids; crosses placenta.

Metabolism: Extensively metabolized in liver.
Elimination: 90–100% excreted as metabolites in urine; excreted in breast milk.

Contraindications and Precautions: Acetaminophen is contraindicated in patients with hypersensitivity to acetaminophen or phenacetin. It is also contraindicated in children under 3 yr, unless directed by a physician; avoid repeated administration to patients with anemia or hepatic disease. Use with caution in arthritic or rheumatoid conditions affecting children under 12 yr; alcoholism; malnutrition; thrombocytopenia.

Adverse/Side Effects: Acute poisoning: CNS: Dizziness, lethargy • GI: Anorexia, nausea, vomiting, epigastric or abdominal pain, diarrhea, onset of hepatotoxicity, hepatic coma, acute renal failure (rare) • Other: Diaphoresis, chills, elevation of serum transaminases (ALT, AST) and bilirubin, hypoglycemia.

Interactions: With chronic coadministration, barbiturates, carbamazepine, phenytoin, and rifampin may increase the potential for chronic hepatotoxicity. Chronic, excessive ingestion of alcohol will increase risk of hepatotoxicity.

Prehospital Considerations
• Individuals with poor nutrition or who have ingested alcohol over prolonged periods are prone to hepatotoxicity even from moderate acetaminophen doses.
• Overdosing and chronic use can cause liver damage and other toxic effects.
• Acetaminophen should not be used for self-medication of pain for more than 10 days in adults or for more than 5 days in children without consulting a physician. It should not be used for fever persisting longer than 3 days and never for fever over 39.5°C (103°F) or for recurrent fever without medical direction. No more than 5 doses in 24 hr should be given to children unless prescribed by a physician.
ACTIVATED CHARCOAL

Class: Adsorbant

Trade Names: Actidose, Actidose-Aqua, CharcoAid, Charcocaps, Charcodote, InstaChar, LiquiChar, SuperChar

Therapeutic Action/Pharmacodynamics: Activated charcoal is a fine black powder that adsorbs many drugs and chemicals. It acts by binding (adsorbing) toxic substances, thereby inhibiting their GI adsorption, enterohepatic circulation, and thus bioavailability. It has a tremendous surface area, allowing for a large amount of adsorption; the combined complex formed by the adsorption process is excreted from the body in the feces. It is a general-purpose emergency antidote in the treatment of poisoning by most drugs and chemicals, e.g., acetaminophen, aspirin, atropine, barbiturates, digitalis glycosides, phenytoin, propoxyphene, strychnine, and tricyclic antidepressants, among many others.

Emergency Use: To treat acute ingested poison. Adult dose: 1 g/kg mixed with at least 6–8 oz water PO or via nasogastric tube. Pediatric dose: Same as adult.

Pharmacokinetics

Absorption: Not absorbed; onset is immediate; peak effect, duration, and half-life are unknown.

Elimination: Excreted in feces.

Contraindications and Precautions: Activated charcoal is contraindicated for treatment of poisonings by cyanide, mineral acids, caustic alkalis, organic solvents, iron, ethanol, and methanol.

Adverse/Side Effects: GI: Vomiting following rapid ingestion of high doses, abdominal cramping, abdominal bloating, constipation (diarrhea from sorbitol additive).

Interactions: May decrease absorption of all other oral medications—administer at least 2 hr apart.
Prehospital Considerations
• Before using charcoal as an antidote, contact your medical
direction physician or your poison control center for advice.
• Activated charcoal tablets or capsules are less adsorptive and
thus less effective than powder or liquid form; therefore,
they are not recommended in treatment of acute poisoning.
• Charcoal is most effective when administered as soon as pos-
sible after acute poisoning (preferably within 30 min). In an
emergency, stir activated charcoal into tap water to make a
slurry (about 20−30 g in at least 240 mL of water).
• Activated charcoal can be swallowed or given through a
nasogastric tube. If administered too rapidly, your patient
may vomit.
• If necessary, palatability may be improved by adding a small
amount of concentrated fruit juice or chocolate powder to
the slurry. Reportedly, these agents do not appreciably alter
adsorptive activity.

ADENOSINE
Class: Antidysrhythmic
Trade Name: Adenocard
Therapeutic Action/Pharmacodynamics: Adenosine is a nat-
urally occurring nucleoside that is present in all body cells.
Adenosine slows conduction time through the AV node, can
interrupt the reentry pathways through the AV and sinoatrial
(SA) nodes, and can restore normal sinus rhythm in patients
with paroxysmal supraventricular tachycardia (PSVT), includ-
ing PSVT associated with Wolff-Parkinson-White syndrome.
Adenosine is antagonized competitively by methylxanthines
such as caffeine and theophylline, and potentiated by blockers
of nucleoside transport such as dipyridamole. Its rapid onset
and short half-life make adenosine a very safe and effective
treatment for PSVT. The usual IV bolus dose of 6 or 12 mg adenosine will have few systemic hemodynamic effects.

**Emergency Use:** To convert PSVT to a sinus rhythm in patients refractory to common vagal maneuvers. *Adult dose:* 6 mg rapid IV bolus (1–2 seconds) followed by a rapid saline flush; may repeat in 1–2 min at 12 mg. May repeat one more time in 1–2 min at 12 mg. *Pediatric dose:* 0.1 mg/kg rapid IV bolus (1–2 seconds) followed by a rapid saline flush; may repeat once in 1–2 min at 0.2 mg/kg. Maximum single dose is 12 mg.

**Pharmacokinetics**

*Absorption:* Rapid uptake by erythrocytes and vascular endothelial cells after IV administration; onset and peak effect within 20–30 seconds, half-life is 10 seconds.

*Metabolism:* Rapid uptake into cells; degraded by deamination to inosine, hypoxanthine, and adenosine monophosphate.

*Elimination:* Route of elimination is unknown.

**Contraindications and Precautions:** Because it slows conduction through the AV junction, adenosine is contraindicated in preexisting second- and third-degree AV block. It may actually produce a short lasting first-, second- or third-degree heart block. In extreme cases, transient asystole may occur. Because of the short half-life (10 seconds), this usually lasts only a few seconds and resolves without intervention. Do not use in patients with sinus node disease, such as sick sinus syndrome or symptomatic bradycardia, except in patients with a functioning pacemaker. In the presence of atrial flutter or atrial fibrillation, a transient modest slowing of ventricular response may occur immediately following adenosine administration. Adenosine has been administered to a limited number of patients with asthma and mild to moderate exacerbation of their symptoms has been reported. Respiratory compromise has occurred during adenosine infusion in patients with obstructive pulmonary disease. Adenosine should be used with caution in patients with obstructive lung disease not associated with bron-
choconstriction (e.g., emphysema, bronchitis, etc.) and should be avoided in patients with bronchoconstriction or bronchospasm (e.g., asthma). Adenosine should be discontinued in any patient who develops severe respiratory difficulties. Never use in patients with a known hypersensitivity to the drug.

**Adverse/Side Effects:**

- **CV:** Facial flushing, headache, sweating, palpitations, atrial fibrillation or flutter, chest pain, hypotension
- **Respiratory:** Shortness of breath/dyspnea, chest pressure, hyperventilation, head pressure
- **CNS:** Lightheadedness, dizziness, tingling in arms, numbness, apprehension, blurred vision, burning sensation, heaviness in arms, neck and back pain
- **GI:** Nausea, metallic taste, tightness in throat, pressure in groin.

**Interactions**

IV adenosine has been effectively administered in the presence of other cardioactive drugs, such as quinidine, beta-adrenergic blocking agents, calcium channel blocking agents, and angiotensin-converting enzyme inhibitors, without any change in the adverse reaction profile. The effects of adenosine are antagonized by methylxanthines such as caffeine and theophylline. In the presence of these methylxanthines, larger doses of adenosine may be required or adenosine may not be effective. Adenosine effects are potentiated by dipyridamole. Thus, smaller doses of adenosine may be effective in the presence of dipyridamole. Carbamazepine has been reported to increase the degree of heart block produced by other agents. Because the primary effect of adenosine is to decrease conduction through the AV node, higher degrees of heart block may be produced in the presence of carbamazepine.

**Prehospital Considerations**

- For rapid bolus IV, administer the drug into a large proximal vein and follow with a rapid saline flush.
- The solution must be clear at time of use. Since it contains no preservatives, discard used portion.
Monitor ECG, BP, and heart rate every 15–30 seconds for several minutes after administration.

Inform your patient that facial flushing and transient symptoms may occur.

ALBUTEROL

**Class:** Sympathomimetic bronchodilator

**Trade Names:** Asmol (Aus), Proventil, Respolin (Aus), Ventolin

**Therapeutic Action/Pharmacodynamics:** Albuterol is a relatively selective beta2 adrenergic. The prime action of beta-adrenergic drugs is to stimulate adenyl cyclase, the enzyme that catalyzes the formation of cyclic-3’, 5’-adenosine monophosphate (cyclic AMP) from adenosine triphosphate (ATP). The cyclic AMP causes relaxation of the smooth muscles of the bronchial tree, decreasing airway resistance, facilitating mucus drainage, and increasing vital capacity. It exerts minimal effects on beta1 (heart) or alpha (peripheral vasculature) receptors. In therapeutic doses, albuterol, by inhibiting histamine release from mast cells, also reduces the mucus secretion, capillary leaking, and mucosal edema caused by an allergic response in the lungs.

**Emergency Uses:** To relieve bronchospasm in patients with reversible obstructive airway disease (asthma, chronic bronchitis, emphysema) and acute attacks of bronchospasm. Adult dose: 90 µg via metered-dose inhaler (2 sprays) or 2.5 mg in 2.5–3.0 mL of NS via nebulizer, may repeat as needed. Ventolin is also supplied in Rotacaps for use in a Rotahaler. Two 200 mg caps should be placed and inhaled. May repeat in 6 hr. Pediatric dose: 0.15 mg/kg in 2.5–3.0 mL of NS via nebulizer, may repeat as needed.
Pharmacokinetics

Absorption: Onset is 5−15 min inhaled; peak effect is 1−1.5 hr; duration is 3−6 hr, half-life is less than 3 hr.
Distribution: When inhaled, albuterol is distributed to muscle cells along the bronchial tree. Very little is systemically absorbed and distributed.
Metabolism: Metabolized in liver; may cross the placenta.
Elimination: 76% of dose eliminated in urine in 3 days.

Contraindications and Precautions: Never use for patients with a known hypersensitivity to the drug.

Adverse/Side Effects: CNS: tremors, anxiety, dizziness, seizures, headache, insomnia • GI: nausea, dyspepsia • ENT: pharyngitis, nasal congestion • CV: palpitations, tachycardia, hypertension • Respiratory: bronchospasm, cough, wheezing.

Interactions: Other sympathomimetic aerosol bronchodilators or epinephrine should not be used concomitantly with albuterol. Albuterol should be administered with extreme caution to patients being treated with monoamine oxidase (MAO) inhibitors or tricyclic antidepressants, since the action of albuterol on the vascular system may be potentiated. Beta-receptor blocking agents and albuterol inhibit the effect of each other. Since albuterol may lower serum potassium, care should be taken in patients also using other drugs that lower serum potassium as the effects may be additive.

Prehospital Considerations

• Your patient may be taking albuterol in an oral form. The most common adverse effect associated with oral drug is fine tremor in fingers, which may interfere with precision handwork.
• Expect children 2−6 yr old to be more prone to symptoms of CNS stimulation (hyperactivity, excitement, nervousness, insomnia), tachycardia, and GI symptoms. Rarely do patients receive adequate directions for correct use of their medica-
tion and inhaler. Do not assume that they have administered their drug properly.

- Significant subjective improvement in pulmonary function should occur within 60–90 min after drug administration. Reevaluate your patient’s condition often and repeat albuterol therapy when indicated.

**ALTEPLASE RECOMBINANT (tPA)**

**Class:** Thrombolytic  
**Trade Names:** Actilyse (Aus), Activase  
**Therapeutic Actions/Pharmacodynamics:** This recombinant DNA-derived form of human tissue-type plasminogen activator (tPA) is a thrombolytic agent. tPA promotes thrombolysis by forming the active proteolytic enzyme plasmin. Plasmin is capable of degrading fibrin, fibrinogen, and factors V, VIII, and XII.

**Emergency Uses:** To thrombolyse in acute myocardial infarction and acute ischemic stroke.  
**Adult dose:** 15 mg IV, then 0.75 mg/kg (up to 50 mg) over 30 min, then 0.5 mg/kg (up to 35 mg) over 60 min.  
**Pediatric dose:** Not used.  
To thrombolyse in pulmonary embolism.  
**Adult dose:** 100 mg IV infusion over 2 hr.  
**Pediatric dose:** Not used.  

**Pharmacokinetics**  
**Absorption:** Onset and peak effects 5–10 min after infusion completed; half-life is 26.5 min.  
**Metabolism:** Metabolized in liver.  
**Elimination:** Excreted in urine.  

**Contraindications and Precautions:** Alteplase is absolutely contraindicated in patients with active internal bleeding, suspected aortic dissection, traumatic CPR (rib fractures, pneumothorax), history of recent hemorrhagic stroke (within 6 months), recent (within 2 months) intracranial or intraspinal
surgery or trauma, intracranial tumors, uncontrolled hypertension, pregnancy, or severe allergic reactions to either anistreplase or streptokinase. Use with caution in patients with recent major surgery (within 10 days), cerebral vascular disease, recent GI or GU bleeding, recent trauma, hypertension, age greater than 75, hemorrhagic ophthalmic conditions; use with caution in patients on oral anticoagulants.

**Adverse/Side Effects:** Hematologic: Internal and superficial bleeding (cerebral, retroperitoneal, GU, GI).

**Interactions:** Use with caution in patients using other anticoagulant therapy.

**Prehospital Considerations**
- IV infusion of alteplase should be started as soon as possible after the thrombolytic event, preferably within 6 hr for AMI; 3 hr for stroke.
- The 100-mg vial does not contain a vacuum. Follow manufacturer’s directions and use supplied transfer device for reconstitution.
- Do not exceed a total dose of 100 mg. Higher doses have been associated with intracranial bleeding.
- Follow infusion of drug by flushing IV tubing with 30–50 mL of NS or D5W.
- While patient is receiving this medication, do not allow patient out of bed.
- Check vital signs frequently. Be alert to changes in cardiac rhythm. Dysrhythmias may signal the need to stop therapy.
- Monitor for excess bleeding every 15 min for the first hour of therapy, every 30 min for second to eighth hour, then every 8 hr.
- Monitor neurologic checks throughout drug infusion every 30 min and then every hour thereafter for the first 8 hr after infusion.
- Spontaneous bleeding occurs twice as often with alteplase as with heparin. Protect patient from invasive procedures. IM
injections are contraindicated. Also avoid physical manipulation of patient during thrombolytic therapy to prevent bruising.

**AMINOPHYLLINE**

**Class:** Methylxanthine bronchodilator

**Trade Names:** Aminophylline, Phyllocontin, Somophyllin, Truphyllin

**Therapeutic Action/Pharmacodynamics:** Methylxanthines cause bronchodilation in a way different from the sympathomimetics. They prolong the effects of beta agonists by blocking the enzyme (phosphodiesterase) that biodegrades them. As a result, the beta₂ effects (bronchodilation and decreased mucus secretion) are prolonged. For this reason, they also produce mild cardiac and central nervous system stimulation and promote diuresis. Although methylxanthines are primarily used for long-term airway maintenance in COPD, aminophylline is sometimes effective for patients refractory to sympathomimetics and other bronchodilators. Efficacy in acute asthma is controversial. It is not indicated for routine treatment of acute exacerbation of asthma in patients who are receiving optimal therapy with inhaled beta₂-adrenergic agonists and steroids.

**Emergency Uses:** To relieve bronchospasm secondary to asthma or COPD (emphysema, chronic bronchitis). *Adult dose:* 250–500 mg over 20–30 min IV infusion. *Pediatric dose:* 6 mg/kg over 20–30 min; maximum dose should not exceed 12mg/kg/24hr.

To relieve bronchospasm in congestive heart failure patients in which additional fluid therapy is contraindicated; also as a cardiac stimulant and diuretic for patients in congestive heart failure. *Adult dose:* 250–500 mg in 20 ml (via Buretrol or Volutrol container) over 20–30 min IV infusion.
Pharmacokinetics

Absorption: Rapid absorption into bloodstream; onset and peak effect in 15 min; duration varies with age, smoking, and liver function (4–8 hr).

Distribution: Crosses placenta.

Metabolism: Extensively metabolized in liver.

Elimination: Parent drug and metabolites excreted by kidneys; excreted in breast milk.

Contraindications and Precautions: Aminophylline is contraindicated in patients with a hypersensitivity to methylxanthines or patients with uncontrolled cardiac dysrhythmias. Use with extreme caution in patients with cardiac disease or hypertension. Also use with caution in patients with impaired liver function; diabetes mellitus; hyperthyroidism; glaucoma; prostatic hypertrophy; fibrocystic breast disease; history of peptic ulcer; COPD; acute influenza or in patients receiving influenza immunization; in neonates and young children; and in patients over age 55.

Adverse/Side Effects: CNS: nervousness, restlessness, depression, insomnia, irritability, headache, dizziness, muscle hyperactivity, convulsions • CV: cardiac dysrhythmias, tachycardia, with rapid IV: hyperventilation, chest pain, severe hypotension, cardiac arrest • GI: nausea, vomiting, anorexia, hematemesis, diarrhea, epigastric pain.

Interactions: In patients with acute exacerbations who are currently taking theophylline-containing preparations (Slo-bid, Theo-dur), serum theophylline levels should be determined prior to the administration of aminophylline, particularly if there are any signs of toxicity. Theophylline preparations interact with many drugs. Cimetidine, erythromycin- and quinolone-class antibiotics can elevate serum theophylline levels.

Prehospital Considerations

• Aminophylline has a very narrow therapeutic range. While administering aminophylline, closely monitor your patient
for signs of hypotension, dysrhythmias, and convulsions. A sudden, sharp, unexplained rise in heart rate is a useful clinical indicator of toxicity. Minor symptoms of toxicity often do not precede cardiac arrhythmias or seizures.

- Rapid infusion of IV aminophylline may cause cardiac arrest. Monitor infusion rate carefully.
- Do not use aminophylline solutions if discolored or if crystals are present.
- The elderly, acutely ill, and patients with severe respiratory problems, liver dysfunction, or pulmonary edema are at greater risk of toxicity because of reduced drug clearance.
- Children appear to be more susceptible than adults to the CNS-stimulating effects of xanthines (nervousness, restlessness, insomnia, hyperactive reflexes, twitching, convulsions). Dosage reduction may be indicated.
- Smoking (tobacco or marijuana) tends to increase aminophylline elimination (reduces half-life) and therefore dosage requirements may be higher and dosage intervals shorter than in nonsmokers.
- Many popular OTC remedies for treatment of asthma or cough contain ephedrine in combination with various forms of methylxanthines. Always include over-the-counter medications in your history and watch for signs of toxicity.

AMIODARONE

**Classes:** Antidysrhythmic

**Trade Names:** Aratac (Aus), Cordarone, Cordarone X (Aus), Pacerone

**Therapeutic Action/Pharmacodynamics:** Amiodarone is a unique antidysrhythmic. Totally unrelated to other antidysrhythmics, it acts directly on all cardiac tissues. It is thought to prolong the duration of the action potential and refractory period without significantly affecting the resting membrane poten-
The IV formulation relaxes vascular smooth muscle, decreases peripheral vascular resistance, and increases coronary blood flow. Amiodarone also blocks effects of sympathetic stimulation.

**Emergency Uses:** To treat life-threatening ventricular and supraventricular dysrhythmias, particularly atrial fibrillation. 

**Adult Dose:** 150–300 mg IV over 10 min followed by 1mg/min over next 6 hr. Maintenance dose is 0.5 mg. The total daily dose should not exceed 2 g. Oral loading dose is 800–1,600 mg/day in 1–2 doses, with a maintenance dose of 400–600 mg/day. **Pediatric Dose:** 5 mg/kg IV/IO by rapid bolus. Maximum dose is 15 mg/kg. Oral loading dose 5–15 mg/kg/day divided in 1–2 doses, with a maintenance dose of 5 mg/kg/day.

**Pharmacokinetics**

Intravenous: Rapid distribution of amiodarone following IV administration, serum concentrations decline to 10% of peak values within 30–45 minutes. Metabolism and elimination are primarily hepatic. No established relationship between concentration and therapeutic response with short-term IV use. Oral: Oral amiodarone is 50% absorbed. The onset of action is 2–3 days. Peak levels are attained at 3–7 hours. Distribution is widespread and includes adipose tissues, lungs, kidneys, and spleen. Elimination is hepatic with a half-life of 40–55 days is common following oral administration. It crosses the placenta and can be found in breast milk.

**Contraindications and Precautions:** IV amiodarone is contraindicated in patients with a known hypersensitivity to the drug. Because it may decrease automaticity, conductivity, and contractility, do not use IV amiodarone in the presence of cardiogenic shock, severe sinus bradycardia, or advanced AV block unless a pacemaker is available. Oral amiodarone is also contraindicated when episodes of bradycardia have caused syncope except when used in conjunction with a pacemaker.
Because it is metabolized in the liver, use with caution in patients with severe liver disease. Also use with caution during pregnancy (category D) and in nursing women; should not be used in children.

**Adverse/Side Effects:** CNS: Peripheral neuropathy (muscle weakness, wasting numbness, tingling), fatigue, abnormal gait, dyskinesias, dizziness, paresthesia, headache. • CV: Bradycardia, hypotension (IV), sinus arrest, cardiogenic shock, CHF, dysrhythmias; AV block • Eye: corneal microdeposits, optic neuritis, optic neuropathy, blurred vision, permanent blindness, corneal degeneration, macular degeneration, photosensitivity • GI: Anorexia, nausea, vomiting, constipation • Respiratory (pulmonary toxicity): Alveolitis, pneumonitis (fever, dry cough, dyspnea), interstitial pulmonary fibrosis • Skin: Slate-blue pigmentation, photosensitivity, rash • Other (with chronic use): angiodema, hyperthyroidism or hypothyroidism; may cause neonatal hypo- or hyperthyroidism if taken during pregnancy.

**Interactions:** Amiodarone significantly increases digoxin levels and enhances pharmacologic effects and toxicities of disopyramide, procainamide, quinidine, flecainide, and lidocaine. It also enhances the anticoagulant effects of oral anticoagulants. Calcium channel blockers and beta blockers may potentiate sinus bradycardia, sinus arrest, or AV block. Amiodarone may increase phenytoin levels 2- to 3-fold. Ritonavir may increase cardiotoxicity. Additional interactions include fentanyl, cyclosporine, cholestyramine, and cimetidine.

**Prehospital Considerations**
- During IV infusion, carefully monitor blood pressure and slow the infusion if significant hypotension occurs. Bradycardia should be treated by slowing the infusion or discontinuing it if necessary. Sustained monitoring is essential because drug has an unusually long half-life.
- Report adverse reactions promptly. Bear in mind that long
elimination half-life means that drug effects will persist long after dosage adjustments are made or drug is discontinued.

- Be alert to signs of pulmonary toxicity: progressive dyspnea, fatigue, cough, pleuritic pain, fever.
- Auscultate chest periodically or when patient complains of respiratory symptoms. Check for diminished breath sounds, rales, pleuritic friction rub; observe breathing pattern. Drug-induced pulmonary function problems must be distinguished from CHF or pneumonia. Keep your medical direction physician informed.
- Monitor heart rate and rhythm and BP until drug response has stabilized. Report promptly symptomatic bradycardia.
- Patients already receiving antidysrhythmic therapy when amiodarone is started must be closely observed for adverse effects, particularly conduction disturbances and exacerbation of dysrhythmias. Dosage of previous agent should be reduced by 30–50% several days after amiodarone therapy is started.

**AMRINONE (Inamrinone)**

**Class:** Cardiac inotrope

**Trade Name:** Inocor

**Therapeutic Action/Pharmacodynamics:** Amrinone is a class of cardiac inotropic agents with vasodilator activity. It is a phosphodiesterase inhibitor whose mode of action differs from that of the digitalis glycosides and beta-adrenergic stimulants. In patients with depressed myocardial function, it enhances myocardial contractility, increases cardiac output and stroke volume, and reduces right and left ventricular filling pressure, pulmonary capillary wedge pressure (PCWP), and systemic vascular resistance. It is often used when traditional therapies such as digitalis, diuretics, vasodilators, and conventional inotropes have failed for patients with severe congestive heart failure.
Emergency Uses: Adult dose: 0.75 mg/kg IV bolus given slowly over 2–3 min, followed by an infusion of 5–15 µg/kg per min. An additional bolus, if needed, can be given in 30 min. Amrinone is used to support cardiac output and systemic vascular resistance in children with septic shock or myocardial dysfunction, such as dilated cardiomyopathy or following cardiac surgery. Pediatric dose: 0.75-1.0 mg/kg over 5 min. If the patient tolerates this load, it may be repeated 2 times up to a total load of 3 mg/kg, followed by an infusion of 5-10 µg/kg/min IV.

Pharmacokinetics
Absorption: Onset in 2–5 min; peak effect in 10 min; duration is 0.5–2 hr. Distribution: Unknown if it crosses placenta or into breast milk.
Metabolism: Metabolized in liver.
Elimination: Excreted primarily in urine.

Contraindications and Precautions: Amrinone is contraindicated in patients with a known hypersensitivity to amrinone or to bisulfites. Use with caution in patients with CHF immediately following acute MI as it may increase myocardial ischemia.

Adverse/Side Effects: CV: Hypotension, dysrhythmias • GI: Nausea, vomiting, anorexia, abdominal cramps • Hematologic: Asymptomatic thrombocytopenia (decreased platelets).

Interactions: Amrinone should not be mixed in dextrose- or sodium bicarbonate-containing solutions or administered into an IV line containing furosemide as it may precipitate.

Prehospital Considerations
• Natural color of IV amrinone is clear yellow. Discard discolored solutions and those that contain a precipitate. Store at 15–30C (59–86F) unless otherwise directed. Protect ampules from light.
• During IV administration, monitor BP, heart rate, and respirations and keep your medical direction physician informed.
If your patient’s BP falls or if dysrhythmias occur, slow or stop the infusion immediately.

- Monitor infusion site to prevent extravasation.
- The chief measurement used to evaluate patient response is relief of symptoms of CHF.
- Amrinone IV preparation contains sodium metabisulfite, a reducing agent to which certain susceptible individuals are allergic. Drug should be discontinued immediately if patient manifests clinical symptoms suggestive of a hypersensitivity reaction.

**AMYL NITRITE**

**Class:** Nitrate vasodilator  
**Trade Name:** Amyl Nitrite

**Therapeutic Action/Pharmacodynamics:** Amyl nitrite is a short-acting vasodilator and smooth muscle relaxant with actions, contraindications, and adverse reactions similar to those of nitroglycerin. Its action in the treatment of cyanide poisoning is based on ability of amyl nitrite to convert hemoglobin to methemoglobin, which forms a nontoxic complex with the cyanide ion to form cyanomethemoglobin, which can be enzymatically degraded. Amyl nitrite is supplied in a glass ampule that is broken and its contents immediately inhaled.

**Emergency Use:** As an adjunct antidote in the immediate treatment of cyanide poisoning.  
**Adult dose:** 0.3 mL ampule crushed every min and inhaled for 15–30 seconds until sodium nitrite infusion is ready. **Pediatric dose:** Same as for adult.

**Pharmacokinetics**  
**Absorption:** Rapidly absorbed from mucous membranes; onset is 10–30 seconds; peak effect is unknown; duration is 3–5 min.

**Contraindications and Precautions:** There are no contraindications to the use of amyl nitrite in the management of acute cyanide poisoning.
Adverse/Side Effects: CNS: Headache, dizziness, weakness, syncope • CV: Transient flushing, orthostatic hypotension, palpitations, cardiovascular collapse, tachycardia • Respiratory: Respiratory depression • GI: Nausea, vomiting.

Interactions: The hypotensive effects of amyl nitrite may be potentiated by antihypertensive agents, beta blockers, and certain antiemetics (phenothiazines).

Prehospital Considerations
• To prepare for administration, wrap the ampule in gauze or cloth and crush between your fingers.
• Syncope, due to a sudden drop in systolic BP, sometimes follows amyl nitrite inhalation, particularly in the elderly. Patient should be sitting while and immediately after drug is administered.
• Amyl nitrite is volatile and highly flammable. When mixed with air or oxygen, it forms a mixture that can explode if ignited.
• After administration of drug, note length of time required for pain to subside; monitor vital signs until they are stable. Rapid pulse, which usually lasts for a brief period, is an expected baroreceptor response to the fall in BP produced by the nitrite ion.
• Inform patient that drug has a strong, unpleasant odor (often compared to an athletic locker room).
• Amyl nitrite is a drug of abuse and should be kept in a secure place with your narcotics.

ANISTREPLASE (APSAC)

Class: Thrombolytic
Trade Name: Eminase

Therapeutic Action/Pharmacodynamics: Anisoylated plasminogen-streptokinase activator complex (APSAC) causes thrombolysis (dissolution of a clot) by converting plasminogen
(present in the blood) to plasmin. Plasmin then digests fibrin and fibrinogen, causing the blood clot to dissolve.

**Emergency Use:** To reduce infarct size in acute MI by thrombolysis. *Adult dose:* 30 units IV push over 2–5 min.

**Pharmacokinetics**

*Absorption:* Immediate onset; peak effect in 45 min; duration is 6 hr to 2 days; half-life 105–120 min.

*Metabolism:* Metabolized in plasma.

**Contraindications and Precautions:** APSAC is absolutely contraindicated in patients with active internal bleeding, suspected aortic dissection, traumatic CPR (rib fractures, pneumothorax), history of recent stroke (within 6 months), recent (within 2 months) intracranial or intraspinal surgery or trauma, intracranial tumors, uncontrolled hypertension, pregnancy, or severe allergic reactions to either anistreplase or streptokinase. Use with caution in patients with recent major surgery (within 10 days), cerebral vascular disease, pregnancy, recent GI or GU bleeding, recent trauma, hypertension, hemorrhagic ophthalmic conditions, and current use of oral anticoagulants, and in those over age 75.

**Adverse/Side Effects:** CV: Hemorrhage, reperfusion dysrhythmias, hypotension * Hypersensitivity: Anaphylactic and anaphylactoid reactions in less than 1% of patients.

**Interactions:** Use with caution in patients on anticoagulant therapy.

**Prehospital Considerations**

- The drug should be administered as soon as possible following the onset of clinical symptoms of acute MI.
- Dilute each dose with 5 mL sterile water for injection. Slowly add diluent, rolling vial to mix; do not shake. Do not further dilute reconstituted solution and discard if it is not used within 30 min.
- Inject over 2–5 min directly into vein or IV line through the most proximal port.
• During administration, only essential handling or moving of the patient should be done.
• Diluted solution may be clear to pale yellow. Do not administer if particulate matter is present.
• Spontaneous bleeding occurs twice as often with anistreplase as with heparin. Protect patient from invasive procedures: IM injections are contraindicated. Also prevent manipulation during thrombolytic therapy to prevent bruising.
• Report signs of bleeding: gum bleeding, epistaxis, hematoma, spontaneous ecchymosis, oozing at catheter site, increased pain from internal bleeding. The anistreplase infusion should be interrupted, then resumed when bleeding stops.
• APSAC may be ineffective if given within 1 year of prior streptokinase or APSAC therapy.
• Be prepared to resuscitate your patient if anaphylaxis or reperfusion dysrhythmias occur.

ASPIRIN (Acetylsalicylic Acid)

Classes: Analgesic; antipyretic; nonsteroidal anti-inflammatory drug; platelet inhibitor
Trade Names: Alka-Seltzer, A.S.A., Aspergum, Aspro (Aus), Astrin (Can), Bayer, Bayer, Bext (Aus), Children’s, Corhyphen (Can), Cosprin, Easprin, Ecotrin, Empirin, Entrophen (Can), Halfprin, Measurin, Novasen (Can), St Joseph Children’s, Solprin (Aus), Supasa (Aus), Triaphen-10, Vincent’s Powders (Aus), Winsprin Capules (Aus), ZORprin.

Therapeutic Action/Pharmacodynamics: Aspirin is an anti-inflammatory agent and an inhibitor of platelet function. The major actions of aspirin appear to be associated primarily with inhibiting the formation of prostaglandins involved in the production of inflammation, pain, and fever. As an anti-inflammatory agent, aspirin appears to be involved in enhancing and in reducing the spread of inflammation by inhibiting prostaglan-
din synthesis. These anti-inflammatory actions also contribute to analgesic effects. As an analgesic, it relieves mild to moderate pain by acting on the peripheral nervous system with limited action in the central nervous system (hypothalamus). In addition to inhibiting prostaglandin synthesis, aspirin lowers body temperature in fever by indirectly causing centrally mediated peripheral vasodilation and sweating. As an antiplatelet agent, aspirin (but not other salicylates) powerfully inhibits platelet aggregation by blocking the formation of thromboxane A2, which causes platelets to aggregate and arteries to constrict. This action results in an overall reduction in mortality associated with myocardial infarction. It also reduces the rate of nonfatal reinfarction and nonfatal stroke.

**Emergency Use:** To inhibit clot formation in the presence of chest pain suggestive of an acute myocardial infarction. To inhibit clot formation associated with thrombotic CVA (“brain attack”). *Adult dose:* 160–325 mg PO (chewable).

**Pharmacokinetics**

**Absorption:** 80–100% absorbed (depending on formulation), primarily in stomach and upper small intestine; onset is 5–30 min; peak levels in 15 min to 2 hr; duration is 1–4 hr; half-life is 15–20 min.

**Distribution:** Widely distributed in most body tissues; crosses placenta.

**Metabolism:** Aspirin is hydrolyzed to salicylate in GI mucosa, plasma, and erythrocytes; salicylate is metabolized in liver.

**Elimination:** 50% of dose is eliminated in the urine in 2–4 hr. Excreted in breast milk.

**Contraindications and Precautions:** Aspirin is contraindicated in patients with a history of hypersensitivity to salicylates including methyl salicylate (oil of wintergreen), active ulcer disease, and asthma. It should be used with caution in patients with allergies to other NSAIDS, and in those who have other
bleeding disorders. Because of the possible association of aspirin usage with Reye’s syndrome, do not give aspirin to children or teenagers with symptoms of varicella (chickenpox) or influenza-like illnesses before consulting a physician.

Adverse/Side Effects: CNS: Dizziness, confusion, drowsiness
• ENT: Tinnitus, hearing loss • GI: Nausea, vomiting, diarrhea, anorexia, heartburn, stomach pains, ulceration, occult bleeding, GI bleeding • Hematologic: Thrombocytopenia, hemolytic anemia • Hypersensitivity: Urticaria, bronchospasm, anaphylactic shock, laryngeal edema • Skin: Petechiae, easy bruising, rash • Other: Impaired renal function, prolonged bleeding time, prolonged pregnancy and labor with increased bleeding.

Interactions: Anticoagulants increase risk of bleeding. Oral hypoglycemic agents increase hypoglycemic activity with aspirin doses greater than 2 g/day.

Prehospital Considerations
• Baby aspirin is preferred in the emergency setting because it can be chewed and swallowed, and it is more palatable to the nauseated MI patient. Administer 2 tablets (82 mg each) as soon as possible when indicated.
• Gastric irritation may be minimized by administering with a full glass of water (240 mL), or with milk, food, or antacid. Enteric-coated tablets dissolve too quickly if administered with milk; also they should not be crushed or chewed.
• In adults, a sensation of fullness in the ears, tinnitus, and decreased or muffled hearing are the most frequent symptoms associated with chronic salicylate overdosage.
• Potential for toxicity is high in elderly chronic aspirin users because they have less serum protein to bind salicylate and also are less able to excrete it.
• Children tend to manifest salicylate toxicity by hyperventilation, agitation, mental confusion, or other behavioral changes, drowsiness, lethargy, sweating, and constipation.
• In children, and infants particularly, salicylate toxicity is enhanced by the dehydration that frequently accompanies fever or illness. Monitor these patients closely.
• GI disturbances may be reduced by use of enteric-coated tablets or extended release tablets.
• Buffered aspirin preparations in an effervescent vehicle, e.g., Alka-Seltzer, are more rapidly absorbed than plain aspirin and reportedly cause less GI irritation and bleeding. Alka-Seltzer, however, has a high sodium content (approximately 24 mEq of sodium per 32-mg tablet).
• Buffered aspirin or aspirin administered with an antacid may be better tolerated than conventional tablets.
• Discontinue use with onset of ringing or buzzing in the ears, impaired hearing, dizziness, or GI discomfort or bleeding, and report to physician. Hearing impairment resulting from salicylate overdosage can generally be reversed within 24 hr by reducing the dose.
• Avoid other medications containing aspirin unless directed by physician, because of danger of overdosing. (There are more than 500 OTC aspirin-containing compounds.)

ATENOLOL

Classes: Antidysrhythmic, antihypertensive
Trade Names: Tenormin, Apo-Atenolol (Can)

Therapeutic Action / Pharmacodynamics: Atenolol is a beta-blocking agent selective for beta1-adrenergic receptors located chiefly in cardiac muscle. With large doses, the selectivity for beta1-adrenergic receptors is lost and inhibition of beta2-adrenergic receptors may lead to increased airway resistance, especially in patients with asthma and COPD. Cardiac effects are primarily due to competitive inhibition of catecholamine binding at beta-adrenergic receptor sites. Atenolol reduces the rate
and force of cardiac contraction (negative inotropic action); cardiac output and blood pressure is reduced. Atenolol increases peripheral vascular resistance.

**Emergency Uses:** To treat acute coronary syndromes including non-Q-wave MI and unstable angina. Beta blockers also reduce the incidence of ventricular fibrillation. Adult dose: 5 mg slow IV (over 5 min); wait 10 min, then if the first dose is well tolerated, give a second 5 mg slow IV (over 5 min). Pediatric dose: 0.8–1.5 mg/kg/day PO (maximum 2 mg/kg/day).

**Pharmacokinetics**
Absorption: 50% of oral dose absorbed; peak effect in 2–4 hr PO, 5 min IV. Duration of effect is 24 hr.
Distribution: Does not readily cross blood brain barrier.
Metabolism: No hepatic metabolism.
Elimination: Half-life 6–7 hr. 40–50% excreted in urine, 50–60% in feces.

**Contraindications and Precautions:** Atenolol is contraindicated in sinus bradycardia, greater than first-degree heart block, CHF, cardiogenic shock. Use caution in asthma, COPD, and CHF controlled by digitalis and diuretics. Safe use during pregnancy (category C), in nursing women, and in children not established.

**Adverse/Side Effects:** CNS: Dizziness, vertigo, light-headedness, syncope, fatigue or weakness, lethargy, drowsiness, insomnia, depression • CV: Bradycardia, hypotension, CHF, cold extremities, leg pains, dysrhythmias • GI: Nausea, vomiting, diarrhea • Respiratory: Pulmonary edema, dyspnea, bronchospasm • Other: May mask symptoms of hypoglycemia.

**Interactions:** Atropine and other anticholinergics can increase atenolol absorption from the GI tract; NSAIDs can decrease hypotensive effects. May mask symptoms of hypoglycemia induced by insulin and sulfonylureas. May increase lidocaine levels and toxicity; pharmacologic effects of atenolol and verapamil are increased when used concomitantly.
Prehospital Considerations
- IV atenolol is given no faster than 1 mg/min (5 mg/5 min).
- IV atenolol may be diluted in up to 50 mL of D5W or NS.
- Store in tightly covered, light-resistant containers at 15–30°C (59–86°F) unless otherwise directed.
- ECG monitoring is essential because of the possibility of drug-induced arrhythmias.
- Instruct patient to report immediately any increased dyspnea or decreased exercise tolerance.
- Monitor pulse, blood pressure, ECG, and respirations throughout therapy.

ATRACURIUM
Class: Nondepolarizing neuromuscular blocker
Trade Name: Tracrium
Therapeutic Action/Pharmacodynamics: Atracurium is a synthetic skeletal muscle relaxant pharmacologically similar to tubocurarine that produces shorter duration of neuromuscular blockade, exhibits minimal direct effects on cardiovascular system, and has less histamine-releasing action. It has minimal cumulative tendency with subsequent doses if recovery from the drug begins before dose is repeated. It inhibits neuromuscular transmission by binding competitively with acetylcholine to muscle end-plate receptors. Atracurium lacks analgesic action and has no apparent effect on pain threshold, consciousness, or cerebration.
Emergency Uses: To produce skeletal muscle relaxation to facilitate endotracheal intubation and positive pressure ventilation. Adult dose: 0.4–0.5 mg/kg IV. Pediatric dose: less than 2 yr: 0.3–0.4 mg/kg IV; more than 2 yr: same as for adult.
Pharmacokinetics
Absorption: Onset is 2 min; peak effect is 3–5 min, duration is
35–70 min, half-life is 20 min.

**Distribution:** Well distributed to tissues and extracellular fluids; crosses placenta.

**Metabolism:** Rapid nonenzymatic degradation in bloodstream.

**Elimination:** 70–90% excreted in urine within 5–7 hr.

**Contraindications and Precautions:** Atracurium is contraindicated in myasthenia gravis. It should be used with caution when appreciable histamine release would be hazardous (as in asthma or anaphylactoid reactions, significant cardiovascular disease).

**Adverse/Side Effects:** CV: Bradycardia, tachycardia • Respiratory: Respiratory depression • Other: Increased salivation, anaphylaxis.

**Interactions:** Neuromuscular blockade may be enhanced in the presence of the following drugs: aminoglycosides, bacitracin, polymyxin B, chindamycin, lidoaine, parenteral magnesium, quinidine, quinine, trimethaphan, verapamil, diuretics, lithium, and succinylcholine. Narcotic analgesics may present the possibility of additive respiratory depression. Phenytoin may cause resistance to or reversal of neuromuscular blockade.

Atracurium is incompatible with alkaline solutions (e.g., barbiturates, sodium bicarbonate). Do not mix in same syringe or administer through same needle as used for alkaline solutions. Reportedly compatible with 5% dextrose and 0.9% NaCl.

**Prehospital Considerations**

- Equipment required for endotracheal intubation, administration of oxygen under positive pressure, artificial respiration, and assisted or controlled ventilation should be immediately available.

- Monitor BP, pulse, and respirations and evaluate your patient’s recovery from neuromuscular blocking (curare-like) effect as evidenced by ability to breathe naturally or to take deep breaths and cough, keep eyes open, lift head keeping
mouth closed, adequacy of hand-grip strength.
• Patient may find oral communication difficult until head and
  neck muscles recover from blockade effects.
• Recovery from neuromuscular blockade usually begins 35–45 min after drug administration and is almost complete in
  about 1 hr. Note that recovery time may be delayed in
  patients with cardiovascular disease, edematous states, and in
  the elderly.

ATROPINE

Class: Parasympatholytic
Trade Name: Atropine

Therapeutic Action/Pharmacodynamics: Atropine exerts its effects on the autonomic nervous system. It is a competitive antagonist that selectively blocks all muscarinic responses to acetylcholine (ACh). By blocking vagal (parasympathetic) impulses to the heart it increases SA node discharge, enhances conduction through the AV junction, and increases cardiac output. Its antisecretory action suppresses sweating, lacrimation, salivation, and secretions from the upper and lower respiratory tract. Atropine is a potent bronchodilator when bronchoconstriction has been induced by parasympathomimetics. Produces mydriasis (dilation of pupils) and cycloplegia (paralysis of accommodation) by blocking responses of iris sphincter muscle and ciliary muscle of lens to cholinergic stimulation.

Emergency Uses: To increase cardiac output in symptomatic bradycardia (e.g., altered mental status, hypotension, cardiac ectopy, chest pain, CHF). Adult dose: 0.5–1.0 mg IV, 2 mg ET. May repeat every 3–5 min up to 0.04 mg/kg. Pediatric dose: 0.02 mg/kg IV, 0.04 mg/kg ET. Minimum dose is 0.1 mg. May repeat in 5 min up to 1 mg. To restore cardiac function in
bradyasystolic cardiac arrest. **Adult dose:** 1 mg IV, 2 mg ET. May repeat every 3–5 min up to 0.04 mg/kg. **Pediatric dose:** Not used in pediatric asystole. As a parasympatholytic in organophosphate poisoning. **Adult dose:** 2–5 mg IV/IM every 10–15 min. **Pediatric dose:** 0.05 mg/kg IV/IM/IO every 10–15 min.

**Pharmacokinetics**

**Absorption:** Atropine is well absorbed from all administration sites; peak effect is 20–60 min IM, 2–4 min IV; duration is 4 hr; half-life is 2–3 hr.

**Distribution:** Distributed in most body tissues; crosses blood-brain barrier and placenta. **Metabolism:** Metabolized in liver. **Elimination:** 77–94% excreted in urine in 24 hr.

**Contraindications and Precautions:** No contraindications in the emergency setting. Use with caution in patients with signs and symptoms of acute myocardial ischemia or infarction. Because it raises intraocular pressure, use with caution in patients with glaucoma.

**Adverse/Side Effects:** CNS: Headache, ataxia, dizziness, excitement, irritability, convulsions, drowsiness, fatigue, weakness; mental depression, confusion, disorientation, hallucinations • CV: Hypertension or hypotension, ventricular tachycardia, palpitations, paradoxical bradycardia, AV dissociation, atrial or ventricular fibrillation • Eye: Mydriasis, blurred vision, photophobia, increased intraocular pressure, cycloplegia, eye dryness, local redness • GI: Dry mouth with thirst, dysphagia, loss of taste; nausea, vomiting, constipation, delayed gastric emptying • GU: Urinary hesitancy and retention, dysuria, impotence • Skin: Flushed, dry skin; anhidrosis, rash, urticaria, contact dermatitis, allergic conjunctivitis.

**Interactions:** Amantadine, antihistamines, tricyclic antidepressants, quinidine, disopyramide, procainamide can add to the anticholinergic effects of atropine. Levodopa effects are decreased. Methotrimeprazine may precipitate extrapyramidal
effects. Phenothiazines’ antipsychotic effects are decreased (decreased absorption).

**Prehospital Considerations**

- Smaller doses of atropine are indicated for the elderly.
- Monitor vital signs. Pulse is a sensitive indicator of patient’s response to atropine. Be alert to changes in quality, rate, and rhythm of pulse and respiration and to changes in blood pressure and temperature.
- Initial paradoxic bradycardia following IV atropine usually lasts only 1–2 min; it most likely occurs when IV is administered slowly (more than 1 min) or when small doses (less than 0.5 mg) are used.
- Atropine may actually worsen the bradycardia associated with Mobitz II and complete AV block. In these cases, use transcutaneous pacing.
- Always rule out hypoxia as the cause for bradycardia in infants and small children. Atropine is indicated only after oxygen and epinephrine fail.

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**BRETYLIUM**

**Class:** Antidysrhythmic

**Trade Names:** Bretylate (Aus), Bretylol

**Therapeutic Action/Pharmacodynamics:** The mechanism of action of bretylium is complex and not fully understood. It suppresses ventricular fibrillation by direct action on the myocardium and ventricular tachycardia by adrenergic blockade. Shortly after administration, norepinephrine is released from adrenergic postganglionic nerve terminals, resulting in a moderate increase in BP, heart rate, and ventricular irritability. Subsequently, drug-induced release and reuptake of norepinephrine are blocked, leading to a state resembling surgical sympathectomy. Bretylium suppresses ventricular tachydys-
rhythms with a reentry mechanism, elevates the fibrillation threshold, and decreases ectopic foci without changing PR, QT, and QRS intervals. Orthostatic hypotension occurs commonly as a result of peripheral adrenergic blockade; some degree of hypotension may occur even while your patient is supine. Tolerance to this effect develops after several days in most patients as adrenergic receptors become more responsive to circulating catecholamines. Because onset of desired action is delayed, bretylium is not a first-line antidysrhythmic agent.

**Emergency Uses:** To treat ventricular tachycardia and ventricular fibrillation refractory to lidocaine. **Adult dose:** 5 mg/kg IV; repeat at 10 mg/kg IV every 15–30 min up to 30 mg/kg. Following conversion, administer IV infusion at 1–2 mg/min. **Pediatric dose:** 5 mg/kg IV; repeat bolus of 10 mg/kg in 15–30 min.

**Pharmacodynamics**

**Absorption:** Onset and peak effect is minutes after IV; duration: 6–24 hr; half-life is 4–17 hr.

**Distribution:** Does not cross blood-brain barrier; not known if crosses placenta or distributed into breast milk.

**Metabolism:** Not metabolized.

**Elimination:** 70–80% excreted in urine in 24 hr.

**Contraindications and Precautions:** There are no contraindications when used in life-threatening refractory ventricular dysrhythmias. Safe use in pregnancy (Category C), in nursing mothers, and in children not established. Use with caution in patients with digitalis-induced dysrhythmias; fixed cardiac output, e.g., severe aortic stenosis or severe pulmonary hypertension (profound hypotension can result without compensatory increase in cardiac output); sinus bradycardia; angina pectoris; or impaired renal function; and in patients on digitalis maintenance.

**Adverse/Side Effects:** CV: Both supine and postural hypotension with dizziness, vertigo, light-headedness, faintness, syncope, transitory hypertension, bradycardia, increased frequency
of PVCs, exacerbation of digitalis-induced dysrhythmias • GI: Nausea, vomiting (particularly with rapid IV) • Other: Respiratory depression.

**Interactions:** Bretylium can interact with other antidysrhythmic drugs (procainamide, quinidine, disopyramide, propranolol, lidocaine) causing either antagonistic or additive effects. Antihypertensive agents will add to hypotensive effects. The initial release of norepinephrine may cause the worsening of digitalis-induced dysrhythmias.

**Prehospital Considerations**
- Anticipate vomiting. IV administration is associated with a high incidence of nausea and vomiting. These side effects can be minimized by slow administration of drug (10 min or more).
- Establish baseline readings and monitor BP and ECG when drug is administered. Observe for initial transient rise in BP, increased heart rate, PVCs and other dysrhythmias, or worsening of existing dysrhythmias, which may occur within a few min to 1 hour after drug administration.
- Initial effect of hypertension is usually followed within 1 hour by a fall in supine BP and by orthostatic hypotension.
- Bretylium has been removed from ACLS algorithms and guidelines because of a high incidence of side effects, the availability of safer agents, and the limited availability and supply of the drug.

**BUMETANIDE**

**Class:** Loop diuretic

**Trade Name:** Bumex, Burinex (Aus)

**Therapeutic Action/Pharmacodynamics:** Bumetanide is a sulfonamide derivative structurally related to furosemide and with similar pharmacologic effects. It features a more rapid rate of onset, a more potent diuretic effect (40 times greater),
and a shorter duration of action than that of furosemide. Bumetanide inhibits sodium and chloride reabsorption by direct action on proximal ascending limb of the loop of Henle. It also appears to inhibit phosphate and bicarbonate reabsorption. At usual diuretic doses, it produces only mild hypotensive effects. Causes both potassium and magnesium wastage.

**Emergency Use:** To promote diuresis in congestive heart failure and pulmonary edema. *Adult dose:* 0.5–1 mg IM/IV over 1–2 min. Repeat doses may be administered in 2–3 hr as needed.

**Pharmacokinetics**

*Absorption:* IV onset is rapid; peak effect in 15–30 min; duration is 3.5–4.0 hr; half-life is 60–90 min.

*Distribution:* Distributed into breast milk.

*Metabolism:* Partially metabolized in liver.

*Elimination:* 80% excreted in urine in 48 hr, 10–20% excreted in feces.

**Contraindications and Precautions:** Bumetanide is contraindicated in patients with known hypersensitivity to bumetanide or to other sulfonamides. Its use in pregnancy should be limited to life-threatening situations in which the benefits of using bumetanide outweigh the risks.

**Adverse/Side Effects:** CNS: Dizziness, headache, weakness, fatigue • CV: Hypotension, ECG changes, chest pain, hypovolemia • GI: Nausea, vomiting, abdominal or stomach pain, GI distress, diarrhea, dry mouth • Musculoskeletal: Muscle cramps, muscle pain, stiffness or tenderness; arthritic pain • Ototoxicity: Ear discomfort, ringing or buzzing in ears, impaired hearing • Other: Sweating, hyperventilation.

**Interactions:** Bumetanide increases the risk of hypokalemia-induced digoxin toxicity; NSAIDs may attenuate diuretic and hypotensive response; probenecid may antagonize diuretic activity; bumetanide may decrease renal elimination of lithium.
**Prehospital Considerations**

- Bumetamide may be used for patients allergic to furosemide who need rapid diuresis.
- Drug will discolor on exposure to light. Inspect parenteral bumetanide before administration. Discard if it contains particles or is discolored.
- Store in tight, light-resistant container at 15–30°C (59–86°F) unless otherwise directed.
- Monitor weight, BP, and pulse rate. Assess for hypovolemia by assessing BP and pulse rate while patient is lying, sitting, and standing.
- High doses or frequent administration, particularly in the elderly, can cause profound diuresis, hypovolemia, and resulting circulatory collapse with development of thrombi and emboli. Careful monitoring is essential.
- Patients with hepatic disease should be carefully observed. Alterations in fluid and electrolyte balance can precipitate encephalopathy (inappropriate behavior, altered mood, impaired judgment, confusion, drowsiness, coma).

**BUTORPHANOL**

**Class:** Synthetic narcotic (opiate) analgesic  
**Trade Names:** Stadol  
**Therapeutic Action/Pharmacodynamics:** Butorphanol is a synthetic, centrally acting analgesic with mixed narcotic agonist and antagonist actions. It acts as an agonist on one type of opioid receptor and as a competitive antagonist at others. The site of analgesic action believed to be subcortical, possibly in the limbic system. On a weight basis, analgesic potency appears to be about 5 times that of morphine, 40 times that of meperidine, and 15–30 times that of pentazocine. Its narcotic antagonist potential is approximately 30 times that of penta-
zocine and 1/40 that of naloxone. Two mg of butorphanol produce about the same degree of respiratory depression as 10 mg of morphine. Respiratory depression does not increase appreciably with higher doses, as it does with morphine, but duration of action increases. Like pentazocine, analgesic doses may increase pulmonary arterial pressure and cardiac workload. Butorphanol appears to have low potential for dependence. It tends to inhibit release of antidiuretic hormone (ADH) from posterior pituitary. It is a Schedule IV narcotic.

**Emergency Use:** To relieve moderate to severe pain. **Adult dose:** 1 mg IV or 3–4 mg IM every 3–4 hr as needed.

**Pharmacokinetics**

**Absorption:** Onset is 10–15 min IM, 2–3 min IV; peak effect is 0.5–1.0 hr IM, 4–5 min IV; duration is 3–4 hr IM/IV; half-life is 3–4 hr.

**Distribution:** Crosses placenta; distributed into breast milk.

**Metabolism:** Metabolized in liver in inactive metabolites.

**Elimination:** Excreted primarily in urine.

**Contraindications and Precautions:** Butorphanol is contraindicated in patients with a known hypersensitivity to the drug. Since it may act as a narcotic antagonist and produce withdrawal symptoms, use it with caution in narcotic-dependent patients. Safe use during pregnancy prior to labor (category C), in nursing mothers, and in children under 18 yr not established. Do not use butorphanol in the presence of a head injury or undiagnosed abdominal pain as it will mask the symptoms before a diagnosis can be confirmed. This drug can also cause an increase in cerebrospinal pressure.

**Adverse/Side Effects:** CNS: Drowsiness, sedation, headache, vertigo, dizziness, floating feeling, weakness, lethargy, confusion, light-headedness, insomnia, nervousness • Respiratory: Respiratory depression • CV: Palpitation, bradycardia • GI: Nausea • Skin: Clammy skin, tingling sensation, flushing and
warmth, cyanosis of extremities, diaphoresis, sensitivity to cold, urticaria, pruritus.

**Interactions:** Alcohol and other CNS depressants augment CNS and respiratory depression.

**Prehospital Considerations**
- Since the effects are unpredictable in older patients, consider reducing the dose and administering smaller repeated doses rather than one large bolus.
- Store at 15–30°C (59–86°F) unless otherwise directed. Protect from light.
- Monitor for respiratory depression. Do not administer drug if respiratory rate is less than 12 breaths/min. If respirations decrease, you may reverse the effects of butorphanol with naloxone.
- Monitor vital signs. Report marked changes in BP or bradycardia.
- Butorphanol has habit-forming potential.
- Because butorphanol has agonist as well as antagonist actions, it can induce acute withdrawal symptoms in opiate-dependent patients.
- Because of its potential for abuse, butorphanol should be secured and inventoried daily.

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**CALCIUM CHLORIDE**

**Class:** Electrolyte  
**Trade Name:** Calcium chloride, Calciject (Aus)

**Therapeutic Action/Pharmacodynamics:** Calcium chloride provides elemental calcium in the form of the cation Ca++. Calcium is necessary for many physiologic activities. It is an essential element for regulating the excitation threshold of nerves and muscles, for blood clotting mechanisms, maintenance of renal function, for body skeleton and teeth. Calcium
causes a significant increase in myocardial contractility and in ventricular automaticity. It also plays a role in regulating the storage and release of neurotransmitters and hormones; regulating amino acid uptake and absorption of vitamin B12; gastrin secretion, and in maintaining structural and functional integrity of cell membranes and capillaries. Its excess chloride ions promote acidosis and temporary (1−2 days) diuresis secondary to the excretion of sodium. It is used as an antidote for some electrolyte imbalances, for magnesium sulfate overdose, and to minimize the side effects from calcium channel blocker usage. The actions of calcium chloride are similar to those of calcium gluconate but, since it ionizes more readily, it is more potent than calcium gluconate and more irritating to tissues. 

**Emergency Uses:** To treat hyperkalemia (elevated potassium), hypocalcemia (decreased calcium), hypermagnesemia (elevated magnesium) and calcium channel blocker toxicity. **Adult dose:** 2−4 mg/kg (10% solution) IV every 10 min as needed.  
**Pediatric dose:** 20 mg/kg (10% solution) IV, repeated once in 10 min as needed.

**Pharmacokinetics**

*Absorption:* Onset and peak effects are immediate; duration is unknown.

*Distribution:* Crosses placenta.

*Elimination:* Primarily excreted in feces; small amounts excreted in urine, pancreatic juice, saliva, and breast milk.

**Contraindications and Precautions:** Calcium chloride is contraindicated in ventricular fibrillation, hypercalcemia, and possible digitalis toxicity. It should be used with caution in patients taking digoxin as it may precipitate toxicity. Safe use during pregnancy prior to labor (category C), in nursing mothers, and in children not established.

**Adverse/Side Effects:** CNS: Tingling sensation, fainting • Skin: With rapid IV, sensations of heat waves (peripheral
vasodilation), pain and burning at IV site, necrosis and sloughing (with extravasation) • CV: Hypotension, bradycardia, cardiac dysrhythmias, cardiac arrest, severe venous thrombosis.

**Interactions:** Calcium chloride will interact with sodium bicarbonate and form a precipitate. It may enhance inotropic and toxic effects of digoxin and antagonize the effects of verapamil and possibly other calcium channel blockers.

**Prehospital Considerations**
- Extravasation must be avoided during IV injection, since cellulitis, necrosis, and sloughing can result. Give at 0.5–1.0 mL/min or more slowly if irritation develops. Use a small-bore needle and inject into a large vein to minimize venous irritation and undesirable reactions. If given IV to children, scalp veins should be avoided.
- IV injection may be accompanied by cutaneous burning sensation and peripheral vasodilation, with moderate fall in BP. Monitor ECG, BP, and flow rate and observe patient closely during administration.
- Always flush your IV line prior to and immediately following administration of drugs such as sodium bicarbonate or catecholamines to avoid forming a precipitate.

**CALCIUM GLUCONATE**

**Class:** Electrolyte  
**Trade Name:** Kalcinate  
**Therapeutic Actions/Pharmacodynamics:** Calcium gluconate provides elemental calcium in the form of the cation $\text{Ca}^{++}$. Calcium is necessary for many physiologic activities. It is an essential element for regulating the excitation threshold of nerves and muscles, for blood clotting mechanisms, maintenance of renal function, and for the development of skeletal
bones and teeth. Calcium causes a significant increase in myocardial contractility and in ventricular automaticity. It also plays a role in regulating the storage and release of neurotransmitters and hormones; regulating amino acid uptake and absorption of vitamin B12; controlling gastrin secretion; and in maintaining structural and functional integrity of cell membranes and capillaries. Calcium gluconate acts like digitalis on the heart, increasing cardiac muscle tone and force of systolic contractions (positive inotropic effect) making it especially useful for patients with sympathetic blockade.

**Emergency Uses:** To treat cardiac toxicity of hyperkalemia, as an antidote for magnesium sulfate, and to treat calcium channel blocker overdose. *Adult dose:* 5–8 mL of a 10% solution. Repeat as necessary in 10 min intervals.

**Pharmacokinetics**
*Absorption:* Onset and peak effects are immediate; duration is unknown.
*Distribution:* Crosses placenta.
*Elimination:* Primarily excreted in feces; small amounts excreted in urine, pancreatic juice, saliva, and breast milk.

**Contraindications and Precautions:** Calcium gluconate is contraindicated in ventricular fibrillation. Use with caution in digitalized patients, renal or cardiac insufficiency, and immobilized patients.

**Adverse/Side Effects:** CNS: Tingling sensations • CV: Hypotension, bradycardia and other dysrhythmias, syncope, cardiac arrest • Local reactions: Tissue irritation, burning, cellulitis, soft tissue calcification, necrosis and sloughing (following IV extravasation).

**Interactions:** Calcium gluconate will interact with sodium bicarbonate and form a precipitate. It may enhance inotropic and toxic effects of digoxin and antagonize the effects of verapamil and possibly other calcium channel blockers.
Prehospital Considerations

- IV calcium should be administered slowly through a small-bore needle into a large vein to avoid possibility of extravasation and resultant necrosis. If calcium is administered to children, scalp veins should be avoided.
- High concentrations of calcium suddenly reaching the heart can cause fatal cardiac arrest.
- Direct IV injection may be accompanied by cutaneous burning sensations and peripheral vasodilation, with moderate fall in BP. Injection should be stopped if patient complains of any discomfort.
- During IV administration, ECG is monitored to detect evidence of hypercalcemia: decreased QT interval associated with inverted T wave.
- Observe IV site closely. Extravasation may result in tissue irritation and necrosis.

CHLORDIAZEPoxide

Classes: Sedative-hypnotic; benzodiazepine

Trade Names: APO-chlordiazepoxide (Can), Librium, Novo-oxide (Can), Solium (Can).

Therapeutic Action/Pharmacodynamics: Chlordiazepoxide is a benzodiazepine derivative that acts on the limbic, thalamic, and hypothalamic areas of the CNS. It produces mild sedative, anticonvulsant, and skeletal muscle relaxant effects and has long-acting hypnotic properties.

Emergency Uses: To manage severe anxiety and tension; to manage acute alcohol withdrawal symptoms (delirium tremens). Adult dose: 50–100 mg IV/IM.

Pharmacokinetics

Absorption: Slow erratic absorption if given IM; peak effect is 15-30 min IM, 3-30 min IV; onset is 1–5 min IV; duration is 15–60 min IV.
**Distribution:** Widely distributed throughout body; crosses placenta.

**Metabolism:** Metabolized in liver to long-acting active metabolite.

**Elimination:** Slowly excreted in urine (may last several days); excreted in breast milk.

**Contraindications and Precautions:** Contraindicated in hypersensitivity to chlordiazepoxide and other benzodiazepines, pregnancy (category D), nursing mothers, lactation, oral use in children under 6 yr, parenteral use in children under 12 yr, acute alcohol intoxication. Use with caution in patients with primary depressive disorder or psychoses, and acute alcohol intoxication.

**Adverse/Side Effects:** CNS: Drowsiness, dizziness, lethargy, changes in EEG pattern, vivid dreams, nightmares, headache, vertigo, syncope, tinnitus, confusion, hallucinations, paradoxic rage, depression, delirium, ataxia • CV: Orthostatic hypotension, tachycardia, changes in ECG patterns seen with rapid IV administration • GI: Nausea, dry mouth, vomiting, constipation, increased appetite • GU: Urinary frequency • Other: Edema, pain in injection site, photosensitivity, skin rash, jaundice, hiccups, respiratory depression.

**Interactions:** It can potentiate CNS depression in patients taking depressants, anticonvulsants, and alcohol. It may increase levels of phenytoin and decrease the antiparkinson effects.

**Prehospital Considerations**

- Chlordiazepoxide is a Schedule IV controlled substance.
- Prepare parenteral solution immediately before use; discard unused portion. Drug is unstable in light and when in solution.
- For IV injection, 5 mL of sterile water for injection or NaCl 0.9% is added to each 100 mg ampule of dry powder and agitated gently until dissolved. Do not use IM diluent for the IV solution because it may contain air bubbles.
- Do not mix any other drug with chlordiazepoxide solution.
- Store in tight, light-resistant containers at 15–30°C (59–86°F) unless otherwise specified by manufacturer. The special diluent supplied by manufacturer for IM preparation should be kept refrigerated, preferably at 2–8°C (36–46°F) until ready for use.
- Orthostatic hypotension and tachycardia occur more frequently with parenteral administration. Patient should stay recumbent 2–3 hr after IM or IV injection; observe closely and monitor vital signs.

**CHLORPROMAZINE**

**Classes:** Tranquilizer; antipsychotic; phenothiazine

**Trade Names:** Chlorpromanyl (Can), Largactil (Aus), Novochlorpromazine (Can), Ormazine, Promapar, Promaz, Sonazine, Thorazine, Thor-Prom

**Therapeutic Action/Pharmacodynamics:** Chlorpromazine is a phenothiazine derivative used to manage severe psychotic episodes. Phenothiazines are believed to block the postsynaptic dopamine receptors in the brain associated with mood and behavior. Its actions on the hypothalamus and reticular formation produce strong sedation, hypotension, and depressed temperature regulation. Its inhibitory effect on dopamine reuptake may cause moderate extrapyramidal symptoms. Antipsychotic drugs are sometimes called neuroleptics (or tranquilizers) because they tend to reduce initiative and interest in environment, decrease displays of emotions or affect, suppress spontaneous movements and complex behavior, and decrease psychotic symptoms. They are also used to manage mild alcohol withdrawal, intractable hiccups, and nausea and vomiting.
Emergency Uses: To manage acute psychotic episodes, intractable hiccups, or nausea and vomiting. Adult dose: 25–50 mg IM. Pediatric dose: 0.5 mg/kg IM; 1.0 mg/kg PR.

Pharmacokinetics
Absorption: Rapid absorption after IM; onset is 30–60 min; peak effect is 15–20 min; duration is 4–6 hr.
Distribution: Widely distributed; accumulates in brain; crosses placenta.
Metabolism: Metabolized in liver.
Elimination: Excreted in urine as metabolites; excreted in breast milk.

Contraindications and Precautions: Chlorpromazine is contraindicated in comatose patients and those who have taken large amounts of sedatives. It is also contraindicated in patients with a hypersensitivity to phenothiazine derivatives; withdrawal states from alcohol; brain damage, bone marrow depression, Reye’s syndrome; and in children younger than 6 mo. Safe use during pregnancy (category C) and in nursing mothers not established. It may produce seizures in patients who have taken hallucinogens. It may impair mental and physical abilities and, on occasion, may produce orthostatic hypotension. It has caused extrapyramidal symptoms, especially in children. Use with caution in patients in agitated states accompanied by depression, seizure disorders, or respiratory impairment due to infection or COPD; patients with glaucoma, diabetes, hypertensive disease, peptic ulcer, prostatic hypertrophy, previously detected breast cancer, and those with thyroid, cardiovascular, and hepatic disorders; patients exposed to extreme heat or organophosphate insecticides.

Adverse/Side Effects: Side effects of chlorpromazine are usually dose related. CNS: Sedation, drowsiness, dizziness, restlessness, dyskinesias, tumor, syncope, headache, weakness, insomnia, reduced REM sleep, bizarre dreams, cerebral edema, convulsive seizures, hypothermia, inability to sweat, depressed
cough reflex, extrapyramidal symptoms, EEG changes • CV: Orthostatic hypotension, palpitation, tachycardia, ECG changes (usually reversible) including prolonged QT and PR intervals, blunting of T waves, ST depression • Eye: Blurred vision, mydriasis, photophobia • GI: Dry mouth, constipation, ileus, cholestatic jaundice, aggravation of peptic ulcer, dyspepsia, increased appetite • GU: Anovulation, infertility, pseudopregnancy, menstrual irregularity, priapism, inhibition of ejaculation, reduced libido, urinary retention and frequency • Respiratory: Laryngospasm • Skin/hypersensitivity: Urticaria, reduced perspiration, contact dermatitis, exfoliative dermatitis, photosensitivity, eczema, anaphylactoid reactions, hypersensitivity vasculitis; hirsutism (long-term therapy) • Other: Weight gain, hypoglycemia, hyperglycemia, glycosuria (high doses), enlargement of parotid glands, idiopathic edema, muscle necrosis (following IM), sudden unexplained death.

Interactions: Alcohol, and other CNS depressants can potentiate CNS depression. Phenobarbital increases the metabolism of chlorpromazine. The antihistamine phenylpropanolamine poses possibility of sudden death; tricyclic antidepressants intensify hypotensive and anticholinergic effects; anticonvulsants decrease seizure threshold (may need to increase anticonvulsant dose to compensate).

Prehospital Considerations
• Avoid parenteral drug contact with skin, eyes, and clothing because of its potential for causing contact dermatitis.
• Inject IM preparations slowly and deep into upper outer quadrant of buttock; massage site well. Avoid SC injection; it may cause tissue irritation and nodule formation. If irritation is a problem, consult physician about diluting medication with normal saline or 2% procaine. Rotate injection sites.
• The patient should remain recumbent for at least 1/2 hr after parenteral administration. Observe closely. Hypotensive reactions may require head-low position and pressor drugs, e.g.,
phenylephrine (Neo-Synephrine), norepinephrine (Levophed). Epinephrine and other pressor agents are contraindicated since they may cause sudden paradoxical drop in BP.

- Lemon yellow color of parenteral preparation does not alter potency; if otherwise colored or markedly discolored, solution should be discarded.
- Before initiating treatment, establish baseline BP (in standing and recumbent positions), pulse, and respiratory capacity values.
- Hypotensive reactions, dizziness, and sedation are common during early therapy, particularly in patients on high doses and in the elderly receiving parenteral doses. Patients usually develop tolerance to these side effects; however, lower doses or longer intervals between doses may be required.
- Smoking increases metabolism of phenothiazines, resulting in shortened half-life and more rapid clearance of drug. Higher dosage in smokers may be required. Advise patient to stop or at least reduce smoking, if possible.
- Extrapyramidal (EPS) or dystonic reactions are common, especially in children. These should be treated with parenteral diphenhydramine (Benadryl) or benztropine (Cogentin).

**DEXAMETHASONE**

**Class:** Steroid

**Trade Names:** Dalalone, Decadron, Decaject, Dexacen, Dexone, Deksone, Hexadrol, Solurex

**Therapeutic Actions/Pharmacodynamics:** Dexamethasone is a long-acting synthetic adrenocorticoid with intense anti-inflammatory (glucocorticoid) activity and minimal mineralocorticoid activity. As an anti-inflammatory agent it prevents accumulation of inflammatory cells at sites of infection; inhibits phagocytosis, lysosomal enzyme release, and synthesis of selected chemical mediators of inflammation; reduces capil-
lary dilation and permeability. Dexamethasone is used to manage the inflammatory response seen in allergic reactions. Once thought to significantly decrease cerebral edema, its use in managing brain and spinal cord injury remains controversial. Because a large single dose of steroids has little harmful effect, it is still used frequently in patients with cerebral edema both in the emergency department and in the field.

**Emergency Uses:** To reduce the inflammatory process in allergic reactions such as anaphylaxis, asthma and COPD; to reduce cerebral edema. *Adult dose:* 4–24 mg IV/IM. *Pediatric dose:* 0.5–1.0 mg/kg.

**Pharmacokinetics**

*Absorption:* Onset and peak effect are less than 1 hr; duration is variable for IV, 6 days IM; half-life is 3.0–4.5 hr. HPA suppression 36–54 hr. (Note: This is more important than the half-life.)

*Distribution:* Crosses placenta; distributed into breast milk.

*Elimination:* Hypothalamus-pituitary axis suppression: 36–54 hr.

**Contraindications and Precautions:** Dexamethasone has no absolute contraindications in the emergency setting. Relative contraindications include patients with systemic fungal infection, acute infections, active or resting tuberculosis, vaccinia, varicella, administration of live virus vaccines (to patient, family members). Use with caution in patients with stromal herpes simplex, keratitis, GI ulceration, renal disease, diabetes mellitus, hypothyroidism, myasthenia gravis, CHF, cirrhosis, psychic disorders, seizures.

**Adverse/Side Effects:** CNS: Euphoria, insomnia, convulsions, increased ICP, vertigo, headache, psychic disturbances • CV: CHF, hypertension, edema • Endocrine: Menstrual irregularities, hyperglycemia; cushingoid state; growth suppression in children; hirsutism • Eye: Posterior subcapsular cataract, increased IOP, glaucoma, exophthalmos • GI: Peptic ulcer with possible
perforation, abdominal distension, nausea, increased appetite, heartburn, dyspepsia, pancreatitis, bowel perforation, oral candidiasis • Musculoskeletal: Muscle weakness, loss of muscle mass, vertebral compression fracture, pathologic fracture of long bones, tendon rupture • Skin: Acne, impaired wound healing, petechiae, ecchymoses, diaphoresis, allergic dermatitis, hypo- or hyperpigmentation, SC and cutaneous atrophy, burning and tingling in perineal area (following IV injection).

**Interactions:** Since barbiturates, phenytoin, and rifampin increase steroid metabolism, the dosage of dexamethasone may need to be increased.

**Prehospital Considerations**
- Administer IM injection deep into a large muscle mass (e.g., gluteus maximus). Avoid SC injection; atrophy and sterile abscesses may occur.
- The repository form, dexamethasone acetate (for IM or local injection only), is a white suspension that settles on standing; mild shaking will resuspend the drug.
- Dexamethasone may be given undiluted by direct IV over 30 seconds or less. Drug may be added to an infusion of D$_5$W or NS and administered over a prescribed period.
- Cushing’s syndrome and other systemic effects can occur. Monitor and report signs and symptoms.

**DEXTROSE 50%**

**Class:** Carbohydrate

**Trade Names:** D$_{50}$W, 50% Dextrose

**Therapeutic Actions/Pharmacodynamics:** Dextrose is the principal form of glucose (sugar) used by the body to create energy. Since serious brain injury can occur in prolonged hypoglycemia, the rapid administration of glucose is essential. Dextrose 50% IV is the treatment of choice for hypoglycemic
patients with an altered mental status or no gag reflex.

**Emergency Use:** To increase blood sugar levels in documented hypoglycemia.

*Adult dose:* 25 g of 50% solution IV. *Pediatric dose:* 2 mL/kg of 25% solution IV.

**Pharmacokinetics**

**Absorption:** Immediate blood levels; onset <1 min; peak effect and duration dependent upon degree of hypoglycemia.

**Distribution:** Widely distributed to all body tissues.

**Metabolism:** Dextrose (glucose) is metabolized to carbon dioxide and water with the release of energy.

**Contraindications and Precautions:** There are no major contraindications to the IV administration of dextrose 50% to a patient with documented or suspected hypoglycemia. Use with caution in patients with increasing intracranial pressure as the added glucose may worsen the cerebral edema.

**Adverse/Side Effects:** Patients may complain of warmth, pain, or burning at the injection site. Dextrose 50% can cause tissue necrosis, phlebitis, sclerosis, or thrombosis at the injection site. Dextrose can cause severe neurologic symptoms (Wernicke’s encephalopathy, Korsakoff’s psychosis) if patient is thiamine deficient. Use with 100 mg thiamine in patients suspected of having thiamine deficiency.

**Interactions:** None in the emergency setting.

**Prehospital Considerations**

- Always ensure a patent IV line before administering dextrose 50% as severe tissue necrosis may occur with extravasation of this solution.
- Report and record blood glucose levels before and after administering this solution.
- Avoid extravasation.
DIAZEPAM

Classes: Sedative-hypnotic; anticonvulsant; benzodiazepine; antianxiety

Trade Name: Apodiazepam (Can), Atenex (Aus), Diazemuls (Can, Aus), Ducene (Aus), Novo-Dipam (Can), Valium, Zetran

Therapeutic Actions/Pharmacodynamics: Diazepam is a benzodiazepine whose exact mechanism is unknown, but many believe it appears to act at both limbic and subcortical levels of the CNS. It is principally used for its anticonvulsant properties; it suppresses the spread of seizure activity through motor cortex of the brain. It does not appear to abolish the abnormal electrical discharge focus. It is also used as a sedative in the management of stress and anxiety and to treat the withdrawal symptoms of alcohol. Diazepam is an effective skeletal muscle relaxant, making it an effective adjunct in managing orthopedic injuries. It is also useful as a premedication for minor surgeries and cardioversion because it induces amnesia, which diminishes the patient’s recall of the procedure.

Emergency Uses: To eradicate seizure activity, especially status epilepticus. Adult dose: 5–10 mg IV/IM. Pediatric dose: 0.5–2 mg IV/IM.
To manage acute anxiety. Adult dose: 2–5 mg IM. Pediatric dose: 0.5–2 mg IM.
As premedication for cardioversion. Adult dose: 5–15 mg IV. Pediatric dose: 0.2–0.5 mg/kg IV.

Pharmacokinetics

Absorption: Erratic IM absorption; onset is 1–5 min IV, 15–30 min IM; peak effect in 15 min IV, 30–45 min IM; duration: 15–60 min; half-life is 20–50 hr.
Distribution: Crosses blood-brain barrier and placenta; distributed into breast milk. Metabolism: Metabolized in liver to active metabolites.
Elimination: Excreted primarily in urine.
Contraindications and Precautions: Diazepam is contraindicated in patients with a hypersensitivity to the drug. IV diazepam is contraindicated in shock, coma, acute alcohol intoxication, depressed vital signs, obstetrical patients, infants less than 30 days old. Use with caution in patients with psychoses, mental depression; myasthenia gravis; impaired hepatic or renal function; and in individuals who are known to abuse drugs or be addiction-prone. Use IV diazepam with extreme caution in the elderly, the very ill, and patients with COPD.

Adverse/Side Effects: CNS: Drowsiness, fatigue, ataxia, confusion, paradoxic rage, dizziness, vertigo, amnesia, vivid dreams, headache, slurred speech, tremor; EEG changes, tardive dyskinesia • CV: Hypotension, tachycardia, edema, cardiovascular collapse • Eye: Blurred vision, diplopia, nystagmus • GI: Nausea, constipation • GU: Incontinence, urinary retention, gynecomastia (prolonged use), menstrual irregularities • Other: Hiccups, coughing, throat and chest pain, laryngospasm, ovulation failure, pain, venous thrombosis, phlebitis at injection site, hepatic dysfunction.

Interactions: Alcohol, CNS depressants, and anticonvulsants can potentiate CNS depression. Cimetidine increases diazepam plasma levels and toxicity. Diazepam may decrease the antiparkinson effects of levodopa and increase phenytoin levels. Smoking decreases its sedative and antianxiety effects.

Prehospital Considerations
- IM administration should be made deep into large muscle mass. Inject slowly. Rotate injection sites.
- To prevent swelling, irritation, venous thrombosis, and phlebitis, give direct IV by injecting drug slowly, taking at least 1 min for each 5 mg (1 ml) given to adults and taking at least 3 min to inject 0.25 mg/kg body weight of children.
- If injection cannot be made directly into vein, manufacturer suggests making injection slowly through infusion tubing as close as possible to vein insertion.
• Avoid small veins and extreme care should be taken to avoid intra-arterial administration or extravasation.
• Because diazepam is a relatively short-acting drug, seizure activity may recur and additional doses may be required.
• Preserve in tight, light-resistant containers at 15–30°C (59–86°F), unless otherwise specified by manufacturer.
• When diazepam is given parenterally, hypotension, muscular weakness, tachycardia, and respiratory depression may occur. Observe patient closely and monitor vital signs.
• Smoking increases metabolism of diazepam; therefore clinical effectiveness is lowered. Heavy smokers may need a higher dose than the nonsmoker.
• Flumazenil (Ronazicon) is an effective benzodiazepine antagonist and should be available in case respiratory depression or other complications ensue.

DIAZOXIDE

Class: Antihypertensive
Trade Name: Hyperstat

Therapeutic Actions/Pharmacodynamics: Rapid-acting thiazide (benzothiadiazine) nondiuretic hypotensive and hyperglycemic agent. In contrast to thiazide diuretics, causes sodium and water retention and decreases urinary output, probably because it increases proximal tubular reabsorption of sodium and decreases glomerular filtration rate. Reduces peripheral vascular resistance and BP by direct vasodilatory effect on peripheral arteriolar smooth muscles, perhaps by direct competition for calcium receptor sites. Its hypotensive effect may be accompanied by marked reflex increase in heart rate, cardiac output, and stroke volume; thus cerebral and coronary blood flow are usually maintained.

Emergency Use: To rapidly decrease BP in hypertensive crisis.
**Adult dose:** 1–3 mg/kg IV up to 150 mg given over 30 sec repeated at 5–15 min intervals as needed. **Pediatric dose:** Same as adult.

**Pharmacokinetics**

**Absorption:** Onset in 30–60 seconds IV; peak effect in 5 min; duration is 2–12 or more hr; half-life is 21–45 hr.

**Distribution:** Crosses blood-brain barrier and placenta.

**Metabolism:** Partially metabolized in the liver.

**Elimination:** Excreted in urine.

**Contraindications and Precautions:** Diazoxide is contraindicated in patients with a hypersensitivity to diazoxide or to other thiazides, cerebral bleeding, eclampsia, and significant coronary artery disease. Safe use during pregnancy (category C) and in nursing mothers not established. Use with caution in patients with diabetes mellitus, impaired cerebral or cardiac circulation, or impaired renal function; in patients taking corticosteroids or estrogen-progestogen combinations; and in those with a history of gout or uremia.

**Adverse/Side Effects:** CNS: Tinnitus, momentary hearing loss, headache, weakness, malaise, dizziness, sleepiness, insomnia, euphoria, anxiety, extrapyramidal signs • CV: Palpitations, atrial and ventricular dysrhythmias, flushing, shock; orthostatic hypotension, CHF, transient hypertension • Eye: Blurred vision, transient cataracts, subconjunctival hemorrhage, diplopia, lacrimation, papilledema • GI: Nausea, vomiting, abdominal discomfort, diarrhea, constipation, ileus, anorexia, transient loss of taste • Hypersensitivity: Rash, fever • Renal: Decreased urinary output, nephrotic syndrome (reversible), hematuria, increased nocturia, proteinuria, azotemia • Skin: Pruritus, flushing, monilial dermatitis, herpes, hirsutism; loss of scalp hair, sweating, sensation of warmth, burning, or itching. Other: hyperglycemia, edema, sodium and water retention.

**Interactions:** The effects of diazoxide can be potentiated when
administered with other antihypertensive agents. It can also increase phenytoin metabolism, decreasing the blood levels of phenytoin and precipitating seizures.

**Prehospital Considerations**
- Hypotension may occur and, if severe, should be treated with sympathomimetics. Because of the rapid onset of diazoxide, frequent monitoring of vital signs is essential to detect a drop in peripheral perfusion. Tachycardia has occurred immediately following IV administration; palpitation and bradycardia have also been reported.
- Since diazoxide causes sodium and water retention, a diuretic is generally prescribed to avoid CHF and drug resistance and to maximize hypotensive effect.
- When a diuretic, e.g., furosemide (Lasix), is prescribed, it is generally given 30–60 min prior to IV diazoxide. Patient should remain recumbent 8–10 hr because of possible additive hypotensive effect.
- Check the IV injection site frequently. The solution is strongly alkaline. Extravasation of medication into SC or IV tissues can cause severe inflammatory reaction. Diazoxide is administered only by peripheral vein.
- If BP continues to fall 30 min or more after IV drug administration, suspect cause other than drug effect. Notify physician immediately.

**DIGOXIN**

**Class:** Cardiac glycoside  
**Trade Names:** Digoxin, Lanoxin, Novodigoxin (Can)  
**Therapeutic Actions/Pharmacodynamics:** Digoxin is a rapid-acting cardiac glycoside used in the treatment of congestive heart failure and rapid atrial dysrhythmias. It acts by increasing
the force and velocity of myocardial systolic contraction through its effects on the sodium-potassium ATPase system. By significantly increasing stroke volume it increases cardiac output (positive inotropic effect). It also decreases conduction velocity through the atrioventricular node. Action is more prompt and less prolonged than that of digitalis and digitoxin, thus decreasing the heart rate (negative chronotropic and dromotropic effects). It is less likely than digitoxin or digitalis to give rise to cumulative effects because it is more readily absorbed and exchanged in the body and is rather rapidly excreted in urine.

**Emergency Uses:** To increase cardiac output in congestive heart failure and to stabilize supraventricular tachydysrhythmias, especially atrial flutter and atrial fibrillation. *Adult dose:* 0.25–0.5 mg slow IV. *Pediatric dose:* 10–50 µg/kg (age dependent) IV.

**Pharmacokinetics**

*Absorption:* Onset is 5–30 min; peak effect is 1–5 hr; duration is 3–4 days in fully digitalized patient; half-life is 34–44 hr.  
*Distribution:* Widely distributed; tissue levels significantly higher than plasma levels; crosses placenta.  
*Metabolism:* Approximately 14% in liver.  
*Elimination:* 80–90% excreted by kidneys; may appear in breast milk.

**Contraindications and Precautions:** Digoxin is contraindicated in digitalis hypersensitivity, ventricular fibrillation, ventricular tachycardia unless due to CHF. Full digitalizing dose not given if patient has received digoxin during previous week or if slowly excreted cardiotonic glycoside has been given during previous 2 wk. Use with extreme caution patients with acute MI as they are prone to digoxin toxicity. Digoxin toxicity is potentiated in patients with hypokalemia, hypocalcemia, hypomagnesemia, advanced heart disease, incomplete AV block, cor pulmonale, hyperthyroidism, lung disease, pregnancy (category
A), nursing women, premature and immature infants, children,
and in elderly or debilitated patients. Since it crosses the pla-
centa, it can affect fetal heart tones in the same manner as the
mother’s.

**Adverse/Side Effects:** CNS: Fatigue, muscle weakness,
headache, facial neuralgia, mental depression, paresthesias,
hallucinations, confusion, drowsiness, agitation, dizziness •
CV: Dysrhythmias, hypotension, AV block • Eye: Visual distur-
bances • GI: Anorexia, nausea, vomiting, diarrhea • Other:
Diaphoresis, recurrent malaise, dysphagia.

**Interactions:** Quinidine, amiodarone, and calcium channel
blockers (verapamil, nifedipine, diltiazem) can increase serum
digoxin levels. Severe bradycardia can occur if digoxin is admin-
istered with IV beta blockers. Diuretics can cause potassium
depletion, which can lead to digoxin toxicity. Succinylcholine
may potentiate the arrhythmogenic effects of digoxin.

**Prehospital Considerations**
- IV administration: Direct IV injection of digoxin may be
  administered undiluted or diluted in 4 mL of sterile water,
  D5W, or NaCl 0.9% (if prescribed). Administer each direct
  IV dose over at least 5 min.
- Infiltration of parenteral drug into subcutaneous tissue can
  cause local irritation and sloughing.
- Be familiar with patient’s baseline data (e.g., quality of
  peripheral pulses, blood pressure, clinical symptoms, serum
  electrolytes, creatinine clearance) as a foundation for making
  assessments.
- Before administering digoxin, check laboratory reports for
  serum levels of digoxin, potassium, magnesium, and calci-
  um. Notify physician of abnormal changes.
- Before administering digoxin, take apical pulse for 1 full
  min noting rate, rhythm, and quality. If changes are noted,
  withhold digoxin, take rhythm strip if patient is on ECG
  monitor, and notify physician promptly.
• Monitor for signs and symptoms of digoxin toxicity.
• Although a fall in ventricular rate to 60/min in adults (70/min in children) is one criterion for withholding medication, any change in pulse rate or rhythm should be interpreted as a sign of digitalis intoxication and should be reported promptly.
• In children, cardiac dysrhythmias are usually reliable signs of early toxicity. Early indicators in adults (anorexia, nausea, vomiting, diarrhea, visual disturbances) are rarely initial signs in children.

DIGOXIN IMMUNE FAB

Class: Antidote
Trade Name: Digibind

Therapeutic Actions/Pharmacodynamics: Digoxin Immune Fab is comprised of purified fragments of antibodies specific for digoxin (but also effective for digitoxin) produced in sheep immunized with digoxin-albumin conjugate. Using fragments of antidigoxin antibodies (Fab) instead of whole antibody molecules permits more extensive and faster distribution to serum and toxic cellular sites. It acts by selectively forming a complex with circulating digoxin or digitoxin, thereby preventing the drug from binding at receptor sites; the complex is then eliminated in urine.

Emergency Uses: To treat potentially life-threatening digoxin or digitoxin intoxication in carefully selected patients. Adult dose: Dosages vary according to amount of digoxin to be neutralized; dosages are based on total body load or steady state serum digoxin concentrations (see package insert); some patients may require a second dose after several hours.

Pharmacokinetics
Absorption: Onset is within 1 min after IV administration; half-life is 14–20 hr.
Elimination: Excreted in urine over 5–7 days.
Contraindications and Precautions: Digoxin Immune Fab is contraindicated in patients with a hypersensitivity to sheep products; renal or cardiac failure. Safe use during pregnancy (category C) or in nursing mothers not established. Use with caution in patients with prior treatment with sheep antibodies or ovine Fab fragments; history of allergies; impaired renal function.

Adverse/Side Effects: Adverse reactions associated with use of digoxin immune Fab are related primarily to the effects of digitalis withdrawal on the heart. Allergic reactions have been reported rarely. Hypokalemia.

Interactions: None established.

Prehospital Considerations
- Reconstitute by dissolving 38 mg (1 vial) in 4 mL of sterile water for injection; mix gently (solution will contain 9.5 mg/mL). For administration by IV infusion, reconstituted solution may be diluted further with sterile isotonic saline injection.
- After reconstitution, digoxin immune Fab is administered by IV infusion over 30 min, preferably through a 0.22-µm membrane filter, or as a bolus injection if cardiac arrest is imminent.
- For infants, reconstitute as directed and administer with a tuberculin syringe. For small doses (e.g., 2 mg or less), dilute the reconstituted 40 mg vial with 36 mL of sterile isotonic saline injection to make a concentration of 1 mg/mL. Children must be closely monitored for fluid overload.
- Reconstituted solutions should be used promptly or refrigerated at 2–8°C (36–46°F) for up to 4 hr.
- Effective treatment should be reflected in improvement in cardiac rhythm abnormalities, mental orientation, and other neurologic symptoms, and GI and visual disturbances.
• Reversal of signs and symptoms of digitalis toxicity occurs in 15–60 min in adults and usually within minutes in children.
• Cardiac status may deteriorate as inotropic action of digitalis is withdrawn by action of immune Fab. Closely monitor for CHF, dysrhythmias, increase in heart rate, and hypokalemia.

DILTIAZEM

Class: Calcium channel blocker
Trade Names: Apo-Diltiaz (Can), Cardizem, Dilacor, Tiazac

Therapeutic Actions/Pharmacodynamics: Diltiazem is a slow calcium channel blocker with pharmacologic actions similar to those of verapamil. It inhibits calcium ion influx through slow channels into cell of myocardial and arterial smooth muscle (both coronary and peripheral blood vessels). As a result, intracellular calcium remains at subthreshold levels insufficient to stimulate cell excitation and contraction. It also dilates coronary arteries and arterioles and inhibits coronary artery spasm; thus myocardial oxygen delivery is increased (antianginal effect). Diltiazem slows SA and AV node conduction (antidysrhythmic effect) without affecting normal arterial action potential or intraventricular conduction. By vasodilation of peripheral arterioles it decreases total peripheral vascular resistance and reduces arterial BP at rest (antihypertensive effect). It may cause slight decrease in heart rate without altering total serum calcium levels.

Emergency Uses: To control supraventricular tachydysrhythmias (atrial fibrillation, atrial flutter, PSVT refractory to adenosine); to increase coronary artery perfusion in angina pectoris. Adult dose: 0.25 mg/kg IV bolus over 2 min; if inadequate response, may repeat in 15 min with 0.35 mg/kg, fol-
lowed by a continuous infusion of 5–10 mg/hr (recommended maximum dose: 15 mg/hr for 24 hr).

**Pharmacokinetics**
*Absorption:* Onset is 3 min; peak effect in 7 min; duration is 1–3 hr; half-life is 2 hr.
*Distribution:* Distributed into breast milk.
*Metabolism:* Metabolized in liver.
*Elimination:* Excreted primarily in urine with some elimination in feces.

**Contraindications and Precautions:** Diltiazem is contraindicated in patients with a known hypersensitivity to the drug; sick sinus syndrome (unless pacemaker is in place and functioning); second- or third-degree AV block; severe hypotension (systolic less than 90 mm Hg or diastolic less than 60 mm Hg). Do not use in patients with wide-complex tachycardia (ventricular tachycardia) in the prehospital setting. Do not use if patient has Wolf-Parkinson-White syndrome. Its safe use in pregnancy (category C), in nursing mothers, and in children has not been established. Use with caution in CHF (especially if patient is also receiving a beta blocker), conduction abnormalities; renal or hepatic impairment; the elderly; nursing mothers.

**Adverse/Side Effects:** CNS: Headache, fatigue, dizziness, asthenia, drowsiness, nervousness, insomnia, confusion, tremor, gait abnormality • CV: Edema, dysrhythmias, angina, second- or third-degree AV block, bradycardia, CHF, flushing, hypotension, syncope, palpitations • GI: Nausea, constipation, anorexia, vomiting, diarrhea, impaired taste, weight increase • Skin: Rash.

**Interactions:** Digoxin should not be used with IV beta blockers because of the increased risk of congestive heart failure, bradycardia, and asystole. Diltiazem may increase digoxin or quinidine levels. Cimetidine may increase diltiazem levels, thus increasing its effects. Diltiazem may increase cyclosporine levels.
Prehospital Considerations

- Calcium chloride can be used to prevent the hypotensive effects of diltiazem in overdose situations.
- Diltiazem may be given by direct IV as a bolus dose over 2 min. A second bolus may be administered after 15 min.
- Diltiazem may be given by continuous IV infusion. The recommended rate is 5−15 mg/hr. Infusion duration longer than 24 hr and infusion rate greater than 15 mg/hr are not recommended.
- For continuous IV infusion, diltiazem may be added to any of the following: D₅W, NS, D₅W/0.45% NaCl.
- Diltiazem should be kept refrigerated but can be kept at room temperature for one month, then discarded if not used.
- Withhold drug if systolic BP is less than 90 mm Hg or diastolic is less than 60 mm Hg.
- BP and ECG should be evaluated before initiation of therapy and monitored particularly during dosage adjustment period. Baseline and periodic tests of liver and renal function are also recommended.

DIMENHYDRINATE

**Class:** Antihistamine

**Trade Names:** Andrumin (Aus), Apo-Dimenhydrinate (Can), Dinate, Dommanate, Dramamine, Gravol (Can), Marmine, Nauseatol (Can), Novo-Dimanate (Can), Travamine (Can)

**Therapeutic Actions/Pharmacodynamics:** Dimenhydrinate is an H₁-receptor antagonist that shares similar properties with diphenhydramine. Although formally classified as an antihistamine, it is rarely used for this purpose. It is most often used in the prevention and treatment of motion sickness, vertigo, and labyrinthitis. Its precise mode of antinauseant action is not known, but it is thought to inhibit cholinergic stimulation in
vestibular and associated neural pathways. It also is used with analgesics, particularly narcotics.

**Emergency Uses:** To relieve nausea/vomiting associated with motion sickness and narcotic use. *Adult dose:* 12.5–25 mg IV; 50 mg IM every 4 hr as needed. *Pediatric dose:* 1.25 mg/kg every 4 hr up to 300 mg/day.

**Pharmacokinetics**

*Absorption:* Onset is immediate if given IV; 20–30 min IM; duration is 3–6 hr. *Distribution:* Distributed into breast milk. *Elimination:* excreted in urine.

**Contraindications and Precautions:** There are no absolute contraindications when used in the emergency setting. Use with caution in patients with seizure disorders and asthma.

**Adverse/Side Effects:** CNS: Drowsiness, headache, incoordination, dizziness, blurred vision, nervousness, restlessness, insomnia (especially children) • CV: Hypotension, palpitation • Other: Dry mouth, nose, throat • Less frequently: anorexia, constipation or diarrhea, urinary frequency, dysuria.

**Interactions:** Alcohol and other CNS depressants enhance CNS depression, drowsiness. Tricyclic antidepressants compound its anticholinergic effects.

**Prehospital Considerations**

- Dimenhydrinate may be given by direct IV. Dilute each 50 mg in 10 mL of NS. Administer 50 mg or fraction thereof over 2 min.
- Examine parenteral preparation for particulate matter and discoloration. Do not use unless absolutely clear.
- Causes high incidence of drowsiness. Side rails and supervision of ambulation may be indicated.
- Tolerance to CNS depressant effects usually occurs after a few days of drug therapy. Some decrease in antiemetic action may result with prolonged use.
- Antihistamines can obscure signs of dizziness, nausea, and
vomiting associated with drug toxicity and serious disease conditions.

- To prevent motion sickness, dimenhydrinate should be taken 30 min before departure and should be repeated before meals and upon retiring.

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**DIMERCAPROL**

**Class:** Antidote

**Trade Names:** BAL in Oil

**Therapeutic Actions/Pharmacodynamics:** Dimercaprol is a dithiol compound that combines with ions of various heavy metals to form relatively stable, nontoxic, soluble complexes called chelates, which can be excreted. This action prevents the inhibition of sulfhydryl enzymes by toxic metal. It may also reactivate affected enzymes but is most effective when administered prior to enzyme damage.

**Emergency Uses:** As an antidote for acute arsenic and gold poisoning. **Adult dose:** 2.5–3.0 mg/kg IM. **Pediatric dose:** Same as for adult.

As an antidote for acute mercury poisoning. **Adult dose:** 5 mg/kg IM. **Pediatric dose:** Same as for adult.

As an antidote for acute lead encephalopathy. **Adult dose:** 4 mg/kg IM. **Pediatric dose:** Same as for adult.

**Pharmacokinetics**

**Absorption:** Onset and peak effect in 30–60 min.

**Distribution:** Distributed mainly in intracellular spaces, including brain; highest concentrations in liver and kidneys.

**Elimination:** Completely excreted in urine and bile within 4 hr.

**Contraindications and Precautions:** Dimercaprol is contraindicated in hepatic insufficiency (with exception of postarsenical jaundice); severe renal insufficiency; poisoning due to cadmium, iron, selenium, or uranium. Use with caution.
in hypertensive patients. Safe use during pregnancy (category D), and in nursing mothers not established.

**Adverse/Side Effects:** CNS: Headache, anxiety, muscle pain or weakness, restlessness, paresthesias, tremors, convulsions, shock • CV: Elevated BP, tachycardia • ENT: Rhinorrhea; burning sensation, feeling of pain and constriction in throat • GI: Nausea, vomiting; burning sensation in lips and mouth, halitosis, salivation; abdominal pain, metabolic acidosis • GU: Burning sensation in penis, renal damage • Other: Pains in chest or hands, pain and sterile abscess at injection site, sweating, dental pain.

**Interactions:** Iron, cadmium, selenium, and uranium form toxic complexes with dimercaprol.

**Prehospital Considerations**
- Because irreversible tissue damage may occur quickly, particularly in mercury poisoning, dimercaprol therapy must be initiated as soon as possible (within 1–2 hr) after ingestion of the poison.
- Administered by deep IM injection only. Local pain, gluteal abscess, and skin sensitization have been reported.
- Contact of drug with skin may produce erythema, edema, dermatitis. Handle with caution.
- Presence of sediment in ampule reportedly does not indicate drug deterioration.
- Monitor vital signs. Elevations of systolic and diastolic BPs accompanied by tachycardia frequently occur within a few minutes following injection and may remain elevated up to 2 hr.
- Fever occurs in approximately 30% of children receiving treatment and may persist throughout therapy.
- Minor adverse reactions usually reach maximum levels 15–20 min after drug administration and generally subside in 30–90 min. Ephedrine or an antihistamine is sometimes administered to prevent symptoms.

**Dimercaprol**
DOBUTAMINE

Class: Sympathomimetic
Trade Name: Dobutrex

Therapeutic Actions/Pharmacodynamics: Dobutamine produces its inotropic effect by acting on beta receptors and primarily on myocardial alpha-adrenergic receptors. It increases cardiac output (positive inotropic effect) and decreases pulmonary wedge pressure and total systemic vascular resistance with comparatively little or no effect on BP or heart rate. Its primary use is inotropic support in short-term treatment of adults with cardiac decompensation due to depressed myocardial contractility (cardiogenic shock) resulting from either organic heart disease or from cardiac surgery. Dobutamine also increases conduction through the AV node. It has lower potential for precipitating dysrhythmias than dopamine. In CHF, increasing cardiac output enhances renal perfusion and increases renal output and renal sodium excretion.

Emergency Uses: To increase cardiac output in congestive heart failure/cardiogenic shock. Adult dose: 2–20 µg/kg/min IV. Pediatric dose: Same as adult.

Pharmacokinetics
Absorption: Onset is 2–10 min; peak effect in 10–20 min; half-life is 2 min.
Metabolism: Metabolized in liver and other tissues by catechol-o-methyl-transferase (COMT).
Elimination: Excreted in urine.

Contraindications and Precautions: Dobutamine is contraindicated in patients with a history of hypersensitivity to other sympathomimetic amines, ventricular tachycardia, or idiopathic hypertrophic subaortic stenosis. Safe use in pregnancy (category C), in nursing mothers, and children, or following acute MI not established. Use with caution in patients with pre-existing hypertension or atrial fibrillation. Dobutamine should
not be used as a sole agent in managing hypovolemic shock until fluid volume is restored. To increase cardiac output in severe emergencies, such as cardiogenic shock, dopamine is the drug of choice.

**Adverse/Side Effects:** CNS: Headache, tremors, paresthesias, mild leg cramps, nervousness, fatigue (with overdosage) • CV: Increased heart rate and BP, premature ventricular beats, palpitation, anginal pain • GI: Nausea, vomiting • Other: Nonspecific chest pain, shortness of breath.

**Interactions:** Beta-adrenergic blocking agents, e.g., metoprolol or propranolol, may make dobutamine ineffective in increasing cardiac output, but total peripheral resistance may increase. Since MAO inhibitors and tricyclic antidepressants may potentiate the vasopressor effects of dobutamine, use with extreme caution.

**Prehospital Considerations**
- Hypovolemia should be corrected by administration of appropriate volume expanders prior to initiation of therapy.
- Since dobutamine enhances AV conduction, patients with atrial fibrillation are generally given a digitalis preparation prior to initiation of therapy to reduce risk of ventricular tachycardia.
- Rate of infusion should be controlled by an infusion pump (preferred) or a microdrip IV infusion set.
- Solutions containing dobutamine may exhibit color changes because of slight oxidation of drug. This does not affect potency.
- Dobutamine is incompatible with sodium bicarbonate and other alkaline solutions.
- At any given dosage level, drug takes 10–20 min to produce peak effects.
- ECG and BP should be monitored continuously during administration of dobutamine.
• IV infusion rate and duration of therapy are determined by heart rate, blood pressure, and ectopic activity.
• Marked increases in blood pressure (systolic pressure is the most likely to be affected) and heart rate, or the appearance of dysrhythmias or other adverse cardiac effects, are usually reversed promptly by reduction in dosage.
• Patients with preexisting hypertension must be closely observed for exaggerated pressor response.

DOPAMINE

Class: Sympathomimetic
Trade Names: Intropin, Revimine (Can)

Therapeutic Actions/Pharmacodynamics: Dopamine is a naturally occurring neurotransmitter and immediate precursor of norepinephrine. Its major cardiovascular effects are produced by direct action on alpha- and beta-adrenergic receptors and on specific dopaminergic receptors in mesenteric and renal vascular beds. Its positive inotropic effect on myocardium increases cardiac output with increase in systolic and pulse pressure and little or no effect on diastolic pressure. Dopamine improves circulation to the renal vascular bed by decreasing renal vascular resistance with a resulting increase in glomerular filtration rate and urinary output.

Emergency Uses: To increase end-organ perfusion in cardiogenic shock and in hemodynamically significant hypotension (70–100 mmHg) not resulting from hypovolemia. Adult dose: 2–5 µg/kg/min up to 20 µg/kg/min, titrated to effect. Pediatric dose: Same as adult

Pharmacokinetics

Absorption: Onset is less than 5 min; duration is less than 10 min; half-life is 2 min.
Distribution: Widely distributed; does not cross blood-brain barrier.
**Metabolism:** Inactive in the liver, kidney, and plasma by MAO and COMT.

**Elimination:** Excreted in urine.

**Contraindications and Precautions:** Dopamine is contraindicated in patients with pheochromocytoma (adrenal gland tumor); tachydyssrhythmias, or ventricular fibrillation. Safe use during pregnancy (category C), in nursing women, and in children not established. Cautious use in patients with history of occlusive vascular disease (e.g., Buerger’s or Raynaud’s disease), cold injury, diabetic endarteritis, or arterial embolism. Always ensure patient is normovolemic prior to using dopamine for hypovolemic shock. Infusion rates above 20 µg/kg/min will cause profound vasoconstriction because of the drug’s predominantly alpha stimulation at that dose range.

**Adverse/Side Effects:** CV: Hypotension, ectopic beats, tachycardia, anginal pain, palpitation, vasoconstriction (indicated by disproportionate rise in diastolic pressure), cold extremities; less frequent: aberrant conduction, bradycardia, widening of QRS complex, elevated blood pressure • GI: Nausea, vomiting • Other: Headache, necrosis, tissue sloughing with extravasation, gangrene, azotemia, piloerection, dyspnea, dilated pupils (high doses).

**Interactions:** Like all catecholamines, dopamine is deactivated by alkaline solutions such as sodium bicarbonate. MAO inhibitors increase the alpha-adrenergic effects of dopamine and cause headache, hyperpyrexia, and hypertension. Phenytoin may decrease dopamine action and actually cause hypotensive effects. Beta blockers will antagonize its cardiac effects just as alpha blockers will antagonize peripheral vasoconstriction.

**Prehospital Considerations**
- Before initiation of dopamine therapy, hypovolemia should be corrected
- IV infusion rate and guidelines for adjusting rate of flow are
in relation to changes in blood pressure. Microdrip or another reliable metering device should be used for accuracy of flow rate.
• Infusion rate must be continuously monitored for free flow, and care must be taken to avoid extravasation, which can result in tissue sloughing and gangrene. For this reason, infusion is made preferably into a large vein of the antecubital fossa.
• Protect dopamine from light. Discolored solutions should not be used.
• Monitor blood pressure, pulse, and peripheral pulses every 5 min. Precise measurements are essential for accurate titration of dosage.
• Close observation is critical when patient is receiving dopamine. The following indicators are used for decreasing or temporarily suspending dose (report promptly to physician): ascending tachycardia; dysrhythmias; disproportionate rise in diastolic pressure (marked decrease in pulse pressure); signs of peripheral ischemia (pallor, cyanosis, mottling, coldness, complaints of tenderness, pain, numbness, or burning sensation). Presence of peripheral pulses is not always indicative of adequate circulation.
• In addition to improvement in vital signs, other indices of adequate dosage and perfusion of vital organs include loss of pallor, increase in toe temperature, adequacy of nail bed capillary filling, and reversal of confusion or comatose state.

DROPERIDOL
Class: Antiemetic
Trade Name: Inapsine
Therapeutic Actions/Pharmacodynamics: Droperidol is a butyrophenone derivative structurally and pharmacologically related to haloperidol. It antagonizes emetic effects of mor-
Pain-like analgesics and other drugs that act on the chemoreceptor trigger zone (CTZ). Its mild alpha-adrenergic blocking activity and direct vasodilator effect may cause hypotension. Droperidol acts primarily at the subcortical level to produce sedation. Its sedative property reduces anxiety and motor activity without necessarily inducing sleep, so the patient remains responsive. It potentiates other CNS depressants.

**Emergency Uses:** To reduce nausea and vomiting in patients refractory to the first-line antiemetics (promethazine, chlorpropazine, etc.) and to produce a tranquilizing effect. It also can be used as an antipsychotic in patients showing marked psychosis requiring pharmacologic therapy. *Adult dose:* 2.5–10.0 mg IV. *Pediatric dose:* 0.088–0.165 mg/kg IV.

**Pharmacokinetics**

**Absorption:** Onset is 3–10 min; peak effect in 30 min; duration is 2–4 hr; may persist up to 12 hr.

**Distribution:** Crosses placenta.

**Metabolism:** Metabolized in liver.

**Elimination:** Excreted in urine and feces.

**Contraindications and Precautions:** Droperidol is contraindicated in patients with a known intolerance to the drug. Safe use during pregnancy (category C) and in children under 2 yr not established. Use with caution in elderly, debilitated, and other poor-risk patients; and patients with Parkinson’s disease, hypotension, liver, kidney, cardiac disease, bradydysrhythmias.

**Adverse/Side Effects:** CNS: Drowsiness, extrapyramidal symptoms: dystonia, akathisia, oculogyric crisis; dizziness, restlessness, anxiety, hallucinations, mental depression • CV: Hypotension, tachycardia • Other: Chills, shivering, laryngospasm, bronchospasm.

**Interactions:** None reported.

**Prehospital Considerations**

• When patient is under the effect of another CNS depressant,
the required dose of droperidol may be less than usual. Postoperative narcotics or other CNS depressants are prescribed in reduced doses since they have additive or potentiating effects.

- Protect from light. Store at 15–30°C (59–86°F), unless otherwise directed by manufacturer.
- Monitor vital signs closely. Hypotension and tachycardia are common side effects.
- Because of possibility of severe orthostatic hypotension, always exercise care in moving and positioning the medicated patient. Avoid abrupt changes in position.
- Patient who receives a narcotic analgesic concurrently should be observed carefully for signs of impending respiratory depression.
- Elevated BP has been reported following administration of droperidol with parenteral analgesics.
- Droperidol may aggravate symptoms of acute depression.

**ENOXAPARIN**

**Class:** Anticoagulant  
**Trade Name:** Lovenox

**Therapeutic Action/Pharmacodynamics:** Enoxaparin is a low-molecular-weight heparin derivative that accelerates the formation of the antithrombin III-thrombin complex and deactivated thrombin, preventing the conversion of fibrinogen to fibrin. It is used primarily to prevent pulmonary embolism and deep vein thrombosis following hip and knee replacement surgery. Evidence also suggests that enoxaparin may be effective in the treatment of deep venous thrombosis and pulmonary embolism. It also can be used, in combination with oral aspirin, for patients with coronary ischemia associated with unstable angina and non-Q-wave myocardial infarction.
Emergency Uses: To inhibit clot formation in unstable angina and non-Q-wave myocardial infarction. Adult dose: 1 mg/kg SC. To treat pulmonary embolism: Adult dose: 0.5 mg/kg IV.

Pharmacokinetics
Absorption: 92% absorbed; onset and peak within 3–5 hr; half-life is 4.5 hr.
Distribution: Preferential to the kidneys, liver, and spleen.
Excretion: Excreted in urine and breast milk.

Contraindications and Precautions: Enoxaparin is contraindicated in patients with a hypersensitivity to the drug, to pork products, and to heparin. Do not use in patients with active major bleeding or thrombocytopenia.

Adverse/Side Effects: CNS: Confusion, dizziness • CV: Edema, peripheral edema, chest pain, irregular heartbeat • GI: Nausea • Other: Irritation, pain, hematoma, or erythema at injection site, ecchymosis, bleeding complications, angioedema, rash, hives.
Interactions: NSAIDs, warfarin, antiplatelet agents (ticlopidine, dipyridamole) increase the risk of bleeding.

Prehospital Considerations
• Use with caution in the elderly and in any patients with an increased risk for bleeding, such as bacterial endocarditis, congenital bleeding disorders, ulcer disease, hemorrhagic stroke or recent brain or spinal surgery.
• Never administer this drug IM.
• Don’t massage the area following injection. Watch for signs of bleeding at site.

**EPINEPHRINE**

*Class:* Sympathomimetic

*Trade Names:* Adrenalin, Epinephrine

*Therapeutic Actions/Pharmacodynamics:* Epinephrine is a
naturally occurring catecholamine obtained from animal adrenal glands; but is also prepared synthetically. Acting directly on both alpha- and beta-adrenergic receptors, it is the most potent activator of alpha receptors. Epinephrine imitates all actions of the sympathetic nervous system except those on the arteries of the face and sweat glands. Its beta₁ effects strengthen myocardial contraction; increase systolic but may decrease diastolic blood pressure; and increase cardiac rate and cardiac output. Its beta₂ effects dilate the bronchiole smooth muscle and inhibit mucus secretion that decreases overall airway resistance. Its alpha effects constrict the bronchial arterioles and inhibit histamine release, thus reducing congestion and edema and increasing tidal volume and vital capacity. Epinephrine also constricts arterioles, particularly in the skin, mucous membranes, and kidneys, but dilates skeletal muscle blood vessels. It relaxes uterine smooth musculature and inhibits uterine contractions. Its CNS actions are believed to result from peripheral effects.

**Emergency Uses:** To restore cardiac rhythm in cardiac arrest.

*Adult dose:* 1 mg 1:10,000 IV every 3–5 min until circulation restored. If given via ET tube, give 2.0–2.5 mg 1:1,000.  
*Pediatric dose:* 0.01 mg/kg 1:10,000 IV/IO. If given via ET, give 0.1 mg/kg of 1:1,000. All subsequent doses (IV/IO) at 0.1 mg/kg of 1:1,000.

For treatment of allergic reactions. *Adult dose:* 0.3–0.5 mg 1:1,000 SC, every 5–15 min as needed; or 0.5–1.0 mg 1:10,000 IV if SC dose ineffective or reaction severe. *Pediatric dose:* 0.01 mg/kg 1:1000 SC or 0.01 mg/kg of 1:10,000 IV every 10–15 min if SC dose ineffective or reaction severe.

**Pharmacokinetics**

*Absorption:* Onset is less than 2 min IV, 3–10 min SC, less than 1 min ET; peak effect in less than 5 min IV/ET, 20 min SC; duration is 5–10 min IV/ET, 20–30 min SC.  
*Distribution:* Widely distributed; does not cross blood- brain barrier.
barrier; crosses placenta. *Metabolism:* Metabolized in tissue and liver by MAO and COMT. 

*Elimination:* Small amount excreted unchanged in urine; excreted in breast milk.

**Contraindications and Precautions:** Epinephrine is contraindicated in patients with a hypersensitivity to sympathomimetic amines; narrow-angle glaucoma; hemorrhagic, traumatic, or cardiogenic shock; cardiac dilatation, cerebral arteriosclerosis, coronary insufficiency, dysrhythmias, organic heart or brain disease; during second stage of labor. Safe use during pregnancy (category C), in nursing women, and in children not established. Use with caution in the elderly or debilitated patients; hypertension; diabetes mellitus; hyperthyroidism; Parkinson’s disease; tuberculosis; in patients with long-standing bronchial asthma and emphysema with degenerative heart disease; in children less than 6 yr of age.

**Adverse/Side Effects:** CNS: Nervousness, restlessness, sleeplessness, fear, anxiety, tremors, severe headache, cerebrovascular accident, weakness, dizziness, syncope • CV: Precordial pain, palpitations, hypertension, MI, tachydysrhythmias including ventricular fibrillation • GI: Nausea, vomiting • Skin: Pallor, sweating, tissue necrosis with repeated injections.

**Interactions:** Epinephrine may increase hypotension in circulatory collapse or hypotension caused by phenothiazines. It has additive toxicities with other sympathomimetics. Alpha- and beta-adrenergic blocking agents antagonize the effects of epinephrine. Epinephrine is pH dependent and can be deactivated when administered with highly alkaline solutions such as bicarbonate.

**Prehospital Considerations**
- A tuberculin syringe may ensure greater accuracy in measurement of parenteral doses.
- Epinephrine injection should be protected from exposure to light at all times. Do not remove ampule or vial from carton until ready to use.
• Carefully aspirate before injecting epinephrine. Inadvertent IV injection of usual SC doses can result in sudden hypertension and possibly cerebral hemorrhage.
• Vascular constriction from repeated injections may cause tissue necrosis. Rotate injection sites and observe for signs of blanching.
• For IV use in cardiac resuscitation, if the 1:1000 1-mL ampules are used, the dose should be further diluted with 10 mL of sodium chloride injection.
• As a maintenance infusion, dilute in 500 mL 5% dextrose.
• IV administration: Give each 1 mg over 1 min or longer; may give more rapidly in cardiac arrest.
• Isoproterenol should not be used concurrently with epinephrine. A 4-hr interval should elapse before a change is made from one drug to the other.

ESMOLOL

Class: Beta blocker
Trade Name: Brevibloc

Therapeutic Actions/Pharmacodynamics: Esmolol is an ultrashort-acting beta1-adrenergic blocking agent with cardioselective properties but devoid of intrinsic sympathetic activity or membrane-stabilizing (quinidine-like) activity. Its hemodynamic effects are mild, with potency as a beta blocker about 1/100th that of propranolol. By competitive binding at beta-adrenergic receptors, it inhibits the agonist effect of catecholamines. Since it binds predominantly to beta1-receptors in cardiac tissue, sympathetically mediated increases in cardiac rate and BP are blocked.

Emergency Use: To convert supraventricular tachydyssrhythmias accompanied by a rapid ventricular response. Adult dose: Loading dose is 500 µg/kg/min IV for 1 min. Maintenance
dose is 50 µg/kg/min IV for 4 min. If unsuccessful, repeat loading dose every 4 min and increase maintenance dose by 50 µg/kg/min until desired effect is reached. Do not exceed maintenance dose of 300 µg/kg/min.

Pharmacokinetics

Absorption: Onset is within 5 min; peak effect in 10–20 min; duration is 10–30 min; half-life is 9 min.

Metabolism: Rapidly hydrolyzed by RBC esterases.

Elimination: Eliminated in urine.

Contraindications and Precautions: Esmolol is contraindicated in cardiac failure, heart block greater than first degree, sinus bradycardia, and cardiogenic shock. Safe use during pregnancy (category C), in nursing mothers, and in children not established. Use with caution in patients with a history of allergy or bronchial asthma, bronchospasm, emphysema; CHF; diabetes mellitus; renal function impairment.

Adverse/Side Effects:

CNS: Headache, dizziness, somnolence, confusion, agitation • CV: Hypotension (dose related), cold hands and feet, bradydysrhythmias, flushing, myocardial depression • GI: Nausea, vomiting • Respiratory: Dyspnea, chest pain, rhonchi, bronchospasm • Skin: Infusion site inflammation (redness, swelling).

Interactions: Esmolol may increase digoxin levels 10–20%. IV morphine may increase esmolol levels by 45%; succinylcholine may prolong neuromuscular blockade. Never administer esmolol to patients receiving IV calcium channel blockers (verapamil) because profound hypotension may occur.

Prehospital Considerations

• Esmolol must be diluted before administration (available as a solution: 250 mg/mL in 10-mL ampules). Dilute each 5g with 500 mL of D2W, NS, or other appropriate diluent (see manufacturer’s directions). Resulting solution yields 10 mg/mL. Caution: A stronger solution (e.g., 20 mg/mL) may cause venous irritation and thrombophlebitis.
• Do not admix with other drugs before dilution in a suitable IV fluid.
• The diluted infusion solution is stable for at least 24 hr at room temperature.
• Monitor BP, pulse, ECG, during esmolol infusion.
• Hypotension may have its onset during the initial titration phase; thereafter the risk increases with increasing doses. Usually the hypotension experienced during esmolol infusion is resolved within 30 min after infusion is reduced or discontinued.
• IV site reactions (burning, erythema) or diaphoresis may develop during infusion. Both reactions are temporary, but injection site should be changed if local reaction occurs. Blood chemistry abnormalities have not been reported.
• Overdose symptoms: Discontinue administration if the following symptoms occur: bradycardia, severe dizziness or drowsiness, dyspnea, bluish-colored fingernails or palms of hands, seizures.

ETOMIDATE
Class: Hypnotic
Trade Name: Amidate
Therapeutic Action/Pharmacodynamics: Etomidate is an ultra-short-acting, nonbarbiturate hypnotic, with no analgesic effects, used for facilitated intubation. It produces a rapid induction of anesthesia with minimal cardiovascular and respiratory effects. It is rapidly distributed following IV injection or infusion and rapidly metabolized and excreted. Etomidate has advantages over other short-acting induction anesthetics, particularly barbiturates, in that it does not cause histamine release; its effects on the cardiovascular and respiratory systems are minimal, and there are no reports of organ toxicity, or biochemical or hematological disturbances. Its short duration
of action and relative safety led investigators to study its use in a variety of surgical procedures, including cesarean section and open heart surgery. In the prehospital setting, it is an effective drug to induce sedation for facilitated intubation.

**Emergency Uses:** To induce sedation for endotracheal intubation. **Adult dose:** 0.1–0.3 mg/kg IV over 15–30 seconds. **Pediatric dose:** Children older than 10 yr, same as for adult.

**Pharmacokinetics**

**Absorption:** Onset in 10–20 seconds, peak effects within 1 min; duration is 3–5 min; half-life is 30–74 min.

**Metabolism:** Rapidly metabolized in the liver with inactive metabolites.

**Elimination:** Excreted mainly through the urine.

**Contraindications and Precautions:** Etomidate is contraindicated in patients with a hypersensitivity to the drug. Use with caution in patients with marked hypotension, severe asthma, or severe cardiovascular disease. Its safety in children under 10 yr has not been established.

**Adverse/Side Effects:** CNS: Myoclonic skeletal muscle movements, tonic movements • Respiratory: Apnea, hyperventilation or hypoventilation, laryngospasm • CV: Either hypertension or hypotension; tachycardia or bradycardia; dysrhythmias • GI: Nausea, vomiting • Miscellaneous: Eye movements (common), hiccups, snoring.

**Interactions:** None in the emergency setting.

**Prehospital Considerations**

- Verapamil may cause prolonged respiratory depression and apnea.
- It is important to remember that etomidate does not have any analgesic properties. Thus, an analgesic should be administered with etomidate for painful procedures such as electrical cardioversion.
- Nausea is common following recovery from etomidate. This side effect should be expected and treated accordingly.
• Myotonic jerks are also common following etomidate administration. Although benign, these jerks can cause pain in patients with injuries such as long bone fractures.
• Flumazenil DOES NOT reverse the effects of etomidate.

FENTANYL CITRATE

Class: Narcotic analgesic
Trade Name: Sublimaze

Therapeutic Actions/Pharmacodynamics: Fentanyl is a potent synthetic narcotic agonist analgesic with pharmacologic actions qualitatively similar to those of morphine and meperidine, but whose action is more prompt and less prolonged. Its principal actions are analgesia and sedation. Drug-induced alterations in respiratory rate and alveolar ventilation may persist beyond the analgesic effect. The emetic effect is less than with either morphine or meperidine.

Emergency Uses: To induce sedation during rapid sequence intubation procedure; to control severe pain. Adult dose: 25–100 µg slow IV (over 2–3 min). Pediatric dose: 2.0 µg/kg slow IV/IM.

Pharmacokinetics
Absorption: Onset is immediate; peak effect in 3–5 min IV; duration is 30–60 min.
Metabolism: Metabolized in liver.
Elimination: Excreted in urine.

Contraindications and Precautions: Fentanyl is contraindicated in patients who have received MAO inhibitors within 14 days; myasthenia gravis. Safe use during pregnancy (category C) and in children under 2 yr not established. Use with caution in head injuries, increased intracranial pressure; elderly, debilitated, poor-risk patients; COPD, other respiratory problems; liver and kidney dysfunction; bradydysrhythmias.
**Adverse/Side Effects:** CNS: Sedation, euphoria, dizziness, diaphoresis, delirium, convulsions with high doses • CV: Hypotension, bradycardia, circulatory depression, cardiac arrest • Eye: Miosis, blurred vision • GI: Nausea, vomiting, constipation, ileus • Respiratory: Laryngospasm, bronchoconstriction, respiratory depression or arrest • Other: Muscle rigidity, especially muscles of respiration after rapid IV infusion, urinary retention, rash.

**Interactions:** Alcohol and other CNS depressants potentiate its effects; MAO inhibitors may precipitate hypertensive crisis.

**Prehospital Considerations**
- Parenteral doses may be given undiluted or diluted in 5 mL sterile water or NS. Administer by direct IV over 1−2 min.
- Store at 15−30 C (59−86 F) unless otherwise directed. Protect drug from light.
- Monitor vital signs and observe patient for signs of skeletal and thoracic muscle (depressed respirations) rigidity and weakness.
- Duration of respiratory depressant effect may be considerably longer than narcotic analgesic effect. Have immediately available oxygen, resuscitative and intubation equipment, and an opioid antagonist such as naloxone.

**FLECAINIDE**

**Class:** Antidysrhythmic

**Trade Name:** Tambocor

**Therapeutic Actions/Pharmacodynamics:** Flecainide is a local (membrane) anesthetic and antidysrhythmic with electrophysiologic properties similar to other class IC antidysrhythmic drugs. It slows conduction velocity throughout the myocardial conduction system and increases ventricular refractoriness but has little effect on repolarization. Flecainide
prolongs the His-ventricular (HQ) and QRS intervals at therapeutic doses. Clinically, flecainide causes both hypotension and negative inotropy (in higher dose ranges) and is an effective suppressant of PVCs and a variety of atrial and ventricular arrhythmias.

**Emergency Uses:** To convert atrial flutter and atrial fibrillation, AV nodal reentrant tachycardia, and SVT associated with Wolff-Parkinson-White syndrome. **Adult dose:** 100 mg PO every 12 hr; may increase by 50 mg twice a day every 4 days to a maximum of 400 mg/day. IV dose is 2 mg/kg administered at 10mg/min. **Pediatric dose:** 1–3 mg/kg/day PO in 3 divided doses (maximum 8 mg/kg/day).

**Pharmacokinetics**

**Absorption:** Readily absorbed from GI tract; peak effect in 2–3 hr; half-life is 7–22 hr.

**Distribution:** Crosses placenta; distributed into breast milk.

**Metabolism:** Metabolized in liver.

**Elimination:** Excreted mainly in urine.

**Contraindications and Precautions:** Flecainide is contraindicated in patients with a hypersensitivity to flecainide; preexisting second- or third-degree AV block, right bundle branch block when associated with a left hemiblock unless a pacemaker is present; cardiogenic shock, significant hepatic impairment. Use with caution in patients with CHF, sick sinus syndrome, and renal impairment.

**Adverse/Side Effects:** CNS: Dizziness, headache, light-headedness, unsteadiness, paresthesias, fatigue • CV: Arrhythmias, chest pain, worsening of CHF • Eye: Blurred vision, difficulty in focusing, and spots before eyes • GI: Nausea, constipation, change in taste perception • Other: Dyspnea, fever, and edema.

**Interactions:** Cimetidine may increase flecainide levels; may increase digoxin levels 15–25%; beta blockers may have additive negative inotropic effects.
Prehospital Considerations

• IV use of flecainide is not approved in the United States.
• Avoid using flecainide in patients who have had MI, because of its negative inotropic effects. It has been observed to increase mortality.
• Flecainide is limited by its need to be infused slowly, which may make it impractical for emergency medicine.
• Dosage increases more frequently than every 4 days are not recommended.
• Store in tightly covered, light-resistant containers at 15–30°C (59–86°F) unless otherwise directed.
• ECG monitoring is essential because of the possibility of drug-induced arrhythmias.
• Once arrhythmia is controlled, dosage reduction may be attempted with caution.
• Impress on patient the importance of taking drug at the prescribed times.
• Instruct patient to report visual disturbances.
• Instruct patient to report immediately passage of dark tarry stools, “coffee ground” emesis, frankly bloody emesis, or other GI distress.
• Advise patient to report immediately to physician the onset of skin rash, pruritus, jaundice.
• Inform patients about possible CNS effects (light-headedness, dizziness, drowsiness), and caution them to avoid dangerous activities until reaction to the drug has been determined.
• Patients should avoid self-medication with ibuprofen if taking prescribed drugs or if being treated for a serious condition without consulting physician.
• Patients should avoid taking aspirin or acetaminophen concurrently with ibuprofen.
• Inform patient that alcohol and NSAIDs may increase risk of GI ulceration and bleeding tendencies and should be avoided, unless otherwise advised by physician.
FLUMAZENIL

Class: Benzodiazepine antagonist
Trade Name: Romazicon

Therapeutic Actions/Pharmacodynamics: Flumazenil antagonizes the sedative effects of benzodiazepines in the central nervous system (sedation, impairment of recall, and psychomotor impairment) by inhibiting their effects on the GABA/benzodiazepine complex. Flumazenil is used to reverse the respiratory depression caused by the following drugs: diazepam (Valium), midazolam (Versed), lorazepam (Ativan), triazolam (Halcion), temazepam (Restoril), zolpidem (Ambien), flurazepam (Dalmane), clorazepate (Tranxene), oxazepam (Serax), clonazepam (Klonopin), quazepam (Doral), estazolam (ProSom), alprazolam (Xanax). It does not reverse the effects of opioids.

Emergency Use: To reverse the respiratory depression caused by benzodiazepines.

Adult dose: 0.2 mg IV over 30 seconds. May be repeated up to 1 mg.

Pharmacokinetics
Absorption: Onset in 1–5 min; peak effect in 6–10 min; duration is 2–4 hr; half-life is 54 min.
Metabolism: Metabolized in the liver to inactive metabolites.
Elimination: 90–95% excreted in urine, 5–10% in feces within 72 hr.

Contraindications and Precautions: Flumazenil should not be used as a diagnostic agent for benzodiazepine overdose in the same manner that naloxone is used for narcotic overdose. The potential for inducing a life-threatening withdrawal reaction in patients addicted to benzodiazepines with flumazenil is not worth the perceived benefits. It is contraindicated in patients with a hypersensitivity to flumazenil or to benzodiazepines; patients given a benzodiazepine for control of a life-threatening condition (such as status epilepticus); patients
showing signs of tricyclic antidepressant overdose; seizure-prone individuals during labor and delivery. Its effects on children are unknown. Use with caution in patients with hepatic function impairment, the elderly, pregnancy (category C), nursing mothers, intensive care patients, head injury, drug- and alcohol-dependent patients, and physical dependence upon benzodiazepines.

**Adverse/Side Effects:** CNS: Emotional lability, headache, dizziness, agitation, resedation, seizures, blurred vision • GI: Nausea, vomiting, hiccups • Other: Shivering, pain at injection site, hypoventilation.

**Interactions:** Few in the emergency setting.

**Prehospital Considerations**
- Ensure patency of IV before administration of flumazenil, since extravasation will cause local irritation.
- Flumazenil should be administered through an IV that is freely flowing into a large vein.
- Flumazenil should not be administered as a bolus dose, but rather each 0.2 mg dose should be given in small quantities over 15 seconds. Doses of flumazenil are given at 60-second intervals.
- In high-risk patients, slow the rate of administration of flumazenil to intervals of 6–10 min to provide the smallest effective dose.
- If resedation occurs, repeat doses may be given at 20-min intervals. Maximum dose for repeat treatment is 1 mg given at a rate of 0.2 mg/min, not to exceed 3 mg in any 1-hr period.
- Monitor patients for reversal of benzodiazepine for up to 120 min for respiratory depression and resedation.
- Benzodiazepine-induced ventilatory insufficiency may not be fully reversed by flumazenil; carefully monitor respiratory status until risk of resedation is unlikely.
- Monitor carefully for seizures and take appropriate precautions.
- Seizures induced by flumazenil administration probably will
not respond to treatment with a benzodiazepine. Instead, antiseizure drugs from another class, such as phenytoin or phenobarbital, must be used instead.

FOSPHENYTOIN

Class: Anticonvulsant
Trade Name: Cerebyx

Therapeutic Actions/Pharmacodynamics: Fosphenytoin is a prodrug of phenytoin. Following administration, fosphenytoin is converted to the anticonvulsant phenytoin. The cellular mechanisms of phenytoin are thought to be responsible for fosphenytoin’s anticonvulsant effects. Fosphenytoin is thought to modulate the sodium channels of neurons, modulate calcium flux across neuronal membranes, and enhance the sodium-potassium ATPase activity of neurons and glial cells.

Emergency Uses: Fosphenytoin is used to control seizures, especially status epilepticus; and to prevent seizures in seizure-prone patients. Adult dose: IV loading dose of 15–20 mg PE/kg administered at 100–150 mg PE/min. Initial maintenance dose is 4–6 mg PE/kg/day. All fosphenytoin doses are expressed in phenytoin sodium equivalents (PE) to ease calculations between phenytoin and fosphenytoin.

Pharmacokinetics

Absorption: completely absorbed after IV/IM administration; peak effect in 30 min IM.
Distribution: 95–99% protein bound. Crosses placenta, distributed into breast milk.
Metabolism: Extensively metabolized in the liver via oxidation.
Elimination: Phenytoin excreted in urine as metabolites.

Contraindications and Precautions: Fosphenytoin is contraindicated in patients with hypersensitivity to the drug.
seizures due to hypoglycemia, sinus bradycardia, complete or incomplete heart block, Stokes-Adams syndrome, pregnancy (category D), lactation. Use with caution in patients with impaired hepatic or renal function, alcoholism, hypotension, heart block, bradycardia, severe CAD, diabetes mellitus, hyperglycemia, and respiratory depression.

**Adverse/Side Effects:** CNS: Usually dose-related: paresthesias, tinnitus, nystagmus, dizziness, drowsiness, confusion, and tremors • CV: arrhythmias, hypotension, hypertension, cardiovascular collapse • GI: Nausea, vomiting, dysphagia • Eye: Conjunctivitis, photophobia, diplopia • Metabolic: Fever, hyperglycemia • Other: Rash, acute renal failure.

**Interactions:** Alcohol decreases fosphenytoin effects. Amiodarone, chloramphenicol, and omperazole increase fosphenytoin levels.

**Prehospital Considerations**
- Fosphenytoin may be administered IM in addition to IV.
- IV fosphenytoin should be diluted in D5W or NS.
- Closely monitor ECG, pulse, blood pressure, and respiratory function during administration.
- Discontinue infusion if rash appears.
- Carefully monitor for adverse effects.
- Fosphenytoin costs considerably more than phenytoin.

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**Furosemide**

**Class:** Loop diuretic

**Trade Names:** Apo-Furosemide (Can), Furoside (Can), Lasix, Novo-Semide (Can), Urex (Aus), Uritrol (Can)

**Therapeutic Actions/Pharmacodynamics:** Furosemide is a rapid-acting potent sulfonamide loop diuretic and antihypertensive with pharmacologic effects and uses almost identical to those of ethacrynic acid. Its exact mode of action is not clearly
defined. Renal vascular resistance decreases, and renal blood
flow may increase during drug administration. It inhibits reab-
sorption of sodium and chloride primarily in Loop of Henle
and also in proximal and distal renal tubules. It is reportedly
less ototoxic than ethacrynic acid. Its venodilating effect
reduces cardiac preload, which decreases the cardiac workload.

**Emergency Uses:** To treat acute pulmonary edema and con-
gestive heart failure. *Adult dose:* 40–120 mg slow IV. *Pediatric
dose:* 1 mg/kg slow IV.

**Pharmacokinetics**

*Absorption:* Onset of vasodilation is 5–10 min, diuresis is 5–30
min. Peak vasodilatory effect in 30 min, peak diuresis in 20–60
min. Vasodilatory duration is less than 2 hr, diuresis duration is
6 hr; half-life is 30 min.

*Metabolism:* Small amount metabolized in liver.

*Elimination:* Rapidly excreted in urine; 80% of IV dose excret-
ed within 24 hr; excreted in breast milk.

**Contraindications and Precautions:** Furosemide is con-
traindicated in patients with a history of hypersensitivity to
furosemide or sulfonamides; increasing oliguria, anuria, fluid
and electrolyte depletion states; hepatic coma; pregnancy (cate-
gory C). Use with caution in infants, elderly patients; hepatic
cirrhosis, nephrotic syndrome; cardiogenic shock associated
with acute MI, history of gout; patients receiving digitalis gly-
cosides or potassium-depleting steroids.

**Adverse/Side Effects:** CV: Postural hypotension, dizziness
with excessive diuresis, acute hypotensive episodes, circulato-
ry collapse • Fluid and electrolyte imbalance: Hypovolemia,
derhydration, hyponatremia, hypokalemia, hypochlorema
metabolic alkalosis, hypomagnesemia, hypocalcemia (tetany) •
GI: Nausea, vomiting, oral and gastric burning, anorexia, diar-
rhea, constipation, abdominal cramping, acute pancreatitis,
jaundice • GU: Allergic interstitial nephritis, irreversible renal
failure, urinary frequency • Hematologic: Anemia, leukopenia, thrombocytopenic purpura • Rare: aplastic anemia, agranulocytosis • Otoxicity: Tinnitus, vertigo, feeling of fullness in ears, hearing loss (rarely permanent) • Skin: Pruritus, urticaria, exfoliative dermatitis, purpura, photosensitivity • Other: Hyperglycemia, glycosuria, elevated BUN, hyperuricemia; increased perspiration; paresthesias; blurred vision, muscle spasms, weakness; thrombophlebitis, pain at IM injection site.

**Interactions:** Other diuretics enhance the diuretic effects of furosemide. There is an increased risk of digoxin toxicity because of hypokalemia. Nondepolarizing neuromuscular blocking agents (e.g., tubocurarine) prolong neuromuscular blockage. Corticosteroids can potentiate hypokalemia. Furosemide decreases lithium elimination and increases its toxicity; it blunts the hypoglycemic effects of insulin. NSAIDs may attenuate diuretic effects.

**Prehospital Considerations**
- Protect syringes from light once they are removed from package.
- IV administration: IV furosemide may be given by direct IV undiluted at a rate of 20 mg or a fraction thereof over 1 min. With high doses a rate of 4 mg/min is recommended to decrease risk of ototoxicity.
- IV administration to neonates, infants, children: Verify correct IV concentration and rate of infusion/injection with physician.
- Infusion solutions in which furosemide has been mixed should be used within 24 hr.
- Patients receiving the drug parenterally should be observed carefully, and BP and vital signs closely monitored. Sudden death from cardiac arrest has been reported.
- Close observation of the elderly patient is particularly essential during period of brisk diuresis. Sudden alteration in fluid
and electrolyte balance may precipitate significant adverse reactions. Report these symptoms to physician.

- Furosemide should not be administered through the same IV line as amrinone/inamrinone as it will cause a precipitate to form in the IV line.

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**GLUCAGON**

**Class:** Hormone  
**Trade Name:** GlucaGen

**Therapeutic Actions/Pharmacodynamics:** Glucagon is a natural polypeptide hormone produced by alpha cells of the Islets of Langerhans in the pancreas. When released it causes a breakdown of stored glycogen to glucose and inhibits the synthesis of glycogen from glucose. Both actions increase the blood levels of glucose. Given via the intramuscular route, it is a useful drug in hypoglycemia when IV access is unsuccessful. Glucagon also increases heart rate and myocardial contractility, and improves AV conduction in a manner similar to that produced by catecholamines. Its actions are independent of beta blockade.

**Emergency Uses:** To increase blood glucose levels in hypoglycemia without IV access. *Adult dose:* 1.0 mg IM/SC, may repeat every 5–20 min. *Pediatric dose:* Less than 10 kg: 0.1 mg/kg IM/SC/IV. More than 10 kg: 1.0 mg/kg IM/SC/IV. May repeat in 20–30 min.

To reverse the effects of beta blocker overdose. *Adult dose:* 50–150 mg/kg IV over 1 min. *Pediatric dose:* 50–150 mg/kg IV over 1 min.

**Pharmacokinetics**

*Absorption:* Onset 5–20 min; peak effects in 30 min; duration is 1–1.5 hr; half-life is 3–10 min.  
*Metabolism:* Metabolized in liver, plasma, and kidneys.
**Elimination:** Eliminated in urine.

**Contraindications and Precautions:** Glucagon is contraindicated in patients with a hypersensitivity to glucagon or protein compounds. Safe use during pregnancy (category B) and in nursing women not established. Glucagon is only effective if there are glycogen stores in the liver. Use with caution in patients with a history of cardiovascular or renal disease.

**Adverse/Side Effects:** CNS: Dizziness, headache • CV: Hypotension • GI: Nausea and vomiting • Other: Hypersensitivity reactions, hyperglycemia, hypokalemia.

**Interactions:** None in the emergency setting.

**Prehospital Considerations**
- After reconstitution of dry powder, use solution immediately.
- Glucagon will form a precipitate in saline solutions and solutions with pH of 3–9.5 (pH of glucagon is 2.5–3). Glucagon should be considered incompatible in syringe with any other drug.
- Patient usually awakens from (diabetic) hypoglycemic coma 5–20 min after glucagon injection. As soon as possible after patient regains consciousness, PO carbohydrate should be given.
- After recovery from hypoglycemic reaction, symptoms such as headache, nausea, and weakness may persist.
- Most emergency departments and EMS services do not carry enough glucagon for a severe adult beta blocker overdose. A kit with an adequate quantity of the drug could be kept with a supervisor unit in case it is needed.

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**HALOPERIDOL**

**Class:** Antipsychotic

**Trade Names:** Haldol, Peridol (Can), Serenace (Aus)

**Therapeutic Actions/Pharmacodynamics:** Haloperidol is a potent, long-acting antipsychotic agent with pharmacologic
actions similar to those of the phenothiazines but with a higher incidence of extrapyramidal effects and less hypotensive and relatively low sedative activity. While its exact mechanism is unclear, it appears to block the dopamine receptors in the brain associated with mood and behavior. It exerts strong antiemetic effects and impairs central thermoregulation. It also produces weak central anticholinergic effects and transient orthostatic hypotension.

**Emergency Use:** To manage acute psychotic disorders. *Adult dose:* 2–5 mg IM. *Pediatric dose:* 0.05–0.15 mg/kg/day PO in 2–3 divided doses.

**Pharmacokinetics**

*Absorption:* Onset in 30–45 min; peak effects in 10–20 min; half-life is 3–35 hr.

*Distribution:* Distributes mainly to liver with lower concentration in brain, lung, kidney, spleen, heart.

*Metabolism:* Metabolized in liver.

*Elimination:* 40% excreted in urine within 5 days; 15% eliminated in feces; excreted in breast milk.

**Contraindications and Precautions:** Haloperidol is contraindicated in Parkinson’s disease, parkinsonism, seizure disorders, coma; alcoholism; severe mental depression, CNS depression; thyrotoxicosis. Safe use during pregnancy (category C), in nursing mothers, and in children under 3 yr not established. Do not administer haloperidol if other sedatives have been given. Use with caution in elderly or debilitated patients or those with urinary retention, glaucoma, severe cardiovascular disorders; patients receiving anticonvulsant, anticoagulant, or lithium therapy.

**Adverse/Side Effects:** CNS: Extrapyramidal reactions: parkinsonism symptoms, dystonia, akathisia, tardive dyskinesia (after long-term use); insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, mental depression, lethargy, fatigue, weakness, tremor, ataxia, headache, confusion, vertigo; neuroleptic
malignant syndrome, hyperthermia, grand mal seizures, exacerbation of psychotic symptoms • CV: Tachycardia, ECG changes, hypotension, hypertension (with overdosage) • Endocrine: Menstrual irregularities, galactorrhea, lactation, gynecomastia, impotence, increased libido, hyponatremia, hyperglycemia, hypoglycemia • Eyes: Blurred vision • Hematologic: Mild transient leukopenia, agranulocytosis (rare) • GI: Dry mouth, anorexia, nausea, vomiting, constipation, diarrhea, hypersalivation • GU: Urinary retention, priapism • Respiratory: Laryngospasm, bronchospasm, increased depth of respiration, bronchopneumonia, respiratory depression • Skin: Diaphoresis, maculopapular and acneiform rash, photosensitivity • Other: Cholestatic jaundice, variations in liver function tests, decreased serum cholesterol.

**Interactions:** CNS depressants, opiates, and alcohol may increase CNS depression. Haloperidol may antagonize the activity of oral anticoagulants. Anticholinergics may increase intraocular pressure. Methyldopa may precipitate dementia.

**Prehospital Considerations**
- Haloperidol should be administered by deep IM injection into a large muscle. Do not exceed 3 mL per injection site.
- Have patient recumbent at time of parenteral administration and for about 1 hr after injection. Assess for orthostatic hypotension.
- Dosing regimen should be tapered when therapy is to be discontinued. Abrupt termination can initiate extrapyramidal symptoms.
- Targeted symptoms expected to decrease with successful haloperidol treatment include hallucinations, insomnia, hostility, agitation, and delusions.
- Because of long half-life, therapeutic effects are slow to
develop in early therapy or when established dosing regimen is changed.

- Therapeutic window effect (point at which increased dose or concentration actually decreases therapeutic response) may occur after long period of high doses. Close observation is imperative when doses are changed.
- In emergency situations where the patient’s behavior poses an immediate risk to rescuers and bystanders, the initial IM injection may be given through the patient’s clothing in order to minimize needle-stick injuries to rescuers.
- Extrapyramidal (EPS) or dystonic reactions are common with Haldol. Parenteral diphenhydramine (Benadryl) or benztropine (Cogentin) should be readily available.

HEPARIN

Class: Anticoagulant
Trade Names: Hepalean (Can), Heparin, Liquaemin Sodium, Uniparen (Aus)

Therapeutic Actions/Pharmacodynamics: Heparin is a rapid-onset anticoagulant prepared from bovine lung tissue or porcine intestinal mucosa. It exerts direct effect on blood coagulation (clotting) by enhancing the inhibitory actions of antithrombin III (heparin cofactor) on several factors essential to normal blood clotting, thereby blocking the conversion of prothrombin to thrombin and fibrinogen to fibrin. Heparin does not lyse already existing thrombi but may prevent their extension and propagation. It also inhibits formation on new clots.

Emergency Use: To prevent thrombus formation in acute MI. 

Adult dose: 5000 units IV; then 20,000–40,000 units IV over 24 hr.
Pharmacokinetics

Onset: Immediate onset; peak effect within minutes; duration is 2–6 hr; half-life is 90 min.

Distribution: Does not cross placenta; not distributed into breast milk.

Metabolism: Metabolized in liver and by reticuloendothelial system.

Elimination: Excreted slowly in urine.

Contraindications and Precautions: Heparin is contraindicated in patients with a history of hypersensitivity to the drug, active bleeding, and bleeding tendencies (hemophilia, purpura, thrombocytopenia). Use with caution in alcoholism; history of atopy or allergy (asthma, hives, hay fever, eczema); during menstruation, pregnancy (category C), especially the last trimester, and immediate postpartum period; patients with indwelling catheters; the elderly; patients in hazardous occupations; cerebral embolism.

Adverse/Side Effects: CNS: Numbness and tingling of hands and feet, headache • CV: Spontaneous bleeding, cyanosis and pains in arms or legs (vasospasm), hypertension, chest pain • Respiratory: Nasal congestion, bronchospasm • Skin: Injection site reactions: pain, itching, ecchymoses, tissue irritation and sloughing, urticaria, pruritus, skin rashes, itching and burning sensations of feet.

Interactions: Aspirin and other NSAIDs increase risk of bleeding. Nitroglycerin IV may decrease anticoagulant activity. Protamine antagonizes effects of heparin.

Prehospital Considerations

• A single dose of IV heparin (adult 5000 U, child 50 U/kg) may be given undiluted by direct IV injection over 60 seconds.
• IV heparin may be added to NS, D5W, or Ringer’s for injection and infused intermittently or continuously. When
heparin is added to an infusion solution, invert container at least 6 times to ensure adequate mixing.

- Continuous IV infusion of heparin requires a constant infusion pump.
- Patients vary widely in their reaction to heparin. The risk of hemorrhage appears to be greatest in women, all patients over 60 yr, and patients with liver disease or renal insufficiency.
- Monitor vital signs. Report fever, drop in BP, rapid pulse, and other signs of hemorrhage.
- A dilute heparin solution may be used to flush IV catheters (heparin locks) to prevent clotting.

HYDRALAZINE

**Class:** Antihypertensive

**Trade Names:** Alphapress (Aus), Apresoline, Novo-Hylazin (Can), Supres (Can)

**Therapeutic Actions/Pharmacodynamics:** Hydralazine reduces blood pressure mainly by direct effect on vascular smooth muscles of arterial-resistance vessels, resulting in vasodilation. It has little effect on venous-capacitance vessels. The diastolic response is often greater than systolic. Vasodilation reduces peripheral vascular resistance and substantially improves cardiac output, and renal and cerebral blood flow. Its hypotensive effects may be limited by sympathetic reflexes, which increase heart rate, stroke volume, and cardiac output. The postural hypotensive effect is reportedly less than that produced by sodium nitroprusside.

**Emergency Uses:** To reduce blood pressure in hypertensive crisis and preeclampsia. *Adult dose:* 20–40 mg IV/IM. May be repeated in 4–6 hr. *Pediatric dose:* 0.1–0.5 mg/kg/day IV/IM.
Pharmacokinetics

**Absorption:** Onset is 5–15 min IV, 10–40 min IM; peak effects in less than 80 min; duration is 2–6 hr; half-life is 2–8 hr.

**Distribution:** Crosses placenta; distributed into breast milk.

**Metabolism:** Metabolized in intestinal wall and liver.

**Elimination:** 90% rapidly excreted in urine; 10% excreted in feces.

Contraindications and Precautions: Hydralazine is contraindicated in patients with a hypersensitivity to the drug, coronary artery disease, or mitral valvular rheumatic heart disease, MI, tachycardia SLE. Safe use during pregnancy (category C) or in nursing mothers not established. Use with caution in patients with cerebrovascular accident, advanced renal impairment, and those taking MAO inhibitors.

Adverse/Side Effects: CNS: Headache, dizziness, tremors • CV: Palpitation, angina, tachycardia, flushing, paradoxical pressor response, dysrhythmia, shock • Eye: Lacrimation, conjunctivitis • GI: Anorexia, nausea, vomiting, diarrhea, constipation, abdominal pain, paralytic ileus • GU: Difficulty in urination, glomerulonephritis • Hematologic: Decreased hematocrit and hemoglobin, anemia • Hypersensitivity: Rash, urticaria, pruritus, fever, chills, arthralgia, eosinophilia, cholangitis, hepatitis, obstructive jaundice • Other: Nasal congestion, muscle cramps, edema.

Interactions: Beta blockers and other antihypertensive agents compound the hypotensive effects.

Prehospital Considerations

- Administer undiluted solution by direct IV. Give each 10 mg or fraction thereof over 1 min. Do not add hydralazine to IV solutions.
- Discontinuation of hydralazine should be accomplished gradually to avoid sudden rise in BP and acute heart failure. Patients should be informed of the dangers of abrupt withdrawal.
• Store at 15–30C (59–86F) in tight, light-resistant containers unless otherwise directed. Avoid freezing.
• Closely monitor BP and heart rate. Check every 5 min until it is stabilized at desired level, then every 15 min thereafter throughout hypertensive crisis.

HYDROCORTISONE

Class: Steroid
Trade Name: Solu-Cortef

Therapeutic Actions/Pharmacodynamics: Hydrocortisone is a short-acting synthetic steroid with both glucocorticoid and mineralocorticoid properties that affect nearly all systems of the body. By inhibiting the formation, storage, and release of histamine from mast cells, it reduces the effects of an allergic response. It also increases the body’s response to circulating catecholamines.

Emergency Uses: To reduce inflammation during an allergic reaction, severe anaphylaxis, asthma, or COPD; to treat urticaria. Adult dose: 40–250 mg IV/IM. Pediatric dose: 4–8 mg/kg/IM/IV.

Pharmacokinetics
Absorption: Onset is immediate; peak effect in 4–8 hr; duration is 1–1.5 days; half-life is 90 min.
Distribution: Distributed primarily to muscles, liver, skin, intestines, kidneys; crosses placenta.
Metabolism: Metabolized in the liver.
Elimination: Metabolites excreted in urine; excreted in breast milk.

Contraindications and Precautions: Hydrocortisone is contraindicated in patients with hypersensitivity to glucocorticoids. In the treatment of anaphylaxis, there are no absolute contraindications. In the prehospital phase of care, give only a sin-
gle bolus. Long-term steroid therapy can cause gastrointestinal bleeding, prolonged wound healing, and suppression of the adrenocortical steroids.

**Adverse/Side Effects:** CNS: Vertigo, headache, nystagmus, ataxia (rare), increased intracranial pressure with papilledema (usually after discontinuation of medication), mental disturbances, aggravation of preexisting psychiatric conditions, insomnia • CV: Syncopeal episodes, thrombophlebitis, thromboembolism or fat embolism, palpitation, tachycardia, necrotizing angiitis • Endocrine: Decreased glucose tolerance; hyperglycemia, manifestations of latent diabetes mellitus; hypocorticism; amenorrhea and other menstrual difficulties • Eye: Posterior subcapsular cataracts (especially in children), glaucoma, exophthalmos, increased intraocular pressure with optic nerve damage, perforation of the globe, fungal infection of the cornea, decreased or blurred vision • Fluid and electrolyte disturbances: Hypocalcemia; sodium and fluid retention; hypokalemia and hypokalemic alkalosis; CHF, hypertension • GI: Nausea, increased appetite, ulcerative esophagitis, pancreatitis, abdominal distention, peptic ulcer with perforation and hemorrhage, melena • Hematologic: Thrombocytopenia • Musculoskeletal (long-term use): Osteoporosis, compression fractures, muscle wasting and weakness, tendon rupture, aseptic necrosis of femoral and humeral heads • Skin: Skin thinning and atrophy, acne, impaired wound healing; petechiae, ecchymosis, easy bruising; suppression of skin test reaction; hypopigmentation or hyperpigmentation, hirsutism, acneiform eruptions, subcutaneous fat atrophy; allergic dermatitis, urticaria, angioneurotic edema, increased sweating • Other: Hypersensitivity or anaphylactoid reactions; aggravation or masking of infections; malaise, weight gain, obesity; decreased serum concentration of vitamins A and C; urinary frequency and urgency, enuresis • Overdose: Anxiety, mental confusion, depression, hyperglycemia, hypokalemia, hypernatremia, poly-
cythemia, hypertension, edema, GI cramping or bleeding, ecchymoses • With parenteral therapy: IV site: Pain, irritation, necrosis, atrophy, sterile abscess; Charcot-like arthropathy following intra-articular use; burning and tingling in perineal area (after IV injection).

**Interactions:** Barbiturates, phenytoin, and rifampin may increase hepatic metabolism, thus decreasing cortisone levels. Estrogens may potentiate the effects of hydrocortisone. NSAIDs compound its ulcerogenic effects. Diuretics, amphotericin B exacerbate hypokalemia; anticholinesterase agents (e.g., neostigmine) may produce severe weakness; immune response to vaccines and toxoids may be decreased.

**Prehospital Considerations**
- Inject IM preparation deep into gluteal muscle.
- IV administration: IV hydrocortisone may be given by direct IV undiluted or diluted in NS or D5W. Administer at a rate of 25 mg or a fraction thereof over 1 min.
- IV administration to infants, children: Verify correct IV concentration and rate of infusion/injection with physician.
- Solutions that have been diluted for IV infusion should be administered within 24 hr of dilution.
- The elderly and the patient with low serum albumin are especially susceptible to adverse or side effects.

**HYDROXYZINE**

**Class:** Antihistamine

**Trade Names:** Anxanil, Atarax, Hydroxacin, Multipax (Can), Quiess, Vistaject, Vistaril

**Therapeutic Actions/Pharmacodynamics:** Hydroxyzine is an antihistamine with CNS depressive, sedative, anticholinergic, antiemetic, and bronchodilator properties. Its tranquilizing (ataractic) effect is produced primarily by depression of hypo-
thalamus and brain-stem reticular formation, rather than cortical areas. It is used frequently in the emergency setting, often in combination with analgesics.

**Emergency Uses:** To manage an acute anxiety attack. *Adult dose:* 50–100 mg deep IM. *Pediatric dose:* 1 mg/kg IM.

To control nausea and vomiting. *Adult dose:* 25–50 mg deep IM. *Pediatric dose:* 1 mg/kg deep IM.

**Pharmacokinetics**

*Absorption:* Onset is 15–30 min; duration is 4–6 hr.

*Distribution:* Not known if it crosses placenta or is distributed into breast milk. *Metabolism:* Metabolized in liver. *Elimination:* Probably excreted in bile.

**Contraindications and Precautions:** Hydroxyzine is contraindicated in patients with known hypersensitivity to the drug. Safe use during pregnancy (category C) or in nursing mothers not established. Use with caution in the elderly.

**Adverse/Side Effects:** CNS: Drowsiness (usually transitory), sedation, dizziness, headache • CV: Chest tightness, hypotension • Respiratory: Dyspnea, wheezing • Skin: Injection site reactions, urticaria, erythematous macular eruptions • Other: Dry mouth, involuntary motor activity, erythema multiforme, phlebitis, hemolysis, thrombosis, digital gangrene from inadvertent IV or intra-arterial injection.

**Interactions:** Alcohol and CNS depressants add to CNS depression; tricyclic antidepressants and other anticholinergics have additive anticholinergic effects; may inhibit pressor effects of epinephrine.

**Prehospital Considerations**

- IM administration should be made deep into body of a relatively large muscle. The Z-track technique of injection is recommended to prevent SC infiltration.
- Recommended IM site: In adult, the gluteus maximus or vastus lateralis; in children, the vastus lateralis.
• Protect hydroxyzine from light. Store at 15–30°C (59–86°F) unless otherwise specified.
• Forewarn about the possibility of drowsiness and dizziness, and caution against hazardous activities until reaction to drug is known.
• Alcohol and hydroxyzine should not be taken at the same time.
• Care must be taken to NEVER give hydroxyzine intravenously.

IBUPROFEN

Class: Nonsteroidal anti-inflammatory drug (NSAID)
Trade Names: Actiprofen (Aus), Advil, Amersol (Can), Brufen (Aus), Children’s Motrin, Excedrin IB, Genpril, Haltran, Ibuprin, Junior Strength Motrin Caplets, Medipren, Motrin, Nuprin, Nurofen (Aus), Pamprin-IB, Pediaprofen, Rafen (Aus), Rufen, Trendar

Therapeutic Action/Pharmacodynamics: Ibuprofen is the prototype NSAID with significant antipyretic and analgesic properties. It blocks prostaglandin synthesis, inhibits platelet aggregation, and prolongs bleeding time, but does not affect prothrombin or whole blood clotting times. Cross-sensitivity with aspirin and other NSAIDs has been reported.

Emergency Uses: To reduce fever; to temporarily relieve mild to moderate pain. Adult dose: 200–400 mg PO every 4–6 hr up to 1200 mg/day. Pediatric dose: 5–10 mg/kg PO every 4–6 hr up to 40 mg/kg/day.

Pharmacokinetics
Absorption: 80% absorbed from GI tract; onset is 1 hr.
Antipyretic effect: peak is 1–2 hr; duration is 6–8 hr; half-life is 2–4 hr.
Metabolism: Metabolized in liver.
Elimination: Excreted primarily in urine; some biliary excretion.

Contraindications and Precautions: Ibuprofen is contraindicated in patients in whom urticaria, severe rhinitis, bronchospasm, angioedema, nasal polyps are precipitated by aspirin or other NSAIDs; active peptic ulcer, bleeding abnormalities. Use with caution in patients with hypertension, history of GI ulceration, impaired hepatic or renal function, chronic renal failure, cardiac decompensation.

Adverse/Side Effects: CNS: Headache, dizziness, light-headedness, anxiety, emotional lability, fatigue, malaise, drowsiness, anxiety, confusion, depression, aseptic meningitis • CV: Hypertension, palpitations, congestive heart failure (patient with marginal cardiac function); peripheral edema • Eye/ear: Blurred vision, decreased visual acuity, changes in color vision, nystagmus, visual-field defects; tinnitus, impaired hearing • GI: Dry mouth, gingival ulcerations, dyspepsia, heartburn, nausea, vomiting, anorexia, diarrhea, constipation, bloating, flatulence, epigastric or abdominal discomfort or pain, GI ulceration, occult blood loss • Hematologic: Rise in bleeding time • Renal: Acute renal failure, polyuria, azotemia, cystitis, hematuria, nephrotoxicity, decreased creatinine clearance • Skin: Maculopapular and vesicobullous skin eruptions, erythema multiforme, pruritus, rectal itching, acne • Other: Fluid retention with edema, toxic hepatitis, hypersensitivity reactions, anaphylaxis, bronchospasm, angioedema.

Interactions: Ibuprofen may prolong bleeding time when used with anticoagulants. Also may increase lithium and methotrexate toxicity.

Prehospital Considerations
• Give on an empty stomach, 1 hr before or 2 hr after meals.
• If GI intolerance occurs, ibuprofen may be taken with meals or milk.
• Tablet may be crushed if patient is unable to swallow it whole and mixed with food or liquid before swallowing.
• Patients with history of cardiac decompensation should be observed closely for evidence of fluid retention and edema.
• Monitor for GI distress and signs of GI bleeding.
• Symptoms of acute toxicity in children are apnea, cyanosis, response only to painful stimuli, dizziness, and nystagmus.

IBUTILIDE

Class: Antidysrhythmic
Trade Name: Corvert

Therapeutic Actions/Pharmacodynamics: Ibutilide is a short-acting class III antidysrhythmic agent. It prolongs the cardiac action potential and increases both atrial and ventricular refractoriness (i.e., class III antidysrhythmic electrophysiologic effects). It is recommended for acute pharmacologic conversion of atrial flutter or atrial fibrillation or as an adjunct to electrical cardioversion in patients in whom electrical cardioversion alone has been ineffective. Its short duration of action makes it less effective than other antidysrhythmic agents for maintaining sinus rhythm once restored.

Emergency Uses: To convert atrial flutter or atrial fibrillation of relatively short duration. Adult dose: In patients weighing more than 60 kg, 1 mg over 10 min IV. In patients weighing less than 60 kg, 0.01 mg/kg IV. May repeat in 10 min if inadequate response.

Pharmacokinetics
Absorption: Onset is 30 min; half-life is 6 hr (range 2–21 hr).
Metabolism: Metabolized in liver.
Elimination: 82% excreted in urine, 19% in feces.
Contraindications and Precautions: Ibutilide is contraindicated in patients with a hypersensitivity to ibutilide, hypokalemia, and hypomagnesemia. Use with caution in patients with history of CHF, low ejection fraction, recent MI, prolonged QT intervals, liver disease, cardiovascular disorder other than atrial dysrhythmias, other drugs that prolong QT interval, lactation. Safety and effectiveness in children under 18 yr not established.

Adverse/Side Effects: CNS: Headache • CV: Proarrhythmic effects (sustained and nonsustained polymorphic ventricular tachycardia), torsade de pointes, AV block, bundle branch block, ventricular extrasystoles, hypotension, postural hypotension, bradycardia, tachycardia, palpitations, prolonged QT segment • GI: Nausea.

Interactions: Increased potential for proarrhythmic effects when administered with astemizole, phenothiazines, tricyclic antidepressants, and terfenadine. Amiodarone, disopyramide, quinidine, procainamide, and sotalol may cause prolonged refractoriness if given within 4 hr of ibutilide.

Prehospital Considerations
• IV preparation and administration: Contents of 1-mg vial may be given undiluted or diluted in 50 mL of 0.9% NaCl or D5W to a concentration of 0.017 mg/mL. Infuse over 10 min.
• Stop IV infusion as soon as presenting dysrhythmia is terminated or with appearance of ventricular tachycardia or marked prolongation of QT or QTc.
• Class Ia and other class III antidysrhythmic drugs should not be given concurrently or within 4 hr of ibutilide.
• Observe with continuous ECG, BP, and HR monitoring during and for 4–6 hr after infusion or until QTc has returned to baseline. Monitor for longer periods with liver dysfunction or if proarrhythmic activity is observed.
• Conversion to normal sinus rhythm normally occurs within 30 min of initiation of infusion.
INSULIN

Class: Hormone

Trade Names: Actrapid (Aus), Humulin, Hypurin (Aus), Iletin, Novolin, Regular Insulin, Regular Purified Pork Insulin, Velosulin (Can)

Therapeutic Actions/Pharmacodynamics: Insulin is a protein secreted by the beta cells of the Islets of Langerhans in the pancreas. Insulin is responsible for promoting the uptake of glucose by the cells. When administered, it is distributed throughout the body. It combines with insulin receptors present on the cell membranes. This promotes glucose entry into the cell. In diabetics, where insulin secretion is diminished, supplemental insulin must be obtained by injection. Older forms of insulin are derived from animals (bovine and porcine). However, animal insulin is not identical to human insulin. Because of this, many patients will develop antibodies to animal insulin rendering it less effective. Human insulin can be manufactured through genetic engineering (recombinant DNA technology). Genetically engineered insulin (Humulin, Novolin) is chemically identical to the insulin hormone secreted by the pancreas. Patients do not develop antibodies to human insulin as they do to animal insulin.

Emergency Uses: To reverse effects of hyperglycemia and diabetic coma. Adult dose: 5–10 units IV/IM/SC of regular insulin loading dose. Maintenance dose is based on blood glucose levels. Pediatric dose: 2–4 units IV/IM/SC of regular insulin loading dose. Maintenance dose is based on blood glucose levels.

Pharmacokinetics

Absorption: Rapidly absorbed from IM and SC injections. Onset is 0.5–1.0 hr; peak effect in 2–3 hr; duration is 5–7 hr; half-life is up to 13 hr.
**Distribution:** Throughout extracellular fluids.

**Metabolism:** Metabolized primarily in liver with some metabolism in kidneys. **Elimination:** less than 2% excreted in urine.

**Contraindications and Precautions:** Seldom given in the prehospital setting, insulin is contraindicated in patients with hypersensitivity to insulin animal protein and in hypoglycemia. Cautious use in pregnancy (category C).

**Adverse/Side Effects:** Hypersensitivity (usually occurs when insulin is at peak action point): Localized allergic reactions at injection site; generalized urticaria or bullae, lymphadenopathy, anaphylaxis (rare) • Hypoglycemia (hyperinsulinism): Profuse sweating, hunger, headache, nausea, tremulousness, tremors, palpitation, tachycardia, weakness, fatigue, nystagmus, circumoral pallor; numb mouth, tongue, and other paresthesias; visual disturbances (diplopia, blurred vision, mydriasis), staring expression, confusion, personality changes, ataxia, incoherent speech, apprehension, irritability, inability to concentrate, personality changes, uncontrolled yawning, loss of consciousness, delirium, hypothermia, convulsions, Babinski reflex, coma • Other: Posthypoglycemia or rebound hyperglycemia • Overdosage: psychic disturbances, (i.e., aphasia, personality changes, maniacal behavior).

**Interactions:** Alcohol, anabolic steroids, MAO inhibitors, guanethidine, salicylates may potentiate hypoglycemic effects. Dextrothyroxine, corticosteroids, epinephrine may antagonize hypoglycemic effects. Furosemide, thiazide diuretics increase serum glucose levels. Propranolol and other beta blockers may mask symptoms of hypoglycemic reaction.

**Prehospital Considerations**

**SC Administration**

- Always use an insulin syringe.
- Regular insulin is generally administered 30 min before a meal.
- Avoid injection of cold insulin; it can lead to lipodystrophy, reduced rate of absorption, and local reactions.
Commonly used injection sites are upper arms, thighs, abdomen (avoid area over urinary bladder and 2 in. [5 cm] around navel), buttocks, and upper back (if fat is loose enough to pick up).

**IV Administration**

- Regular insulin may be given by direct IV undiluted. Administer 10 U or a fraction thereof over 1 min. When insulin is administered by continuous infusion, rate must be ordered by physician.
- Regular insulin may be adsorbed into the container or tubing when added to an IV infusion solution. Amount lost is variable and depends on concentration of insulin, infusion system, contact duration, and flow rate. Monitor patient response closely.
- Insulin is stable at room temperature up to 1 month. Avoid exposure to direct sunlight or to temperature extremes (safe range is wide: 5−38°C [40−100°F]). Refrigerate but do not freeze stock supply. Insulin tolerates temperatures above 38°C with less harm than freezing.
- Frequency of blood glucose monitoring is determined by the type of insulin regimen and health status of the patient.
- Monitor for hypoglycemia at time of peak action of insulin. Onset of hypoglycemia (blood sugar: 50−40 mg/dL) may be rapid and sudden.
- During treatment for ketoacidosis with IV insulin, check BP and blood glucose often.

**IPECAC SYRUP**

**Class:** Emetic

**Trade Name:** Syrup of Ipecac

**Therapeutic Action/Pharmacodynamics:** Ipecac is a potent and effective emetic used in the management of poisonings
when vomiting is indicated. It acts as a local irritant on the gastric mucosa to induce vomiting and centrally on chemoreceptor trigger zone (CTZ) in the medulla to induce vomiting. 

**Emergency Uses:** To induce vomiting of unabsorbed ingested poisons. *Adult dose:* 30 mL PO followed by 1–2 glasses of water; may repeat once in 20 min if necessary. *Pediatric dose:* 15 mL followed by 1–2 glasses of water; may repeat once in 20 min if necessary.

**Pharmacokinetics**

*Absorption:* Onset is 15–30 min, duration is 25 min.

*Elimination:* Metabolite can be detected in urine up to 60 days after excessive doses.

**Contraindications and Precautions:** Ipecac is contraindicated in any patient with an altered mental status or depressed gag reflex because of the risk of aspiration. Vomiting is also contraindicated in patients who have ingested caustic substances such as petroleum products, strong acids or alkalis, corrosives, and fast-acting CNS depressants. Avoid using ipecac when the ingested drug is an antiemetic, especially a phenothiazine. Because of the risk of aspiration associated with vomiting, the trend in the management of acute poisonings is to use activated charcoal.

**Adverse/Side Effects:** CNS: Convulsions, coma, sensory disturbances • CV: Cardiomyopathy, cardiotoxicity, cardiac dysrhythmias, atrial fibrillation, tachycardia, chest pain, hypotension, fatal myocarditis • Respiratory: Dyspnea • GI: Diarrhea, mild GI upset, vomiting, gastroenteritis, bloody diarrhea, stomach cramps, tremor.

**Interactions:** Do not administer syrup of ipecac with activated charcoal as the activated charcoal will nullify the effects of ipecac.

**Prehospital Considerations**

- Emetic effect occurs in 15–30 min and continues for 20–25 min. If vomiting does not occur in 20–30 min, the dose may be repeated once.
• If vomiting does not occur within 15–20 min after a second
dose, contact physician immediately. Dosage should be recov-
ered by gastric lavage and activated charcoal if necessary.
• The prehospital use of ipecac should be very limited to cases
where the benefit of emesis outweighs the potential risks.

IPRATROPIUM

Class: Parasympatholytic bronchodilator
Trade Name: Atrovent

Therapeutic Actions/Pharmacodynamics: Ipratropium is an
anticholinergic agent, chemically related to atropine. Given in a
nebulized form, it acts directly on the smooth muscle of the
bronchial tree by inhibiting acetylcholine at receptor sites. By
blocking parasympathetic action, it dilates the bronchial
smooth muscle and decreases secretions. It also abolishes the
vagally mediated reflex bronchospasm caused by inhaled irri-
tants such as smoke, dust, cold air, and by a range of inflam-
matory mediators (e.g., histamine).

Emergency Uses: To relieve bronchospasm in patients with
reversible obstructive airway disease (asthma, chronic bronchi-
tis, emphysema) and acute attacks of bronchospasm. Adult
dose: 500 µg in 2.5–3.0 mL via nebulizer or 2 inhalations from
a metered dose inhaler (MDI). Pediatric dose: 125–250 µg in
2.5–3.0 mL via nebulizer, or 1–2 inhalations from MDI.

Pharmacokinetics
Absorption: 10% of inhaled dose reaches lower airway;
approximately 0.5% of dose is systemically absorbed; peak
effect in 1.5–2.0 hr; duration is 4–6 hr; half-life is 1.5–2 hr.
Elimination: 48% of dose excreted in feces; less than 5%
excreted in urine.

Contraindications and Precautions: Ipratropium is con-
traindicated in patients with hypersensitivity to atropine or its
derivatives. It should not be used as the primary treatment for acute episodes of bronchospasm. Cautious use in pregnancy (category B) and nursing mothers.

Adverse/Side Effects: Eye: Blurred vision (especially if sprayed into eye), difficulty in accommodation, acute eye pain, worsening of narrow-angle glaucoma • GI: Bitter taste, dry oropharyngeal membranes • With higher doses: nausea, constipation • Respiratory: Cough, hoarseness, exacerbation of symptoms, drying of bronchial secretions, mucosal ulcers, epistaxis, nasal dryness • Other: Rash, hives, urinary retention, headache.

Prehospital Considerations
• Monitor respiratory status; auscultate lungs before and after inhalation.
• Treatment failure (exacerbation of respiratory symptoms) should be reported to physician.
• Ipratropium is almost always administered in conjunction with a beta-adrenergic agent such as albuterol.

ISOETHARINE

Class: Sympathomimetic bronchodilator
Trade Name: Bronkosol

Therapeutic Action/Pharmacodynamics: Isoetharine is a synthetic sympathomimetic stimulant with relatively rapid onset and long duration of action. The prime action of beta-adrenergic drugs is to stimulate adenyly cyclase, the enzyme which catalyzes the formation of cyclic-3’, 5’-adenosine monophosphate (cyclic AMP) from adenosine triphosphate (ATP). The cyclic AMP causes relaxation of the smooth muscles of the bronchial tree, decreasing airway resistance, facilitating mucus drainage, and increasing vital capacity. It exerts minimal effects on beta1 (heart) or alpha (peripheral vasculature) receptors. In therapeutic doses, isoetharine, by inhibiting
histamine release from mast cells, also reduces the mucus secretion, capillary leaking, and mucosal edema caused by an allergic response in the lungs.

**Emergency Uses:** To relieve bronchospasm in patients with reversible obstructive airway disease (asthma, chronic bronchitis, emphysema) and acute attacks of bronchospasm. *Adult dose:* 1–2 inhalations via metered-dose inhaler; 0.5 mL in 2–3 mL saline via nebulizer; 0.5 mL in 2–3 mL saline via bag-valve-mask (BVM). *Pediatric dose:* 0.01 mL/kg of 1% solution (maximum 0.5 mL) diluted in 2–3 mL normal saline.

**Pharmacodynamics**
*Absorption:* Onset is immediate, peak effect in 5–15 min; duration is 1–4 hr. *Metabolism:* Metabolized in lungs, liver, GI tract, and other tissues.
*Elimination:* Excreted by kidneys.

**Contraindications and Precautions:** Isoetharine is contraindicated in patients with a known hypersensitivity to sympathomimetic amines and to bisulfites; concomitant use with epinephrine or other sympathomimetic amines; patients with pre-existing cardiac dysrhythmias associated with tachycardia. The preservative sodium bisulfite is in the hydrochloride formulation. If patient has a history of allergy to sulfite agents, this product should not be used. Use with caution in elderly patients; hypertension; acute coronary artery disease; CHF; cardiac asthma; hyperthyroidism, diabetes mellitus; tuberculosis; history of seizures.

**Adverse/Side Effects:**
*CNS:* Headache, anxiety, tension, restlessness, insomnia, tremor, weakness, dizziness, excitement.
*CV:* Tachycardia, palpitations, changes in BP, cardiac arrest.
*GI:* Nausea, vomiting.
*Respiratory:* Cough, bronchial irritation, edema.

**Interactions:** Expect additive effects when used with epinephrine, other sympathomimetic bronchodilators. MAO inhibitors
and tricyclic antidepressants may potentiate its action on the vascular system. If your patient is taking beta blockers, the effects of isoetharine will be antagonized.

**Prehospital Considerations**
- When using a metered-dose inhaler, instruct the patient to shake the container, exhale through nose as completely as possible, administer aerosol while inhaling deeply through mouth, and to hold breath about 10 seconds before exhaling slowly. Administer second inhalation 10 min after first.
- Monitor respiratory status. Measure peak flow and auscultate lungs before and after inhalation to determine efficacy of drug in decreasing airway resistance.
- Drug may have shorter duration of action after long-term use. Instruct to report failure to respond to usual dose.
- Advise that tremor is an anticipated side effect.
- Isoetharine inhalation may be alternated with epinephrine or albuterol administration but may not be administered simultaneously because of danger of excessively rapid heartbeat.
- Do not use discolored or precipitated solutions.
- Elderly patients may be especially sensitive to adrenergic drug effects. Monitor cardiac status and report tachycardia and palpitations.
- Consider isoetharine for patients who have been extensively using another beta agonist such as albuterol. Because of tachyphylaxis, patients may experience decreased effectiveness of their usual beta agonist and isoetharine may prove beneficial.

**ISOPROTERENOL**

**Class:** Sympathomimetic  
**Trade Name:** Isuprel  
**Therapeutic Actions/Pharmacodynamics:** Isoproterenol is a
synthetic sympathomimetic agonist with pure \( \beta_1 \) and \( \beta_2 \) adrenergic effects. Drug induced stimulation of \( \beta_1 \)-adrenergic receptors results in increased cardiac output and work by increasing strength of cardiac contraction and, to a slight degree, rate of contraction. It produces a slight increase in systolic BP and decrease in diastolic pressure. Isoproterenol reduces total peripheral resistance and increases venous return to the heart by mobilizing blood from vascular reservoirs. Stimulation of \( \beta_2 \)-adrenoceptors relaxes bronchospasm and, by increasing ciliary motion, facilitates expectoration of pulmonary secretions. It also may dilate the trachea and main bronchi past the resting diameter.

**Emergency Use:** To increase cardiac output by increasing the heart rate in symptomatic bradycardia refractory to atropine when transcutaneous pacing is not available. **Adult dose:** 2–10 \( \mu \)g/min titrated to rate. **Pediatric dose:** 0.1 \( \mu \)g/kg/min titrated to rate. To dilate the bronchial tree in severe status asthmaticus: **Adult dose:** 1–2 inhalations of MDI. **Pediatric dose:** Same as for adult.

**Pharmacokinetics**

**Absorption:** Rapidly absorbed IV; immediate onset.

**Metabolism:** Action terminated by tissue uptake and metabolized by COMT in liver, lungs, and other tissues.

**Elimination:** 40–50% excreted in urine unchanged.

**Contraindications and Precautions:** Because isoproterenol increases preload, myocardial oxygen consumption, and demand, and reduces total peripheral resistance, it is contraindicated in patients in cardiogenic shock. Use with caution in patients with preexisting tachydysrhythmias associated with tachycardia; tachycardia caused by digitalis intoxication, and acute myocardial infarction.

**Adverse/Side Effects:** CNS: Headache, mild tremors, nervousness, anxiety, insomnia, excitement, fatigue • CV: Flushing, palpitations, tachycardia, unstable BP, anginal pain, ventricular
dysrhythmias • Overdosage (especially after excessive use of aerosols): Tachycardia, palpitations, nervousness, nausea, vomiting.

**Interactions:** Epinephrine and other sympathomimetic amines’ effects are increased by isoproterenol and can potentially cause cardiac toxicity. Beta blockers antagonize these effects.

**Prehospital Considerations**
- IV infusion: Dilute 10 mL 1:5000 solution in 500 mL 5% dextrose to produce a 1:250,000 solution.
- Microdrip or constant-infusion pump is recommended to prevent sudden influx of large amounts of drug.
- Infusion rate is generally decreased or infusion may be temporarily discontinued if heart rate exceeds 110 bpm, because of the danger of precipitating dysrhythmias.
- IV administration is regulated by continuous ECG monitoring. Patient must be observed and response to therapy must be monitored continuously.
- Isoproterenol solutions lose potency with standing. Discard if precipitate or discoloration is present.
- Incidence of dysrhythmias is high, particularly when drug is administered IV to patients with cardiogenic shock or ischemic heart disease, digitalized patients, or to those with electrolyte imbalance. Check pulse before and during IV administration. Rate greater than 110 usually indicates need to slow infusion rate or discontinue infusion. Consult physician for guidelines.
- Tolerance to bronchodilating effect and cardiac stimulant effect may develop with prolonged use.
- Rebound bronchospasm may occur when effects of drug end. Once tolerance has developed, continued use can result in serious adverse effects.
- With the advent of transcutaneous pacing, isoproterenol has a very minimal role in modern prehospital care.
KETOROLAC

Class: Nonsteroidal anti-inflammatory drug (NSAID)
Trade Names: Toradol

Therapeutic Actions/Pharmacodynamics: Ketorolac is an injectable NSAID that exhibits analgesic, anti-inflammatory, and antipyretic activity. It works by inhibiting the synthesis of prostaglandins. Ketorolac does not have any known effects on opiate receptors. Unlike narcotics, which act on the central nervous system, ketorolac does not have the sedative properties and is a peripherally acting analgesic. It is the first injectable NSAID to become available in the United States.

Emergency Use: To relieve mild to moderate pain. Adult dose: IV loading dose: 30 mg (15 mg if patient is older than 65 yr, or weighs less than 50 kg). IM: 30–60 mg loading dose. Pediatric dose: Rarely used.

Pharmacokinetics
Absorption: Peak effects in 45–60 min; half-life is 4–6 hr.
Distribution: Distributed into breast milk.
Metabolism: Metabolized in liver.
Elimination: Excreted in urine.

Contraindications and Precautions: Ketorolac is contraindicated in patients with asthma and with hypersensitivity to ketorolac, aspirin, or other NSAIDs. Use with caution in patients with a history of peptic ulcers; impaired renal or hepatic function; elderly.

Adverse/Side Effects: CNS: Drowsiness, dizziness, headache • GI: Nausea, dyspepsia, GI pain, hemorrhage • Other: Edema, sweating, pain at injection site.

Interactions: Ketorolac may increase lithium levels and toxicity. It also may worsen the side effects of aspirin and other NSAIDs when used in conjunction with them. Ketorolac has been found to reduce the diuretic response to furosemide.
Prehospital Considerations  
- When used for pain relief, reduced dosages may be necessary with the elderly because of renal effects of NSAIDs.  
- Inject IM ketorolac slowly and deeply into a large muscle.  
- Injection site pain has been reported in some patients receiving multiple doses. Rotate injection sites.  
- Administer IV bolus dose over at least 15 seconds.  
- Hypovolemia should be corrected prior to administration of ketorolac.  
- Monitor for signs and symptoms of bleeding. Ktorolac decreases platelet aggregation and thus may prolong bleeding time.  
- Monitor for signs and symptoms of GI distress or bleeding including nausea, GI pain, diarrhea, melena, or hematemesis. GI ulceration with perforation can occur anytime during treatment.  
- Patients with a history of cardiac decompensation should be observed closely for evidence of fluid retention and edema.  
- When administering ketorolac intravenously, always use a dose of 30 mg or less. The intramuscular dose is 30–60 mg.

LABETALOL  
Class: Beta blocker  
Trade Names: Normodyne, Presolol (Aus), Trandate  
Therapeutic Actions/Pharmacodynamics: Labetalol is an adrenergic-receptor blocking agent that combines selective alpha activity and nonselective beta-adrenergic blocking actions. Both activities contribute to reduce blood pressure. Alpha blockade results in vasodilation, decreased peripheral resistance, and orthostatic hypotension and only slightly affects cardiac output and coronary artery blood flow. Beta-blocking effects on sinus node, AV node, and ventricular muscle lead to bradycardia,
delay in AV conduction, and depression of cardiac contractility.

Emergency Use: To manage an acute hypertensive crisis. **Adult dose:** 20 mg slow IV with 40–80 mg every 10 minutes as needed up to 300 mg total, or 2 mg/min continuous infusion up to 300 mg total dose. **Pediatric dose:** Safety in children has not been established.

**Pharmacokinetics**

**Absorption:** Onset is 2–5 min IV; peak effects in 5–15 min; duration is 2–4 hr; half-life is 3–8 hr.

**Distribution:** Crosses placenta; distributed into breast milk.

**Metabolism:** Metabolized in liver.

**Elimination:** 60% excreted in urine, 40% in bile.

**Contraindications and Precautions:** Because it is a nonselective beta blocker, labetalol is contraindicated in bronchial asthma. It is also contraindicated in uncontrolled cardiac failure, heart block (greater than first degree), cardiogenic shock, and severe bradycardia. Safe use during pregnancy (category C), in nursing women, and in children not established. Use with caution in patients with nonallergic bronchospastic disease (COPD), well-compensated patients with history of heart failure; pheochromocytoma; impaired hepatic function, jaundice; diabetes mellitus; peripheral vascular disease.

**Adverse/Side Effects:** CNS: Dizziness, fatigue/malaise, headache, tremors, transient paresthesias (especially scalp tingling), hypoesthesia (numbness), mental depression, drowsiness, sleep disturbances, nightmares • CV: Postural hypotension, angina pectoris, palpitation, bradycardia, syncope, pedal or peripheral edema, pulmonary edema, CHF, flushing, cold extremities, dysrhythmias (following IV), paradoxical hypertension (patients with pheochromocytoma) • Eye: Dry eyes, vision disturbances • GI: Nausea, vomiting, dyspepsia, constipation, diarrhea, taste disturbances, cholestasis with or without jaundice, increases in serum transaminases, dry mouth • GU: Acute urinary retention, difficult micturition, impotence, ejacu-
lation failure, loss of libido • Respiratory: Dyspnea, bronchospasm • Skin: Rashes of various types, increased sweating, pruritus • Other: Nasal stuffiness, rhinorrhea, myalgia, muscle cramps, pain at IV injection site.

**Interactions:** Cimetidine may increase the effects of labetalol; beta agonists antagonize effects of labetalol. Other antihypertensive agents may potentiate the effects of labetalol. Do not use with IV calcium channel blockers.

**Prehospital Considerations**
- Patient should be supine when receiving labetalol IV due to orthostatic hypotension. Take BP immediately before administration.
- Drug may be given undiluted by direct IV or further diluted in most IV solutions and administered as a continuous infusion.
- Continuous IV infusion: Rate is adjusted according to BP response. Normal rate is 2 mg/min. Once the desired BP is attained, labetalol is discontinued.
- Controlled infusion pump device is recommended for maintaining accurate flow rate during IV infusion. Usually administered at rate of 2 mg/min.
- Monitor BP and pulse during dosage adjustment period. Standing BP is used often as indicator for making dosage adjustments and for assessing patient’s tolerance of dosage increases. Generally taken after patient stands for 10 min. Clarify with physician.
- After IV administration, (1) monitor BP at 5-min intervals for 30 min; (2) then at 30-min intervals for 2 hr; (3) then hourly for about 6 hr, and as indicated thereafter.
- Supine position should be maintained for at least 3 hr after IV administration. At the end of this time, determine patient’s ability to tolerate elevated and upright positions before allowing ambulation. Manage this slowly.
- Instruct patient to make all position changes slowly and in stages, particularly from recumbent to upright position.
Elderly patients are especially sensitive to hypotensive effects.

- Since labetalol can cause dizziness and light-headedness, advise to avoid potentially hazardous activities until reaction to drug is known.
- Diabetic patients should be closely monitored. Labetalol may mask usual cardiovascular response to acute hypoglycemia, e.g., tachycardia.
- Reassure that most adverse effects (e.g., scalp tingling) are mild, transient, and dose related and occur early in therapy.

LIDOCAINE

**Class:** Antidysrhythmic

**Trade Names:** Xylocaine, Xylocard (Aus, Can)

**Therapeutic Actions/Pharmacodynamics:** Lidocaine has cardiac actions similar to those of procainamide and quinidine but has little effect on myocardial contractility, AV and intraventricular conduction, cardiac output, and systolic arterial pressure in equivalent doses. It exerts antidysrhythmic action (class Ib) by suppressing automaticity in the His-Purkinje system and by elevating electrical stimulation threshold of ventricle during diastole. It is used to raise the threshold for ventricular dysrhythmias and to lower the threshold for defibrillation and cardioversion. Progressive depression of CNS occurs with increasing blood concentration; produces anticonvulsant, sedative, and analgesic effects.

**Emergency Uses:** To convert ventricular dysrhythmias (ventricular fibrillation, ventricular tachycardia) in cardiac arrest to sinus rhythm. *Adult dose:* 1.0–1.5 mg/kg IV. Repeat every 3–5 min as needed up to 3 mg/kg. Following conversion, begin infusion at 2–4 mg/min. *Pediatric dose:* 1 mg/kg IV. Repeat as needed.
needed every 3–5 min up to 3 mg/kg. Following conversion, begin infusion at 20–50 µg/kg/min.
To convert ventricular tachycardia with a pulse to sinus rhythm.  
**Adult dose:** 1.0–1.5 mg/kg slow IV. May repeat at one-half dose every 5–10 min until conversion up to 3 mg/kg. Following conversion, begin infusion at 2–4 mg/min. **Pediatric dose:** 1 mg/kg IV followed by an infusion at 20–50 µg/kg/min.

**Pharmacokinetics**

**Absorption:** Onset in under 3 min; peak effects in 5–7 min; duration is 10–20 min; half-life is 1.5–2.0 hr.

**Distribution:** Crosses blood-brain barrier and placenta; distributed into breast milk.  
**Metabolism:** Metabolized in liver.

**Elimination:** Excreted in urine.

**Contraindications and Precautions:** Lidocaine is contraindicated in patients with a history of hypersensitivity to amide-type local anesthetics, supraventricular dysrhythmias, Stokes-Adams syndrome, untreated sinus bradycardia, severe degrees of sinoatrial, atrioventricular, and intraventricular heart block. Use with caution in patients with liver or renal disease, CHF, marked hypoxia, respiratory depression, hypovolemia, shock; myasthenia gravis; debilitated patients, the elderly; family history of malignant hyperthermia (fulminant hypermetabolism).

**Adverse/Side Effects:** CNS: Drowsiness, dizziness, light-headedness, restlessness, confusion, disorientation, irritability, apprehension, euphoria, wild excitement, numbness of lips or tongue, and other paresthesias including sensations of heat and cold, chest heaviness, difficulty in speaking, difficulty in breathing or swallowing, muscular twitching, tremors, psychosis • With high doses: convulsions, respiratory depression and arrest • CV: (with high doses): Hypotension, bradycardia, conduction disorders including heart block, cardiovascular collapse, cardiac arrest • Ears: Tinnitus, decreased hearing • Eye: Blurred or double vision, impaired color perception • Other: Anorexia, nausea, vomiting, excessive perspiration, soreness at
IM site, local thrombophlebitis (with prolonged IV infusion), hypersensitivity reactions (urticaria, rash, edema, anaphylactoid reactions).

**Interactions:** Barbiturates decrease lidocaine activity. Cimetidine, beta blockers, and quinidine increase the pharmacologic effects of lidocaine. Phenytoin increases its cardiac depressant effects; procainamide compounds neurologic and cardiac effects.

**Prehospital Considerations**
- Only lidocaine hydrochloride injection without preservatives that is specifically labeled for IV use should be used for IV injection or infusion.
- Bolus dose of lidocaine may be given undiluted by direct IV at a rate of 50 mg or fraction thereof over 1 min.
- Lidocaine may be added to D$_5$W or NS for infusion. For adults, add 1 g to 250–500 mL; for children, add 120 mg to 100 mL.
- For IV infusion, use microdrop tubing and infusion pump. Rate of flow is usually no more than 4 mg/min.
- IV infusion should be terminated as soon as patient’s basic cardiac rhythm stabilizes or at earliest signs and symptoms of toxicity (infusions are rarely continued beyond 24 hr).
- Inspect solutions for particulate matter and discoloration prior to administration and discard if either is present.
- If ECG signs of excessive cardiac depression occur, such as prolongation of PR interval or QRS complex and the appearance or aggravation of dysrhythmias, infusion should be stopped immediately.
- Constant ECG monitoring and frequent determinations of BP, respirations, and CNS status are essential to avoid potential overdosage and toxicity.
- Auscultate lungs for basilar rales, especially in patients who tend to metabolize the drug slowly (e.g., CHF, cardiogenic shock, hepatic dysfunction).
• In patients receiving IV infusions of lidocaine or those with high lidocaine blood levels, watch for neurotoxic effects: drowsiness, dizziness, confusion, paresthesias, visual disturbances, excitement, dysarthria, behavioral changes.

• The role of lidocaine in advanced life support is more limited based on recent AHA guidelines. Prophylactic lidocaine should not be used. Lidocaine is now considered second-line therapy behind amiodarone, procainamide, and sotalol.

**LORAZEPAM**

**Class:** Sedative

**Trade Names:** Ativan, Novo-Lorazepam, Nu-Loraz (Can), Apo-Lorazepam (Aus)

**Therapeutic Actions/Pharmacodynamics:** Lorazepam is the most potent of the available benzodiazepines. Its effects (anxiolytic, sedative, hypnotic, and skeletal muscle relaxant) are mediated by the inhibitory neurotransmitter GABA. Action sites include the thalamic, hypothalamic, and limbic levels of CNS. Lorazepam has a shorter half-life than diazepam and is used as a premedication for cardioversion and minor surgery because it induces amnesia and the patient’s recall of the procedure. Like diazepam, it suppresses the spread of seizure activity through the motor cortex of the brain while not abolishing the abnormal discharge focus. Because of its short half-life, it is a preferred drug for pediatric seizures. Unlike diazepam (Valium), lorazepam is water soluble and can be easily diluted for administration.

**Emergency Uses:** To induce sedation for cardioversion. *Adult dose:* 2–4 mg IM; 0.5–2 mg IV. *Pediatric dose:* 0.03–0.05 mg/kg IV/IM/PR up to 4 mg.

To manage status epilepticus. *Adult dose:* 2 mg slow IV (2 mg/min). *Pediatric dose:* 0.1 mg/kg slow IV (over 2–5 min).
repeat one-half dose as needed. The drug may be given rectally if an IV cannot be placed.

**Pharmacokinetics**

*Absorption:* Onset is 1−5 min IV, 15−30 min IM; peak effects in 15−20 min IV, 2 hr IM; duration is 6−8 hr; half-life is 10−20 hr.

*Distribution:* Crosses placenta; distributed into breast milk.

*Metabolism:* Not metabolized in liver.

*Elimination:* Excreted in urine.

**Contraindications and Precautions:** Lorazepam is contraindicated in patients with known sensitivity to benzodiazepines, children younger than 12 yr (PO preparation), pregnancy (category D), and nursing mothers. Use with caution in patients with acute narrow-angle glaucoma; primary depressive disorders or psychosis; coma, shock, acute alcohol intoxication, renal or hepatic impairment; organic brain syndrome; myasthenia gravis; narrow-angle glaucoma; suicidal tendency; GI disorders; elderly and debilitated patients; limited pulmonary reserve.

**Adverse/Side Effects:** CNS: Anterograde amnesia, drowsiness, sedation, dizziness, weakness, unsteadiness, disorientation, depression, sleep disturbance, restlessness, confusion, hallucinations • CV: Hypertension or hypotension • Eye: Blurred vision, diplopia • Ear: Depressed hearing • GI: Nausea, vomiting, abdominal discomfort, anorexia.

**Interactions:** Alcohol, CNS depressants, and anticonvulsants may potentiate CNS depression. Cimetidine increases lorazepam plasma levels, and increases toxicity. Lorazepam may decrease antiparkinsonism effects of levodopa; may increase phenytoin levels; smoking decreases its sedative and antianxiety effects.

**Prehospital Considerations**

- IM lorazepam is injected undiluted, deep into a large muscle mass.
• IV preparation: Prepare lorazepam immediately before use. Dilute with an equal volume of sterile water, D5W, or NS. Do not use a discolored solution or one that has a precipitate.

• IV administration: Diluted drug is injected directly into vein or into IV infusion tubing at rate not to exceed 2 mg/min and with repeated aspiration to confirm IV entry.

• IV administration to neonates, infants, children: Verify correct IV concentration and rate of infusion with physician.

• Flumazenil, a benzodiazepine antagonist, should be available.

• Extreme precautions should be taken to prevent intra-arterial injection and perivascular extravasation.

• Patients older than 50 yr may have more profound and prolonged sedation with IV lorazepam. Usually, an initial dose of 2 mg should not be exceeded.

• Keep parenteral preparation in refrigerator; do not freeze. Store tablets at 15–30°C (59–86°F) unless manufacturer specifies otherwise.

• Equipment for maintaining patent airway should be immediately available before IV administration.

• IM or IV lorazepam injection of 2–4 mg is usually followed by a depth of drowsiness or sleepiness that permits patient to respond to simple instructions whether patient appears to be asleep or awake.

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MAGNESIUM SULFATE

**Class:** Electrolyte  
**Trade Name:** Magnesium

**Therapeutic Actions/Pharmacodynamics:** Magnesium sulfate is an essential element in many biochemical processes that occur within the body. It acts as a physiologic calcium channel blocker and blocks neuromuscular transmission. Hypomagnesemia (decreased magnesium levels) can cause cardiac dysrhythmias, including refractory ventricular fibrillation.
It also can result in symptoms of cardiac insufficiency and sudden cardiac death. When given parenterally, it acts as a CNS depressant and also a depressant of smooth, skeletal, and cardiac muscle function. It has anticonvulsant properties thought to be produced by CNS depression, principally by decreasing the amount of acetylcholine liberated from motor nerve terminals, thus producing peripheral neuromuscular blockade.

**Emergency Uses:** To reverse refractory ventricular fibrillation and pulseless ventricular tachycardia. *Adult dose:* 1–2 g IV over 1–2 min. *Pediatric dose:* 25–50 mg/kg IV/IM.

To reverse torsades de pointes: *Adult dose:* 1–2 g IV followed by infusion of 0.5–1.0 g/hr IV. *Pediatric dose:* 25–50 mg/kg IV/IM.

To provide prophylaxis following acute myocardial infarction. *Adult dose:* 1–2 g IV over 5–30 min. *Pediatric dose:* Not used.

To manage seizures caused by eclampsia. *Adult dose:* 2–4 g IV/IM. *Pediatric dose:* Not used.

**Pharmacokinetics**

*Absorption:* Onset is immediate IV, 1 hour IM; duration is 30 min.

*Distribution:* Crosses placenta; distributed into breast milk.

*Elimination:* Eliminated in kidneys.

**Contraindications and Precautions:** Magnesium is contraindicated in patients with myocardial damage, heart block, shock, persistent hypertension, hypocalcemia. Use with caution in patients with impaired renal function, digitalized patients, concomitant use of other CNS depressants or neuromuscular blocking agents.

**Adverse/Side Effects:** CNS: Sedation, confusion, depressed reflexes or no reflexes, muscle weakness, flaccid paralysis • CV: Hypotension, depressed cardiac function, complete heart block, circulatory collapse • Respiratory: Respiratory paralysis • Other: Flushing, sweating, extreme thirst, hypothermia, respiratory paralysis, hypocalcemia.
Interactions: Neuromuscular blocking agents add to respiratory depression and apnea. If administered in conjunction with digitalis, cardiac conduction abnormalities can occur.

Prehospital Considerations

• Administer magnesium slowly to minimize side effects.
• IV administration to infants, children: Verify correct IV concentration and rate of infusion with physician.
• When magnesium sulfate is given IV, patient requires constant observation. Check BP and pulse every 10–15 min or more often if indicated.
• Early indicators of magnesium toxicity (hypermagnesemia) include cathartic effect, profound thirst, feeling of warmth, sedation, confusion, depressed deep tendon reflexes, and muscle weakness.
• Before each repeated parenteral dose, patellar reflex should be tested. Depression or absence of reflexes is a useful index of early magnesium intoxication.
• Calcium chloride or calcium gluconate should be available as an antidote if serious side effects occur.
• Newborns of mothers who received parenteral magnesium sulfate within a few hours of delivery should be observed for signs of toxicity, including respiratory and neuromuscular depression.
• The role of magnesium sulfate in the management of refractory bronchospasm remains controversial. Consult with your medical director regarding use of magnesium sulfate in such conditions.

MANNITOL

Class: Osmotic diuretic
Trade Name: Osmitrol

Therapeutic Actions/Pharmacodynamics: Mannitol is an osmotic diuretic that, through its hypertonic effects, draws
water into the intravascular space. Then it induces diuresis by raising osmotic pressure of glomerular filtrate, thereby inhibiting tubular reabsorption of water and solutes. It is an effective way to reduce intracranial pressure and cerebral edema. In large doses, it may increase rate of electrolyte excretion, particularly sodium, chloride, and potassium. Mannitol reduces elevated intraocular and cerebrospinal pressures by increasing plasma osmolality, thus inducing diffusion of water from these fluids back into plasma and extravascular space.

**Emergency Use:** To reduce acute cerebral edema. **Adult dose:** 1.5–2.0 g/kg slow IV infusion. **Pediatric dose:** 0.25–0.5 g/kg IV over 60 min.

**Pharmacokinetics**

**Absorption:** Onset is 15 min; duration is 3–8 hr; half-life is 100 min.

**Distribution:** Confined to extracellular space; does not cross blood-brain barrier except with very high plasma levels in the presence of acidosis.

**Metabolism:** Small quantity metabolized to glycogen in liver.

**Elimination:** Rapidly excreted by kidneys.

**Contraindications and Precautions:** Mannitol is contraindicated in patients with marked pulmonary edema or CHF, organic CNS disease, intracranial bleeding; shock, severe dehydration, or history of allergy; pregnancy (category C).

**Adverse/Side Effects:** CNS: Headache, tremor, convulsions, dizziness, transient muscle rigidity • CV: Edema, CHF, anginalike pain, hypotension, hypertension, thrombophlebitis • Eye: Blurred vision. GI: dry mouth, nausea, vomiting • GU: Marked diuresis, urinary retention, nephrosis, uricosuria • Metabolic: Fluid and electrolyte imbalance, especially hyponatremia; dehydration, acidosis • Other: With extravasation: local edema, skin necrosis; chills, fever, allergic reactions.

**Interactions:** Mannitol should not be administered with whole blood or packed red cells as it can damage the red blood cells.
Prehospital Considerations

- Concentrations higher than 15% have a greater tendency to crystallize. Administration set with an in-line IV filter should be used when infusing concentrations of 15% or above.
- Store preferably at 15–30°C (59–86°F) unless otherwise directed. Avoid freezing. If mannitol does crystallize, warm it slowly in boiling water until the crystals disappear.
- Care should be taken to avoid extravasation. Observe injection site for signs of inflammation or edema.
- Monitor vital signs and carefully note possible indications of fluid and electrolyte imbalance (e.g., thirst, muscle cramps or weakness, paresthesias, and signs of CHF).
- Be alert to the possibility that a rebound increase in ICP sometimes occurs about 12 hr after drug administration. Patient may complain of headache or confusion.

MEPERIDINE

Class: Narcotic analgesic
Trade Name: Demerol

Therapeutic Actions/Pharmacodynamics: Meperidine is a synthetic narcotic central nervous system depressant with analgesic and sedative properties comparable to morphine, but without the hemodynamic effects. Meperidine is chemically dissimilar to morphine yet it has the same tendency for physical dependency and abuse as morphine. Also, unlike morphine, it has little or no antidiarrheic or antitussive action and produces CNS stimulation in toxic doses. Its rate of onset is slightly faster than morphine, yet its effects are much shorter in duration. Usual doses produce either no pupillary change or slight miosis, but overdosage results in marked miosis or mydriasis.

Emergency Uses: To relieve moderate to severe pain. Adult
dose: 25–50 mg IV; 50–100 mg IM. Pediatric dose: 1 mg/kg IV/IM.

Pharmacokinetics
Absorption: Onset is 5 min IV, 10 min IM; peak effect in 1 hour; duration is 2 hr IV, 2–4 hr IM; half-life is 3–5 hr.
Distribution: Crosses placenta; distributed into breast milk.
Metabolism: Metabolized in liver.
Elimination: Excreted in urine.

Contraindications and Precautions: Meperidine is contraindicated in patients with hypersensitivity to meperidine, convulsive disorders, acute abdominal conditions prior to diagnosis. Use with caution in patients with head injuries, increased intracranial pressure, asthma and other respiratory conditions, supraventricular tachycardias, prostatic hypertrophy, urethral stricture, glaucoma, elderly or debilitated patients, impaired renal or hepatic function, hypothyroidism, Addison’s disease.

Adverse/Side Effects: Allergic: Pruritus, urticaria, skin rashes, wheal and flare over IV site • CNS: Dizziness, weakness, euphoria, dysphoria, sedation, headache, uncoordinated muscle movements, disorientation, decreased cough reflex, miosis, corneal anesthesia, respiratory depression. Toxic doses: muscle twitching, tremors, hyperactive reflexes, excitement, hypersensitivity to external stimuli, agitation, confusion, hallucinations, dilated pupils, convulsions • CV: Facial flushing, light-headedness, hypotension, syncope, palpitation, bradycardia, tachycardia, cardiovascular collapse, cardiac arrest (toxic doses) • GI: Dry mouth, nausea, vomiting, constipation, biliary tract spasm • Other: Oliguria, urinary retention, profuse perspiration, respiratory depression in newborn, bronchoconstriction (large doses), phlebitis (following IV use), pain, tissue irritation and induration.

Interaction: Alcohol and other CNS depressants and cimetidine cause additive sedation and CNS depression.
Amphetamines may potentiate CNS stimulation. MAO inhibitors, selegiline, and furazolidone may cause excessive and prolonged CNS depression, convulsions, cardiovascular collapse. Phenytoin may increase toxic meperidine metabolites.

**Prehospital Considerations**

- Meperidine may cause respiratory depression. Have nalaxone available as an antidote.
- Meperidine is a Schedule II controlled substance. Always keep secured in a locked box.
- Carefully aspirate before giving IM injection to avoid inadvertent IV administration. IV injection of undiluted drug can cause a marked increase in heart rate and syncope.
- IV injection: When meperidine is given by direct IV, dilute 50 mg in a minimum of 5 mL of NS or sterile water to yield 10 mg/mL. Inject it slowly at a rate not to exceed 25 mg/min. Slower injection preferred.
- IV infusion: When meperidine is given by continuous infusion, dilute it to a concentration of 1–10 mg/mL in NS, D5W, or other compatible solution. Infusion rate should not exceed 25 mg/min. Slower rate is preferred.
- IV administration to infants, children: Verify correct IV concentration and rate of infusion/injection with physician.
- Narcotic analgesics should be given in the smallest effective dose and for the least period of time compatible with patient’s needs.
- In patients receiving repeated doses, note respiratory rate, depth, and rhythm, and size of pupils. If respirations are 12/min or below and pupils are constricted or dilated (see actions and uses) or breathing is shallow, or if signs of CNS hyperactivity are present, consult physician before administering drug.
- Vital signs should be monitored closely. Heart rate may increase markedly, and hypotension may occur. Meperidine may cause severe hypotension in postoperative patients and
those with depleted blood volume.
• Deep breathing, coughing (unless contraindicated), and changes in position at scheduled intervals may help to overcome the respiratory depressant effects of meperidine.
• Parenteral administration has caused corneal anesthesia and thus abolishment of corneal reflex in some patients. Be alert for this possibility.
• Chart the patient’s response to meperidine and evaluate continued need for the drug. Suggest to physician a change to a milder analgesic when in your judgment it is indicated.

METAPROTERENOL

Class: Sympathomimetic bronchodilator
Trade Names: Alupent, Metaprel

Therapeutic Actions/Pharmacodynamics: Potent synthetic sympathomimetic amine similar to isoproterenol in chemical structure and pharmacologic actions. Metaproterenol is a relatively selective beta2-adrenergic. The prime action of beta-adrenergic drugs is to stimulate adenyl cyclase, the enzyme that catalyzes the formation of cyclic-3’, 5’-adenosine monophosphate (cyclic AMP) from adenosine triphosphate (ATP). The cyclic AMP causes relaxation of the smooth muscles of the bronchial tree, decreasing airway resistance, facilitating mucus drainage, and increasing vital capacity. It exerts minimal effects on beta1 (heart) or alpha (peripheral vasculature) receptors. In therapeutic doses, metaproterenol, by inhibiting histamine release from mast cells, also reduces the mucus secretion, capillary leaking, and mucosal edema caused by an allergic response in the lungs.

Emergency Uses: To relieve bronchospasm in patients with reversible obstructive airway disease (asthma, chronic bronchitis, emphysema) and acute attacks of bronchospasm. Adult
**dose:** 0.65 mg via metered-dose inhaler (2 sprays); 0.2–0.3 mL in 2.5–3.0 mL saline via nebulizer. **Pediatric dose:** 0.1–0.2 mL/kg of 5% solution in 2.5–3.0 mL saline via nebulizer.

**Pharmacokinetics**

**Absorption:** Onset is 1 min; peak effect in 1 hour; duration is 1–5 hr.

**Metabolism:** Metabolized in liver.

**Elimination:** Excreted in urine.

**Contraindications and Precautions:** Metaproterenol is contraindicated in patients with sensitivity to other sympathomimetic agents; cardiac dysrhythmias associated with tachycardia; and hyperthyroidism. Use with caution in the elderly, hypertension, coronary artery disease, and diabetes.

**Adverse/Side Effects:** CNS: Nervousness, weakness, drowsiness, tremor, headache, fatigue • CV: Tachycardia, hypertension, cardiac arrest, palpitation • GI: Nausea, vomiting, bad taste • Other: Occasional difficulty in micturition and muscle cramps, throat irritation, cough, exacerbation of asthma.

**Interactions:** Expect additive effects when used with epinephrine, other sympathomimetic bronchodilators. MAO inhibitors and tricyclic antidepressants may potentiate its action on the vascular system. If your patient is taking beta blockers, the effects of metaproterenol will be antagonized.

**Prehospital Considerations**

- When using a metered-dose inhaler, instruct the patient to shake the container, exhale through nose as completely as possible, administer aerosol while inhaling deeply through mouth, and to hold breath about 10 seconds before exhaling slowly. Administer second inhalation 10 min after first.
- Monitor respiratory status. Measure peak flow and auscultate lungs before and after inhalation to determine efficacy of drug in decreasing airway resistance.
• Drug may have shorter duration of action after long-term use. Instruct to report failure to respond to usual dose.
• Advise that tremor is an anticipated side effect.
• Do not use discolored or precipitated solutions.
• Elderly patients may be especially sensitive to adrenergic drug effects. Monitor cardiac status and report tachycardia and palpitations.

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**METARAMINOL**

**Class:** Sympathomimetic

**Trade Name:** Aramine

**Therapeutic Actions/Pharmacodynamics:** Metaraminol is a potent synthetic sympathomimetic agonist. Its overall effects are similar to those of norepinephrine, but it is not as potent, has more gradual onset and longer duration of action, and usually lacks CNS stimulant effects. Metaraminol acts directly on alpha-adrenergic receptors (vasoconstriction) and also directly stimulates beta_1_ receptors of heart (positive inotropic effect); indirectly causes release of norepinephrine from storage sites. Its vasoconstrictor action increases pulmonary arterial pressure, produces sustained rise in systolic and diastolic pressures, and reduces blood flow to the kidneys. Once used to raise blood pressure in low-flow states, it is rarely used today as dopamine has become the preferred agent.

**Emergency Uses:** To manage hemodynamically significant hypotension not due to hypovolemia. *Adult dose:* 100 mg in 500 mL of D5W or NS titrated to BP response; 5–10 mg IM. *Pediatric dose:* Not used.

**Pharmacokinetics**

*Absorption:* Onset is 1–2 min IV, <10 min IM; duration is 20–90 min. *Metabolism:* Metabolized in tissues. *Elimination:* Excreted in urine.
Contraindications and Precautions: Metaraminol is contraindicated in patients with hypovolemia, within 14 days of MAO inhibitor therapy; peripheral or mesenteric thrombosis; pulmonary edema, cardiac arrest; untreated hypoxia, hypercapnea, and acidosis. Safe use during pregnancy (category D) not established. Use with caution in digitalized patients and those with hypertension, thyroid disease, diabetes mellitus, cirrhosis of liver, and history of malaria (may produce relapse).

Adverse/Side Effects: CNS: Apprehension, restlessness, headache, tremor, weakness, convulsions • CV: Precordial pain, palpitation, tachycardia, bradycardia, severe hypertension, dysrhythmias, cardiac arrest • Respiratory: Acute pulmonary edema • GI: Nausea, vomiting • Skin: Flushing, pallor, sweating, tissue necrosis, sloughing • Metabolic: Metabolic acidosis (hypovolemic patients), hyperglycemia.

Interactions: Metaraminol can be deactivated by alkaline solutions such as sodium bicarbonate. Concomitant use with ergot alkaloids, MAO inhibitors, or tricyclic antidepressants may cause an excessive vasopressor response. Phentolamine may decrease the vasopressor response.

Prehospital Considerations
• IV preparation: For adults, 15–100 mg may be dissolved in 500 mL of D5W, NS, or other compatible IV fluid and titrated to maintain BP at a desired level.
• IV administration: IV flow rate will be prescribed by physician (usually, systolic BP is maintained at 80–100 mm Hg for previously normotensive patients; for previously hypertensive patients it is maintained at 30–40 mm Hg below the usual pressure).
• When infusion is to be discontinued, flow rate should be reduced gradually, and abrupt withdrawal avoided. Equipment for reinstituting therapy should be immediately available.
• Patients receiving drug IV must be constantly attended, with
infusion flow rate being closely monitored. Changes in flow rate must be made cautiously, since the drug has cumulative effect and prolonged action.
• Care should be taken to avoid extravasation during IV infusion. Injury to local tissue and necrosis may result.
• Avoid exposure of drug to excessive heat, and protect it from light.
• During IV infusion, check BP every 5 min until it is stabilized at prescribed level, then every 15 min thereafter throughout therapy. Also note pulse rate and quality.
• Continue monitoring at regular intervals for several hr after infusion is complete.
• Metaraminol primarily works by stimulating the release of stored catecholamines. Patients who have been stressed by cardiac arrest or acute myocardial infarction have depleted stores of catecholamines and the effects of metaraminol may be much less than expected.

METHYPREDNISOLONE

Class: Steroid
Trade Names: A-MethaPred, Solu-Medrol
Therapeutic Actions/Pharmacodynamics:
Methylprednisolone is an intermediate-acting synthetic adrenal corticosteroid with similar glucocorticoid activity but considerably fewer sodium and water retention effects than hydrocortisone. Like the other steroids, its pharmacologic actions are vast and complex and, in medicine, have a wide range of uses.
Emergency Uses: To reduce the inflammation caused by severe anaphylaxis and asthma/COPD; to treat urticaria. Adult dose: 125–250 mg IV/IM. Pediatric dose: 1–2 mg/kg/dose IV/IM. For treatment of suspected spinal cord injury: 30 mg/kg loading dose IV, followed by IV infusion of 5.4 mg/kg/hr for 23 hours.
Pharmacokinetics

Absorption: Peak effects in 4–8 days IM; duration is 1–5 weeks IM; half-life is 3.5 hr.

Metabolism: Metabolized in liver.

Elimination: HPA suppression in 18–36 hr.

Contraindications and Precautions: There are no major contraindications to using methylprednisolone in the management of acute anaphylaxis.

Adverse/Side Effects: CNS: Euphoria, headache, insomnia, confusion, psychosis, vertigo • CV: CHF, edema, hypertension • GI: Nausea, vomiting, peptic ulcer, abdominal distention • Musculoskeletal: Muscle weakness, delayed wound healing, muscle wasting, osteoporosis, aseptic necrosis of bone, spontaneous fractures • Endocrine: Fluid retention, Cushingoid features, growth suppression in children, carbohydrate intolerance, hyperglycemia • Other: Cataracts, leukocytosis, hypokalemia, malaise, hiccups.

Interactions: Furosemide and thiazide diuretics may increase potassium loss. Phenytoin, phenobarbital, isoniazid, and rifampin may decrease the effectiveness of methylprednisolone and increase the metabolism of steroids.

Prehospital Considerations

• Give IM injection deep into large muscle (not deltoid).

• Provided in Mix-O-Vial from which solution is withdrawn and given by direct IV at a rate of 500 mg or fraction thereof over 60 seconds or longer.

• IV administration to infants, children: Verify correct IV concentration and rate of infusion with physician.

• Methylprednisolone sodium succinate solution should be used within 48 hr after preparation.

• Most EMS agencies do not carry enough methylprednisolone for high-dose steroid therapy for spinal cord injuries. Systems that encounter, or are apt to encounter, spinal injuries should give consideration to preparing a spinal
injury pack with enough drug to handle a 220 pound (100 kg) adult.

**METOCLOPRAMIDE**

**Class:** Antiemetic

**Trade Names:** Clopra, Emex (Can), Maxeran (Can), Maxolon, Octamide, Pramin (Aus), Reclomide, Reglan

**Therapeutic Actions/Pharmacodynamics:** Metoclopramide is a potent central and peripheral dopamine receptor antagonist. It is structurally related to procainamide but has little antidysrhythmic or anesthetic activity. The exact mechanism of action is not clear but it appears to sensitize GI smooth muscle to effects of acetylcholine by direct action. It also increases the resting tone of the esophageal sphincter and the tone and amplitude of upper GI contractions. As a result, gastric emptying and intestinal transit are accelerated with little effect if any on gastric, biliary, or pancreatic secretions. Antiemetic action results from drug-induced elevation of CTZ threshold and enhanced gastric emptying.

**Emergency Use:** To relieve severe nausea and vomiting: *Adult dose:* 10–20 mg IM; 10 mg slow IV (over 1–2 min). *Pediatric dose:* 1–2 mg/kg/dose.

**Pharmacokinetics**

**Absorption:** Onset is 10–15 min IM, 1–3 min IV; peak effect in 1–2 hr; duration is 1–3 hr; half-life is 2.5–6 hr.

**Distribution:** Distributed to most body tissues including CNS; crosses placenta; distributed into breast milk.

**Metabolism:** Minimally metabolized in liver.

**Elimination:** Excreted in urine, 5% in feces.

**Contraindications and Precautions:** Metoclopramide is contraindicated in patients with sensitivity or intolerance to metoclopramide, allergy to sulfite agents, history of seizure disorder.
ders, concurrent use of drugs that can cause extrapyramidal symptoms, pheochromocytoma, mechanical GI obstruction or perforation; history of breast cancer. Use with caution in patients with CHF, hypokalemia; renal dysfunction; GI hemorrhage; history of intermittent porphyria.

**Adverse/Side Effects:** CNS: Mild sedation (50% of patients), fatigue, restlessness, agitation, headache, insomnia, disorientation, extrapyramidal symptoms (acute dystonic type) • GI: Nausea, constipation, diarrhea, dry mouth • Other: Urticarial or maculopapular rash, glossal or peri orbital edema, amenorrhea, impotence, altered drug absorption, hypertensive crisis (rare).

**Interactions:** Alcohol and other CNS depressants add to sedation. Anticholinergics and opiate analgesics may antagonize effect on GI motility. Pheno thiazines may potentiate extrapyramidal symptoms. Hypertension may occur when metoclopramide is administered to patients taking MAO inhibitors.

**Prehospital Considerations**
- The injection form contains sodium metabisulfite as antioxidant. If patient has history of allergy to sulfite agents, this product should be avoided.
- Store in light-resistant bottle at 15–30°C (59–86°F). Tablets are stable for 3 yr; solutions and injections, for 5 yr.
- Extrapyramidal symptoms are most likely to occur in children, young adults, and the elderly, and with high-dose treatment of vomiting associated with cancer chemotherapy. Report immediately the onset of restlessness, involuntary movements, facial grimacing, rigidity, or tremors. Have diphenydramine available as an antidote.

**METOPROLOL**
**Class:** Beta blocker
**Trade Names:** Lopressor, Apo-Metoprolol (Can), Betaloc (Can), Minax (Aus), Novometoprol (Can), Nu-Metop (Can)
**Therapeutic Actions/Pharmacodynamics:** Metoprolol is a beta-adrenergic blocking agent with preferential effect on beta₁ adrenoreceptors located primarily on cardiac muscle. At higher doses, metoprolol also inhibits beta₂ receptors located chiefly on bronchial and vascular musculature. It reduces heart rate and cardiac output at rest and during exercise; lowers both supine and standing BP, slows sinus rate and decreases myocardial automaticity. Its antihypertensive action may be due to competitive antagonism of catecholamines at cardiac adrenergic neuron sites, drug-induced reduction of sympathetic outflow to the periphery, and to suppression of renin activity. Antianginal effect is like that of propranolol. Metoprolol and other beta blockers are cardioprotective in the period following an acute myocardial infarction.

**Emergency Uses:** To reduce the incidence of ventricular fibrillation and other complications in patients who have recently suffered an MI (especially in those who did not receive thrombolytic therapy). *Adult dose:* 5 mg slow IV every 5 min up to three times (if patient remains stable), resulting in a total dose of 15 mg. *Pediatric dose:* Not used.

**Pharmacokinetics**

*Absorption:* Onset is immediate; peak effect in 20 min; duration is 13–19 hr; half-life is 3–4 hr.

*Distribution:* Crosses blood-brain barrier and placenta; distributed into breast milk. *Metabolism:* Extensively metabolized in liver.

*Elimination:* Excreted in urine.

**Contraindications and Precautions:** Metoprolol is contraindicated in patients in cardiogenic shock (BP less than 100 mm Hg), sinus bradycardia (less than 45 bpm), heart block greater than first degree (or PRI greater than 0.24), overt cardiac failure, and right ventricular failure secondary to pulmonary hypertension. It should not be used in patients with asthma or COPD in the prehospital setting. Use with caution in patients...
with impaired hepatic or renal function; cardiomegaly, CHF controlled by digitalis and diuretics; AV conduction defects; bronchial asthma and other bronchospastic diseases; history of allergy; thyrotoxicosis; diabetes mellitus; or peripheral vascular disease.

**Adverse/Side Effects:** CNS: Dizziness, fatigue, insomnia, increased dreaming, mental depression • CV: Bradycardia, palpitation, cold extremities, intermittent claudication, angina pectoris, CHF, intensification of AV block, AV dissociation, complete heart block, cardiac arrest • Respiratory: Bronchospasm (with high doses), shortness of breath • GI: Nausea, heartburn, gastric pain, diarrhea or constipation, flatulence • Skin: Dry skin, pruritus, skin eruptions • Other: Dry mouth and mucous membranes, hypoglycemia.

**Interactions:** Barbiturates and rifampin may decrease the effects of metoprolol. Cimetidine, methimazole, propylthiouracil, and oral contraceptives may increase effects of metoprolol. You may see additive bradycardic effects with digoxin. The effects of both metoprolol and hydralazine may be increased if used together. Beta agonists and metoprolol are mutually antagonistic. Verapamil may increase the risk of heart block and bradycardia.

**Prehospital Considerations**
• IV metoprolol may be given by direct IV undiluted at a rate of 5 mg over 60 seconds.
• Store at 15–30°C (59–86°F). Protect from heat, light, and moisture.
• Take apical pulse and BP before administering drug. Report to physician significant changes in rate, rhythm, or quality of pulse or variations in BP prior to administration.
• During IV administration, BP, heart rate, and ECG should be carefully monitored.
• Hypertensive patients with CHF controlled by digitalis and diuretics must be closely observed for impending heart fail-
ure: dyspnea on exertion, orthopnea, night cough, edema, distended neck veins.

**MIDAZOLAM**

**Class:** Sedative

**Trade Name:** Versed, Hypnovel (Aus)

**Therapeutic Actions/Pharmacodynamics:** Midazolam is a short-acting parenteral benzodiazepine with CNS depressant, muscle relaxant, anticonvulsant, and anterograde amnestic effects. Its exact mechanism of action is unclear. Intensifies activity of gamma-aminobenzoic acid (GABA), a major inhibitory neurotransmitter of the brain, by interfering with its reuptake and promoting its accumulation at neuronal synapses. This calms the patient, relaxes skeletal muscles, and in high doses produces sleep. Like the other benzodiazepines, it has no effect on pain. Midazolam has considerably less muscle relaxant properties than diazepam.

**Emergency Uses:** To induce sedation and amnesia prior to cardioversion and other painful procedures. *Adult dose:* 1.0–2.5 mg slow IV; 0.07–0.08 mg/kg IM (usual dose is 5 mg). *Pediatric dose:* 0.05–0.20 mg/kg IV; 0.10–0.15 mg/kg IM; 3 mg intranasal.

**Pharmacokinetics**

*Absorption:* Onset is 3–5 min IV, 15 min IM, 6–14 min intranasal; peak effects in 20–60 min; duration is less than 2 hr IV, 1–6 hr IM; half-life is 1–4 hr.

*Distribution:* Crosses blood-brain barrier and placenta.

*Metabolism:* Metabolized in liver.

*Elimination:* Excreted in urine.

**Contraindications and Precautions:** Midazolam is contraindicated in patients with intolerance to benzodiazepines, acute narrow-angle glaucoma, shock, coma, and acute alcohol
intoxication. Use with caution in patients with COPD, chronic renal failure, CHF, and in the elderly.

**Adverse/Side Effects:** CNS: Retrograde amnesia, headache, euphoria, drowsiness, excessive sedation, confusion • CV: Hypotension • Eye: Blurred vision, diplopia, nystagmus, pinpoint pupils • GI: Nausea, vomiting • Respiratory: Coughing, laryngospasm (rare), respiratory arrest • Skin: Hives, swelling, burning, pain, induration at injection site, tachypnea • Other: Hiccups, chills, weakness.

**Interactions:** Alcohol, CNS depressants, and anticonvulsants potentiate CNS depression. Cimetidine increases midazolam plasma levels, increasing its toxicity. Midazolam may decrease the antiparkinsonism effects of levodopa. It also may increase phenytoin levels. Smoking decreases its sedative and antianxiety effects.

**Prehospital Considerations**
- Inject IM drug deep into a large muscle mass, not the deltoid.
- IV midazolam is given diluted to a concentration of 0.25 mg/mL in NS or D5W. Avoid rapid injection, which may cause respiratory depression.
- IV administration to neonates, infants: Verify correct IV concentration and rate of infusion with physician.
- During IV infusion, inspect injection site for redness, pain, swelling, and other signs of extravasation.
- If the patient is premedicated with a narcotic agonist analgesic, the conscious sedation period may be marked by hypotension.
- Anterograde amnesia (dose related) correlates well with degree of drowsiness and is about the same as that for lorazepam. Most patients do not recall induction.
- In the obese patient, half-life is prolonged; therefore, duration of effects is prolonged (i.e., amnesia, postoperative recovery). Monitor vital signs for entire recovery period.
- Overdose symptoms include somnolence, confusion, seda-
tion, diminished reflexes, coma, and untoward effects on vital signs.
- Prepare patient for the amnesia to prevent an upsetting postoperative period.
- Have resuscitative equipment and flumazenil (antidote) available prior to administering midazolam.

**MILRINONE**

**Classes:** Cardiac inotrope; vasodilator

**Trade Name:** Primacor

**Therapeutic Actions/Pharmacodynamics:** Milrinone is a member of a new class of inotropic/vasodilator agents (same family as amrinone). It is a positive inotrope and vasodilator, with little chronotropic activity; mode of action and structure are different from digitalis and catecholamines as well as beta-adrenergic agents. Milrinone inhibits cyclic-AMP phosphodiesterase in cardiac and smooth vascular muscle. In therapeutic doses, milrinone increases cardiac contractility. Therefore, milrinone increases cardiac output and vascular resistance without increasing myocardial oxygen demand or significantly increasing heart rate.

**Emergency Uses:** Short term management of CHF. **Adult dose:** Loading dose of 50 µg/kg IV over 10 min followed by maintenance infusion of 0.375–0.75 µg/kg/min IV. Milrinone is used occasionally to increase cardiac output and decrease systemic vascular resistance in pediatric septic shock. **Pediatric dose:** Loading dose of 50–75 µg/kg IV followed by an IV infusion of 0.5–0.75 µg/kg/min.

**Pharmacokinetics**

**Absorption:** Peak effect is 2 min; duration is 2 hr.

**Elimination:** Half-life 1.7–2.7 hr; 80–85% excreted unchanged in the urine.
Contraindications and Precautions: Milrinone is contraindicated in patients with a hypersensitivity to milrinone. Use with caution in elderly patients, pregnancy category C, and nursing mothers.

Adverse/Side Effects: CV: Increased ectopic activity, PVCs, ventricular tachycardia, ventricular fibrillation, supraventricular dysrhythmias, possible increase in angina symptoms, hypotension.

Interactions: Disopyramide may cause excessive hypotension.

Prehospital Considerations
- IV preparation and administration: Dissolve 20 mg of milrinone in 180 mL of NS or D5W to yield 100 MOOg/mL.
- Give loading dose of 50 MOOg/kg IV over 10 min followed by maintenance dose.
- Dosages may be titrated for maximum hemodynamic effects.
- Do not administer to patients with known preexisting hypokalemia.
- Closely monitor cardiac status, ECG, pulse, blood pressure, and respirations during milrinone therapy.

MORPHINE

Class: Narcotic analgesic

Trade Names: Anamorph (Aus), Astramorph, Duramorph, Epimorph (Can), Infumorph, Kadian, Morphine, Roxanol, Statex

Therapeutic Actions/Pharmacodynamics: Morphine is a natural opium alkaloid that acts on opiate receptors in the brain providing both analgesia and sedation. Morphine is one of the most potent analgesics known, yet its hemodynamic properties make it extremely useful in managing patients with acute myocardial infarction and pulmonary edema. Its vasodilatory effects increase peripheral venous capacitance and reduce
venous return. This reduces the cardiac workload and decreases myocardial oxygen demand, thus reducing infarction size in acute MI. In pulmonary edema, reducing preload significantly decreases pulmonary venous congestion.

**Emergency Uses:** To relieve moderate to severe pain. *Adult dose:* 2.5–15 mg IV; 5–20 mg IM/SC. *Pediatric dose:* 0.05–0.1 mg/kg IV; 0.1–0.2 mg/kg IM/SC.

To reduce venous return in acute MI and acute pulmonary edema. *Adult dose:* 1–2 mg every 6–10 min until desired response. *Pediatric dose:* Not used.

**Pharmacokinetics**

*Absorption:* Onset is immediate IV, 15–30 min IM/SC; peak effect in 20 min IV, 30–60 min IM/SC; duration is 2–7 hr.

*Distribution:* Crosses blood-brain barrier and placenta; distributed in breast milk. *Metabolism:* Metabolized primarily in liver.

*Elimination:* 90% of drug and metabolites excreted in urine in 24 hr, 10% excreted in bile.

**Contraindications and Precautions:** Morphine is contraindicated in patients with hypersensitivity to opiates. Because it may mask symptoms, morphine should not be administered in the prehospital setting to patients with undiagnosed head injury or acute abdomen. Because of its vasodilatory effects, do not administer morphine to patients who are volume depleted or severely hypotensive. Do not use in patients with acute bronchial asthma, chronic pulmonary diseases, severe respiratory depression, and pulmonary edema induced by chemical irritants. Use with caution in very old, very young, or debilitated patients.

**Adverse/Side Effects:** Allergic: Pruritus, rash, urticaria, edema, hemorrhagic urticaria (rare), anaphylactoid reaction (rare) • CNS: Respiratory depression, euphoria, insomnia, disorientation, visual disturbances, dysphoria, paradoxic CNS stimulation (restlessness, tremor, delirium, insomnia), convul-
sions (infants and children); decreased cough reflex, drowsi-
ness, dizziness, miosis. CV: Bradycardia, palpitations, syncope;
flushing of face, neck, and upper thorax; orthostatic hypoten-
sion • GI: Constipation, anorexia, dry mouth, biliary colic, nau-
sea, vomiting, elevated transaminase levels • GU: Urinary
retention or urgency, dysuria, oliguria, reduced libido or poten-
cy (prolonged use) • Other: Sweating, prolonged labor and respi-
atory depression of newborn, precipitation of porphyria
• Overdosage: Severe respiratory depression (as low as 2–
4/min) or arrest; pulmonary edema, deep sleep, coma; skeletal
muscle flaccidity; cold, clammy skin; hypotension, bradycar-
dia, cardiac arrest, marked miosis, hypothermia.

**Interactions:** CNS depressants, sedatives, barbiturates, alco-
hol, benzodiazepines, and tricyclic antidepressants may poten-
tiate the CNS depressant effects. Use MAO inhibitors cautiously;
they may precipitate hypertensive crisis. Phenothiazines
may antagonize analgesia.

**Prehospital Considerations**
• Dosage for elderly or debilitated patients is lower than for
adult.
• IV administration: Morphine may be given by direct IV,
diluted in 5 mL of sterile water for injection, over 4–5 min.
• IV administration to neonates, infants, children: Verify cor-
rect IV concentration and rate of infusion/injection with
physician.
• Before administering the drug, note respiratory rate, depth,
and rhythm and size of pupils. Respirations of 12/min or
below and miosis are signs of toxicity. Withhold drug and
report to physician.
• Observe patient closely to be certain pain relief is achieved.
Record relief of pain (preferably in patient’s own words) and
duration of analgesia for reference when dosage modifica-
tion is being considered.
• Elevated pulse or respiratory rate, restlessness, anorexia, or drawn facial expression may indicate need for analgesia.
• Differentiate among restlessness as a sign of pain and the need for medication, restlessness associated with hypoxia, and restlessness caused by morphine-induced CNS stimulation (a paradoxic reaction that is particularly common in women and elderly patients).
• Monitor vital signs at regular intervals. Morphine-induced respiratory depression may occur even with small doses, and it increases progressively with higher doses (generally reaching maximum within 90 min following SC, 30 min after IM, and 7 min after IV administration).
• Nausea and orthostatic hypotension (with light-headedness and dizziness) most often occur in ambulatory patients or when a supine patient assumes the head-up position or in patients not experiencing severe pain.
• Closely observe the patient with increased intracranial pressure or with head injury. Morphine effects may obscure neurologic signs of further increase in intracranial pressure.
• Have naloxone available as an antidote prior to administration.
• Morphine is a Schedule II controlled substance.

NALBUPHINE

Class: Narcotic analgesic
Trade Name: Nubain

Therapeutic Actions/Pharmacodynamics: Nalbuphine is a synthetic narcotic analgesic with both agonist and antagonist properties. Its analgesic potency is equal to that produced by equivalent doses of morphine. On a weight basis, it produces respiratory depression about equal to that of morphine; however, in contrast to morphine, higher doses (greater than 10 mg) produce no further respiratory depression. Its antagonistic potency is approximately one-fourth that of naloxone.
Emergency Use: To relieve moderate to severe pain. *Adult dose:* 5 mg IV/IM/SC; repeat 2 mg doses as needed up to 20 mg. *Pediatric dose:* 0.10–0.15 mg/kg IV/IM/SC (rarely used).

**Pharmacokinetics**
*Absorption:* Onset is 2–3 min IV, 15 min IM; peak effect in 30 min IV; duration is 3–6 hr; half-life is 5 hr.
*Distribution:* Crosses placenta.
*Metabolism:* Metabolized in liver.
*Elimination:* Eliminated in urine.

**Contraindications and Precautions:** Nalbuphine is contraindicated in patients with a history of hypersensitivity to the drug. Like morphine, it should not be administered to patients with undiagnosed head injury or acute abdomen in the prehospital setting. Use with caution in patients with impaired respirations. Because it may reverse the effects of narcotics, use with caution in patients with narcotic dependency.

**Adverse/Side Effects:** CNS: Nervousness, depression, restlessness, crying, euphoria, dysphoria, distortion of body image, unusual dreams, confusion, hallucinations; numbness and tingling sensations, headache, miosis • CV: Hypertension, hypotension, bradycardia, tachycardia, flushing • Respiratory: Dyspnea, asthma, respiratory depression • GI: Abdominal cramps, bitter taste, nausea, vomiting • Hypersensitivity: Pruritus, urticaria, burning sensation • Other: Speech difficulty, urinary urgency, blurred vision.

**Interactions:** Alcohol and other CNS depressants add to CNS depression. Nalbuphine can cause withdrawal symptoms in narcotic-addicted patients.

**Prehospital Considerations**
- Nalbuphine may be given by direct IV undiluted at a rate of 10 mg over 3–5 min.
- IV administration to infants, children: Verify correct rate of IV injection with physician.
• Protect nalbuphine from light and store at 15−30°C (59−86°F) unless otherwise directed.
• Assess respiratory rate before drug administration. Withhold drug and notify physician if respiratory rate falls below 12.
• Nalbuphine may produce allergic response in persons with sulfite sensitivity.
• Administer with caution to patients with hepatic or renal impairment.
• Nalbuphine may produce drowsiness. Monitor ambulatory patients.
• Use of drug during labor and delivery may cause respiratory depression of newborn.
• Because of its antagonistic properties, nalbuphine makes it difficult for anesthesia personnel to provide a balanced narcotic anesthesia to patients who received the drug in the field. Thus, there is an increased emphasis on utilizing the traditional analgesics (morphine, meperidine) in prehospital care.

**NALOXONE**

**Class:** Narcotic antagonist  
**Trade Name:** Narcan  

**Therapeutic Actions/Pharmacodynamics:** Naloxone, an analog of oxymorphone, is a pure narcotic antagonist, essentially free of agonistic (morphine-like) properties. Thus it produces no significant analgesia, respiratory depression, psychotomimetic effects, or miosis when administered in the absence of narcotics and possesses more potent narcotic antagonist action. Naloxone competes for and displaces narcotic molecules from opiate receptors in the brain. It is used mainly to reverse the respiratory depression associated with overdose of the following narcotic agents: morphine, heroin, methadone, codeine, paregoric, meperidine (Demerol), hydromorphone (Dilaudid), fentanyl (Sublimaze), hydrocodone (Percodan), nal-
buphine (Nubain), propoxyphene (Darvon), pentazocine (Talwin), butorphanol (Stadol).

**Emergency Uses:** To reverse the effects of narcotic analgesics; to manage coma of unknown origin. *Adult dose:* 0.4–2.0 mg IV/IM, 2.0–2.5 times dose ET; may be repeated every 2–3 min up to 10 mg until respirations are restored. *Pediatric dose:* 0.01 mg/kg IV/IM, 2.0–2.5 times dose ET; may be repeated every 2–3 min up to 10 mg until respirations are restored.

**Pharmacokinetics**

*Absorption:* Onset and peak effects in less than 2 min IV, 2–10 min IM/ET; duration is 20–120; half-life is 60–90 min.

*Distribution:* Crosses placenta.

*Metabolism:* Metabolized in liver.

*Elimination:* Excreted in urine.

**Contraindications and Precautions:** Naloxone is contraindicated in patients with a hypersensitivity to the drug and in patients whose respiratory depression is due to nonopioid drugs. Safe use during pregnancy (other than labor) (category B) and in nursing mothers not established. Because naloxone may cause abrupt and complete reversal of the narcotic effects and the resulting withdrawal, use with caution in patients who are known or suspected narcotic addicts. This includes newborn infants of mothers with known or suspected narcotic dependence.

**Adverse/Side Effects:** Excessive dosage in narcotic depression: Reversal of analgesia, increased BP; tremors, hyperventilation, slight drowsiness, elevated partial thromboplastin time

- Too rapid reversal: Nausea, vomiting, sweating, tachycardia.

**Interactions:** Naloxone may cause withdrawal symptoms in narcotic addicts. Use only enough to reverse respiratory depression.
Prehospital Considerations

- Naloxone may be given rapidly by direct IV in 0.1–0.2 mg increments at 2–3-min intervals until desired narcotic reversal is achieved.
- Duration of action of some narcotics may exceed that of naloxone; therefore, patient must be closely observed. Keep physician informed; repeat naloxone dose may be necessary.
- Narcotic abstinence symptoms induced by naloxone generally start to diminish 20–40 min after administration and usually disappear within 90 min.
- Monitor respirations and other vital signs.
- The goal of prehospital naloxone therapy is to reverse any respiratory depression. Large, one-time boluses can induce an opiate withdrawal reaction that can be difficult to manage (massive diarrhea, vomiting, runny nose, coughing, abdominal pain, and agitation).

NIFEDIPINE

Class: Calcium channel blocker

Trade Names: Adalat, Novo-Nifedin (Can), Procardia

Therapeutic Actions/Pharmacodynamics: Nifedipine is a calcium channel blocking agent that selectively blocks calcium ion influx across cell membranes of cardiac muscle and vascular smooth muscle without changing serum calcium concentrations. It reduces myocardial oxygen utilization and supply and relaxes and prevents coronary artery spasm but has little or no effect on SA and AV nodal conduction with therapeutic dosing. Nifedipine decreases peripheral vascular resistance and increases cardiac output. Vasodilation of both coronary and peripheral vessels is greater than that produced by verapamil or diltiazem and frequently results in reflex tachycardia.
Decreased peripheral vascular resistance also leads to a rise in peripheral blood flow, the basis for use of this drug in treatment of Raynaud’s phenomenon. It has minimal effect on myocardial contractility. Nifedipine is a class IV anti-dysrhythmic.

**Emergency Uses:** To increase coronary artery perfusion in angina pectoris; to manage severe hypertension.

**Adult dose:** 10–20 mg capsule SL/PO.

**Pediatric dose:** Not used.

**Pharmacokinetics**

**Absorption:** Onset is 1–5 min SL, 5–20 min PO; peak effects in 20–30 min SL, 1–2 hr PO; duration is 2–5 hr; half-life is 2–5 hr.

**Distribution:** Distributed into breast milk.

**Metabolism:** Metabolized in liver.

**Elimination:** 75–80% excreted in urine, 15% in feces.

**Contraindications and Precautions:** Nifedipine is contraindicated in patients with known hypersensitivity to nifedipine and in hypotensive patients.

**Adverse/Side Effects:** CNS: Dizziness, light-headedness, nervousness, mood changes, weakness, jitteriness, sleep disturbances, blurred vision, retinal ischemia, difficulty in balance, headache • CV: Hypotension, facial flushing, heat sensation, palpitations, peripheral edema, MI (rare) • GI: Nausea, heartburn, diarrhea, constipation, cramps, flatulence • Musculoskeletal: Inflammation, joint stiffness, muscle cramps • Other: Sore throat, weakness, dermatitis, pruritus, urticaria, gingival hyperplasia, fever, sweating, chills, febrile reaction, nasal congestion, sexual difficulties, dyspnea, cough, wheezing • Overdose: Prolonged systemic hypotension.

**Interactions:** Beta blockers may increase the likelihood of CHF, bradycardia, and asystole. Nifedipine may increase the risk of phenytoin toxicity.
Prehospital Considerations

• To administer nifedipine sublingually, place several puncture holes in the capsule before placing it under the tongue where it can be absorbed.
• Nifedipine should not be given within the first 1–2 wk following an MI.
• Nifedipine should not be used in a hypertensive emergency. As a rule, high blood pressure is a chronic illness. If the blood pressure must be lowered because of end-organ changes, then labetalol or sodium nitroprusside should be used.
• Protect capsules from light and moisture; store at 15–25°C (59–77°F).
• Careful monitoring of BP during titration period is indicated. Severe hypotension may be produced, especially if patient is also taking other drugs known to lower BP. Withhold drug and notify physician if systolic BP is less than 90.
• Monitor the blood sugar in diabetic patients. Nifedipine has diabetogenic properties.

NITROGLYCERIN

Class: Nitrate
Trade Names: Anginine (Aus), Deponit, GTN-Pohl (Aus), Minitran, Nitradisc (Aus), Nitro-Bid, Nitrocap, Nitrocine, Nitrodisc, Nitro-Dur, Nitrogard, Nitroglyc, Nitroject, Nitrol, Nitrolate (Aus), Nitrolingual, Nitrong, Nitrostat, Transderm-Nitro (Aus), Tridil

Therapeutic Actions/Pharmacodynamics: Nitroglycerin is an organic nitrate and potent vasodilator with antianginal, anti-ischemic, and antihypertensive effects. It relaxes vascular smooth muscle by unknown mechanism, resulting in dose-related dilation of both venous and arterial blood vessels. It also promotes peripheral pooling of blood, reduction of periph-
eral resistance, and decreased venous return to the heart. Both left ventricular preload and afterload are reduced and myocardial oxygen consumption or demand is decreased. Therapeutic doses may reduce systolic, diastolic, and mean BP; heart rate is usually slightly increased.

**Emergency Uses:** To increase coronary artery perfusion and relieve chest pain in angina and acute myocardial infarction; to reduce preload in acute pulmonary edema. **Adult dose:** 0.4 mg SL, may repeat every 3−5 min up to 3 tablets; or 1/2 to 1 inch (1.25−2.50 cm) ointment applied topically; or 0.4 mg (1 spray) SL spray up to 3 sprays/25 min. **Pediatric dose:** Not used.

**Pharmacokinetics**

**Absorption:** Onset is 1−3 min SL; 30 min transdermal; peak effect in 5−10 min SL; duration is 20−30 min SL; 3−6 hr transdermal; half-life is 1−4 min.

**Distribution:** Widely distributed; not known if distributes to breast milk.

**Metabolism:** Extensively metabolized in liver.

**Elimination:** Inactive metabolites excreted in urine.

**Contraindications and Precautions:** Nitroglycerin is contraindicated in patients with hypersensitivity, idiosyncrasy, or tolerance to nitrates; patients taking sildenafil; severe anemia; head trauma, increased ICP; glaucoma (sustained release forms). Do not administer to patients in shock.

**Adverse/Side Effects:** CNS: Headache, apprehension, blurred vision, weakness, vertigo, dizziness, faintness • CV: Postural hypotension, palpitations, tachycardia (sometimes with paradoxical bradycardia), increase in angina, syncope, and circulatory collapse • GI: Nausea, vomiting, involuntary passing of urine and feces, abdominal pain, dry mouth • Skin: Cutaneous vasodilation with flushing, rash, exfoliative dermatitis, contact dermatitis with transdermal patch; topical allergic reactions with ointment: pruritic eczematous eruptions, anaphylactoid reaction characterized by oral mucosal and conjunctival edema
Other: Muscle twitching, pallor, perspiration, cold sweat; local sensation in oral cavity at point of dissolution of sublingual forms.

**Interactions:** Alcohol and antihypertensive agents may compound the hypotensive effects. It may cause orthostatic hypotension when used in conjunction with beta blockers. Patients taking sildenafil are at risk for severe cardiac event.

**Prehospital Considerations**

**General**
- Approximately 50% of all patients experience mild to severe headaches following nitroglycerin. Assess patient and consult as needed with physician about analgesics and dosage adjustment.
- Transient headache usually lasts about 5 min after sublingual administration and seldom longer than 20 min. Assess degree of severity.
- Postural hypotension may occur even with small doses of nitroglycerin. Patient may complain of dizziness or weakness due to postural hypotension. Supervision of ambulation may be indicated, especially with the elderly or debilitated patient.
- Use with caution in patients who have taken Sildenafil (Viagra) in the last 6 hr as hypotension can result.
- Overdose symptoms include hypotension, tachycardia; warm, flushed skin becoming cold and cyanotic; headache, palpitations, confusion, nausea, vomiting, moderate fever, and paralysis. Tissue hypoxia leads to coma, convulsions, cardiovascular collapse. Death can occur from asphyxia.

**Sublingual Tablet**
- Instruct patient to sit or lie down upon first indication of oncoming anginal pain and to place tablet under tongue or in buccal pouch (hypotensive effect of drug is intensified in the upright position).
- Instruct patient to allow tablet to dissolve naturally and not to swallow until drug is entirely dissolved. Advise patient...
Nitroglycerin

with dry mouth to take a sip of water or place 1 mL saline under the tongue before taking the nitroglycerin tablet.
• If pain is not relieved after 1 tablet, additional tablets may be taken at 5-min intervals, but not more than 3 tablets should be taken in a 15-min period. Taking more tablets than necessary can further decrease coronary blood flow by producing systemic hypotension.
• Any local burning or tingling from the sublingual form has no clinical significance.
• Always make sure that nitroglycerin tablets are fresh. The drug readily degrades upon exposure to air and light.

Sublingual Spray
• Do not shake canister. Spray preferably on or under tongue. Do not inhale spray.
• Spray may be repeated every 5 min for a maximum of 3 metered doses.
• Wait at least 10 seconds before swallowing.

Transdermal
• Using dose-determining applicator (patch) supplied with package, squeeze prescribed dose onto the applicator. Place patch with ointment-side down onto desired site. Using applicator spread ointment in a thin, uniform layer to pre-marked 5.5 cm by 9 cm (2¼ in by 3½ in) square nonhairy skin surface (areas commonly used: chest, abdomen, anterior thigh, forearm). Cover with transparent wrap and secure with tape. Avoid getting ointment on fingers.
• Keep ointment container tightly closed and store in cool place.
• Before initiation of treatment with transdermal preparations, take baseline BP and heart rate, with patient in sitting position.
• Assess for and report blurred vision or dry mouth.
• Assess for and report the following topical reactions to drug administration: contact dermatitis from the transdermal patch; pruritus and erythema from the ointment.
NITROUS OXIDE

Class: General anesthetic
Trade Name: Nitronox

Therapeutic Actions/Pharmacodynamics: Nitrous oxide (N₂O) is a general inhalation anesthetic and is the principal adjunct to anesthesia. It is almost odorless, nonexplosive gas with relatively low anesthetic potency and muscle relaxant properties; however, the drug has strong analgesic properties. Nitronox is a blended mixture of 50% nitrous oxide and 50% oxygen that has potent analgesic effects. It is a self-administered CNS depressant whose effects quickly dissipate within 2–5 min after cessation of administration. It is used in many situations to manage pain and, at the same time, reduce hypoxia.

Emergency Uses: To relieve pain of musculoskeletal origin (especially fractures), burns, suspected ischemic chest pain, and severe states of anxiety including hyperventilation. Adult dose: Self-administered inhalation until pain is relieved or patient drops mask. Pediatric dose: Same as adult.

Pharmacokinetics
Absorption: Onset, peak effect, and duration are 2–5 min.
Distribution: Generally to all body areas.
Excretion: Rapidly eliminated via the lungs, with small amounts being eliminated through the skin and breast milk.

Contraindications and Precautions: Nitronox is contraindicated in a patient who cannot understand verbal instructions, who is intoxicated with alcohol or other drugs, or who has an altered mental status following a head injury. Do not use with patients with COPD for two reasons: (1) if they have hypoxic drive, the 50% oxygen may cause respiratory depression or arrest; (2) many COPD patients have blebs in their lungs and nitrous oxide tends to diffuse into these closed spaces and cause swelling. Swollen blebs may rupture causing a pneumothorax. For this same reason, do not administer Nitronox to
patients with thoracic injuries, because a simple pneumothorax can be greatly worsened, or to patients with bowel obstruction.

**Adverse/Side Effects:** CNS: Dizziness, light-headedness, altered mental status, hallucinations • GI: Nausea and vomiting.

**Interactions:** Nitrous oxide can potentiate the effects of other CNS depressants such as narcotics, sedatives, hypnotics, and alcohol.

**Prehospital Considerations:** Only use nitrous oxide in areas that are well ventilated. When used in the back of your ambulance, it is recommended that a scavenging system be in place. In countries where single cylinder nitrous-oxide/oxygen (Dolonoxy) systems are used, be sure that the equipment is kept above 40°F as the nitrous oxide will assume the liquid state and oxygen only will be administered.

**NOREPINEPHRINE**

**Class:** Sympathomimetic

**Trade Names:** Levarterenol, Levophed, Noradrenaline

**Therapeutic Actions/Pharmacodynamics:** Norepinephrine is a direct-acting sympathomimetic amine identical to body catecholamine norepinephrine. It acts directly and predominantly on alpha-adrenergic receptors; little action on beta receptors except in heart (beta1 receptors). Its main therapeutic effects are vasoconstriction and cardiac stimulation. It has powerful vasoconstrictor action on resistance and capacitance blood vessels. Peripheral vasoconstriction and moderate inotropic stimulation of heart result in increased systolic and diastolic blood pressure, myocardial oxygenation, coronary artery blood flow, and work of heart. Cardiac output varies reflexively with systemic BP.

**Emergency Uses:** To restore blood pressure in certain acute
hypotensive states in patients refractory to other sympathomimetics; neurogenic shock. **Adult dose:** 0.5–30 µg/min IV infusion titrated to BP. **Pediatric dose:** 0.01 µg/kg/min (rarely used).

**Pharmacokinetics**  
*Absorption:* Onset and peak effects are very rapid; duration is 1–2 min after termination of infusion.  
*Distribution:* Localizes in sympathetic nerve endings; crosses placenta.  
*Metabolism:* Metabolized in liver and other tissues by COMT and MAO.  
*Elimination:* Excreted in urine.

**Contraindications and Precautions:** Norepinephrine is contraindicated as the sole therapy in hypovolemic states, except as a temporary emergency measure. Use with caution in patients with hypertension; hyperthyroidism; severe heart disease; in elderly patients; within 14 days of MAO inhibitor therapy; and in patients receiving tricyclic antidepressants.

**Adverse/Side Effects:** CNS: Headache, restlessness, anxiety, tremors, dizziness, weakness, insomnia • CV: Palpitation, hypertension, reflex bradycardia, fatal dysrhythmias (large doses) • Resp: Respiratory difficulty • Skin: Pallor, tissue necrosis at injection site (with extravasation) • With prolonged administration: Plasma volume depletion, edema, hemorrhage, intestinal, hepatic, and renal necrosis • Overdosage or individual sensitivity: Blurred vision, photophobia, hyperglycemia, retrosternal and pharyngeal pain, profuse sweating, vomiting, severe hypertension, violent headache, cerebral hemorrhage, convulsions.

**Interactions:** Alpha and beta blockers antagonize its pressor effects. Tricyclic antidepressants may potentiate its pressor effects.

**Prehospital Considerations**  
• IV infusion of norepinephrine in saline alone is not recom-
Norepinephrine

mended. Dextrose (in distilled water or saline solution) is
used to prevent oxidation and thus loss of potency. Usual
dilution is a 4-mg ampule in 1000 mL diluent to yield 4
µg/mL.
• Do not use solution if discoloration or precipitate is present.
  Protect from light.
• Initial rate of infusion is 0.5–1.0 µg/min; then titrated to
  maintain BP. An infusion pump is used. Consult medical
director for specific titration guidelines.
• Risk of extravasation is reportedly reduced if infusion is
  administered through a plastic catheter inserted deep into vein.
• Flow rate must be constantly monitored. Check infusion site
  frequently (tape should not obscure injection site). Report
  immediately any evidence of extravasation: blanching along
  course of infused vein (may occur without obvious extra-
  vasation), cold, hard swelling around injection site.
• When therapy is to be discontinued, infusion rate is slowed
  gradually. Abrupt withdrawal should be avoided.
• Patient should be monitored constantly while receiving nor-
  epinephrine. Take baseline BP and pulse before start of ther-
  apy, then every 2 min from initiation of drug until stabiliza-
  tion occurs at desired level, then every 5 min during drug
  administration.
• In normotensive patients, it is recommended that flow rate be
  adjusted to maintain BP at low normal (usually 80–100 mm
  Hg systolic). In previously hypertensive patients, systolic is
  generally maintained no higher than 40 mm Hg below preex-
  isting systolic level.
• In addition to vital signs, carefully observe and record men-
  tal status (index of cerebral circulation), skin temperature of
  extremities, and color (especially of earlobes, lips, nail beds).
• Be alert to patient’s complaints of headache, vomiting, palpi-
tations, dysrhythmias, chest pain, photophobia, and blurred vision as possible symptoms of overdosage. Reflex bradycardia may occur as a result of rise in BP.

• Continue to monitor vital signs and observe patient closely after cessation of therapy for clinical sign of circulatory inadequacy.

• Extravasation may require treatment with phentolamine (Regitine), an alpha adrenergic blocker. 5–10 mg should be diluted in 10 mL of NS and injected into the affected area.

OXYGEN

Class: Gas
Trade Name: Oxygen

Therapeutic Actions/Pharmacodynamics: Oxygen is a tasteless, odorless, colorless gas necessary for life. Oxygen enters the body through the respiratory system and is transported to the cells by hemoglobin, found in the red blood cells. Oxygen is required for the efficient breakdown of glucose into a usable energy form. The administration of enriched oxygen increases the oxygen concentration in the alveoli, which subsequently increases the oxygen saturation of available hemoglobin.

Emergency Uses: To manage any situation in which hypoxia is suspected. Adult dose: 100% if patient is hypoxic. Pediatric dose: Same as adult.

Pharmacokinetics
Absorption: Onset is immediate; peak effect is within 1 min; duration is less than 2 min.

Contraindications and Precautions: There are no contraindications to oxygen. Use with caution in patients with chronic obstructive pulmonary disease who may have hypoxic drive. If these patients suffer respiratory depression from the enriched oxygen, simply perform positive pressure ventilation as need-
ed. Never withhold oxygen from a hypoxic patient, regardless of the history or diagnosis. In a prolonged transport of a neonate, high concentrations of oxygen may damage the infant’s eyes (retrolental fibroplasia). This is rarely a prehospital concern, but is a consideration. Oxygen delivered at a rate of greater than 6 L/min should be humidified to prevent drying of the mucus membranes of the upper respiratory tract. Use a pulse oximeter to monitor the oxygen saturation of hemoglobin. This is an easy, reliable indicator of your patient’s oxygen delivery status.

**Adverse/Side Effects:** Respiratory: Dried mucous membranes, irritation of upper respiratory tract.

**Interactions:** Oxygen may increase the toxicity of certain herbicides (i.e., paraquat, diaquat) in patients who may have ingested these poisons. These chemicals are sometimes sprayed onto illicit agricultural products such as marijuana.

**Prehospital Considerations**: Use the oxygen delivery system indicated by the situation and desired concentration:

<table>
<thead>
<tr>
<th>Device</th>
<th>Flow Rate</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasal cannula</td>
<td>1–6 L/min</td>
<td>24–44%</td>
</tr>
<tr>
<td>Simple face mask</td>
<td>8–10 L/min</td>
<td>40–60%</td>
</tr>
<tr>
<td>Venturi mask</td>
<td>4–12 L/min</td>
<td>24–50%</td>
</tr>
<tr>
<td>Partial rebreather</td>
<td>6–10 L/min</td>
<td>35–60%</td>
</tr>
<tr>
<td>Nonrebreather</td>
<td>6–10 L/min</td>
<td>60–90%</td>
</tr>
<tr>
<td>BVM with reservoir</td>
<td>10–15 L/min</td>
<td>40–90%</td>
</tr>
<tr>
<td>Demand valve</td>
<td>10–15 L/min</td>
<td>100%</td>
</tr>
</tbody>
</table>

**OXYTOCIN**

**Class:** Hormone

**Trade Names:** Oxytocin, Pitocin, Syntocinon

**Therapeutic Actions/Pharmacodynamics:** Oxytocin is a synthetic, water-soluble polypeptide identical pharmacologically to
the oxytocin secreted by the posterior pituitary. By direct action on myofibrils, it produces phasic contractions characteristic of normal delivery. Oxytocin also promotes milk ejection (letdown) reflex in nursing mother, thereby increasing flow (not volume) of milk, and facilitates flow of milk during period of breast engorgement. Uterine sensitivity to oxytocin increases during gestation period and peaks sharply before parturition. It is used to induce labor in selected cases.

**Emergency Use:** To control postpartum hemorrhage. *Adult dose:* 3–10 units IM following delivery of the placenta; 10–20 units in 1000 mL of D$_5$W or NS IV infusion titrated to the severity of the bleeding. *Pediatric dose:* Not used.

**Pharmacokinetics**

*Absorption:* Onset is immediately IV; 3–7 min IM; duration is up to 1 hr IV, 2–3 hr IM; half-life is 3–5 min.

*Distribution:* Distributed throughout extracellular fluid; small amount may cross placenta.

*Metabolism:* Rapidly destroyed in liver and kidneys.

*Elimination:* Small amounts excreted unchanged in urine.

**Contraindications and Precautions:** Oxytocin is contraindicated prehospital prior to delivery of the baby and in patients with hypersensitivity to oxytocin. When administered prior to delivery, oxytocin may cause fetal hypoxia, fetal asphyxia, fetal dysrhythmias, and possible fetal intracranial bleeding.

**Adverse/Side Effects:** CNS: Subarachnoid hemorrhage, anxiety • CV: Postpartum hemorrhage, cardiac dysrhythmias, pelvic hematoma, hypotension, ECG changes, PVCs, precordial pain, edema, hypertensive episodes, cardiovascular spasm and collapse • Respiratory: Dyspnea • GI: Nausea, vomiting • Hypersensitivity: Hypersensitivity leading to uterine hypertonicity, tetanic contractions, uterine rupture, anaphylactic reactions • Metabolic: Antidiuretic hormone effects leading to severe water intoxication and hyponatremia • Skin: Cyanosis or redness of skin.

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**Oxytocin**
Interactions: Vasoconstrictors can cause severe hypertension.

Prehospital Considerations
• Ensure that the baby and placenta have been delivered and that there is not an additional fetus in the uterus.
• Oxytocin should never be administered by more than one route at a time.
• During delivery, IM oxytocin is most easily injected deep into deltoid muscle. Massage injection site to assist quick absorption.
• When diluting oxytocin for IV infusion, rotate bottle gently to distribute medicine throughout solution.
• IV preparation: For postpartum bleeding, add 10–40 U of oxytocin to 1 L of D5W or NS to give 10–40 mU/mL.
• Administer properly diluted IV solution by continuous infusion only.
• Unless otherwise directed by manufacturer, store oxytocin solution in refrigerator but do not freeze.
• The fundus should be checked frequently during the first few postpartum hours and several times daily thereafter.
• Incidence of hypersensitivity or allergic reactions is higher when oxytocin is given by IM or IV injection rather than by IV infusion (diluted solution).

PANCURONIUM BROMIDE
Class: Non-depolarizing neuromuscular blocker
Trade Name: Pavulon
Therapeutic Actions/Pharmacodynamics: Pancuronium is a synthetic derivative of curare used to facilitate endotracheal intubation. Pancuronium competes with acetylcholine at its receptor sites on the postsynaptic membrane. This results in paralysis of muscle fibers served by the neuromuscular junction. It is reported to be five times as potent as curare but produces little or no histamine release or ganglionic blockade and
thus does not cause bronchospasm or hypotension. It does not cause an initial depolarization wave as does succinylcholine.

**Emergency Use:** To facilitate endotracheal intubation. **Adult dose:** 0.04–0.1 mg/kg IV. **Pediatric dose:** Same as adult.

**Pharmacokinetics**

**Absorption:** Onset is 30–45 seconds; peak effect in 3–5 min; duration is 30–60 min; half-life is 2 hr.

**Distribution:** Well distributed to tissues and extracellular fluids; crosses placenta in small amounts.

**Metabolism:** Small amount metabolized in liver.

**Elimination:** Excreted primarily in urine.

**Contraindications and Precautions:** Pancuronium is contraindicated in patients with hypersensitivity to the drug or bromides. Use with caution in debilitated patients and those with myasthenia gravis, pulmonary, hepatic or renal disease, fluid or electrolyte imbalance.

**Adverse/Side Effects:** CNS: Skeletal muscle weakness, respiratory depression • CV: Increased pulse rate and BP, ventricular extrasystoles, burning sensation along course of vein • GI: Salivation.

**Interaction:** Lidocaine, procainamide, beta blockers, magnesium sulfate, quinidine, verapamil, and other neuromuscular blockers enhance the neuromuscular blocking action of pancuronium. Diuretics may increase or decrease neuromuscular blockade. Lithium prolongs duration of neuromuscular blockade. Narcotic analgesics possibly add to respiratory depression. Succinylcholine increases the onset and depth of neuromuscular blockade. Phenytoin may cause resistance to or reversal of neuromuscular blockade.

**Prehospital Considerations**

- Pancuronium bromide may be given by direct IV undiluted over 30–90 seconds.
- Refrigerate at 2–8°C (36–46°F). Do not freeze.
- Observe patient closely for residual muscle weakness and...
signs of respiratory distress during recovery period. Monitor BP and vital signs.
• Peripheral nerve stimulator may be used to assess the effects of pancuronium and to monitor restoration of neuromuscular function.
• Have resuscitative equipment available prior to administration.
• Remember that pancuronium, like other neuromuscular blockers, has no effect on mental status. Sedatives/hypnotics should be administered prior to administering pancuronium in patients who are not unconscious.

PHENOBARBITAL

Class: Anticonvulsant
Trade Name: Luminal

Therapeutic Actions/Pharmacodynamics: Phenobarbital is a long-acting barbiturate. The sedative and hypnotic effects of barbiturates appear to be due primarily to interference with impulse transmission of cerebral cortex by inhibition of the reticular activating system. Phenobarbital has no analgesic properties, and small doses may increase reaction to painful stimuli. CNS depression may range from mild sedation to coma, depending on dosage, route of administration, degree of nervous system excitability, and drug tolerance. Phenobarbital limits spread of seizure activity by increasing threshold for motor cortex stimuli. Barbiturates are habit forming.

Emergency Uses: To control seizures, status epilepticus, and acute anxiety attacks:
Adult dose: 100–300 mg slow IV/IM. Pediatric dose: 6–10 mg/kg slow IV/IM.

Pharmacokinetics
Absorption: Onset is 3–30 min IV; peak effect in less than 30 min IV; duration is 4–6 hr; half-life is 2–6 days.
Distribution: 20–45% protein bound; crosses placenta; enters breast milk. 

Metabolism: Oxidized in liver to inactivated metabolites. 

Elimination: Excreted in urine. 

Contraindications and Precautions: Phenobarbital is contraindicated in patients with sensitivity to barbiturates. Use with caution in patients with impaired hepatic, renal, cardiac, or respiratory function; history of allergies; elderly or debilitated patients; patients with fever; hyperthyroidism; diabetes mellitus or severe anemia; during labor and delivery; lactation; patients with borderline hypoadrenal function. 

Adverse/Side Effects: CNS: Somnolence, nightmares, insomnia, hangover, headache, anxiety, thinking abnormalities, dizziness, nystagmus, irritability, paradoxic excitement and exacerbation of hyperkinetic behavior (in children); confusion or depression or marked excitement (elderly or debilitated patients); ataxia, CNS depression, coma, and death • CV: Bradycardia, syncope, hypotension • Respiratory: Respiratory depression • GI: Nausea, vomiting, constipation, diarrhea, epigastric pain • Hypersensitivity: Rash, angioneurotic edema, fever, serum sickness, urticaria; hypoventilation, apnea, laryngospasm, circulatory collapse • Injection site (extravasation): Thrombosis, gangrene transient pain, tenderness, redness. IV: coughing, hiccuping, laryngospasm • Other: Liver damage, hypocalcemia. 

Interactions: Alcohol and other CNS depressants compound CNS depression. Phenobarbital may decrease absorption and increase metabolism of oral anticoagulants. It increases the metabolism of corticosteroids, oral contraceptives, anticonvulsants, and digitoxin, possibly decreasing their effects. Antidepressants potentiate the adverse effects of phenobarbital. 

Prehospital Considerations: • Administer IM deep into large muscle mass; volume should not exceed 5 mL at any one site.
• Commercially prepared solutions for injection (sodium phenobarbital) may be diluted with most IV infusion solutions. If not absolutely clear, discard.
• IV preparation: Slowly introduce sterile water for injection into ampule with sterile syringe. Use at least 10 mL of diluent. Rotate ampule to hasten dissolving drug (may take several minutes). If solution is not clear in 5 min or if a precipitate remains, discard.
• IV administration: No greater than 60 mg/min. Administer reconstituted IV solution no later than 30 min after preparation.
• Extravasation of IV phenobarbital may cause necrotic tissue changes that may necessitate skin grafting. Frequently check the injection site.
• Patients receiving large doses should be closely observed for at least 30 min to ensure that sedation is not excessive.
• Keep patient under constant observation when drug is administered IV, and record vital signs at least every hour or more often if indicated.
• Barbiturates do not have analgesic action, and they may be expected to produce restlessness when given to patients in pain.
• The elderly or debilitated patient and children sometimes have paradoxic response to barbiturate therapy, i.e., irritability, marked excitement (inappropriate tearfulness and aggression in children), depression, and confusion. Be alert to unexpected responses and report promptly. Protect the elderly patient from falling, irrational behavior, and effects of depression (anorexia, social withdrawal).
• Barbiturates increase the metabolism of many drugs, leading to decreased pharmacologic effects of those drugs. Whenever a barbiturate is added to an established regimen of another drug, close observation for changes in effectiveness of the first drug is essential, at least during early phase of barbiturate use.
PHENYTOIN

Class: Anticonvulsant
Trade Names: Dilantin, Phenyte

Therapeutic Actions/Pharmacodynamics: Phenytoin is a hydantoin derivative chemically related to phenobarbital. Its precise mechanism of anticonvulsant action is not known, but it appears to reduce the voltage, frequency, and spread of electrical discharges within the motor cortex, resulting in seizure activity inhibition. It also has class IB antidysrhythmic properties similar to those of lidocaine and tocainamide (also class IB agents). In abnormal tissue phenytoin causes a slight increase in AV conduction velocity depressed by digitalis glycosides, prolongs effective refractory period, suppresses ventricular pacemaker automaticity, and may slow conduction or cause complete block in abnormal ventricular fibers.

Emergency Uses: To control seizures, status epilepticus. Adult dose: 10–15 mg/kg slow IV. Pediatric dose: 8–10 mg/kg slow IV. To convert dysrhythmias induced by digitalis toxicity. Adult dose: 100 mg slow IV (over 5 min) to a maximum loading dose of 1000 mg. Pediatric dose: 3–5 mg/kg slow IV.

Pharmacokinetics
Absorption: Onset in 3–5 min; peak effect in 1–2 hr; half-life is 22 hr.
Distribution: 95% protein bound; crosses placenta; small amount in breast milk. Metabolism: Oxidized in liver to inactive metabolites.
Elimination: Metabolites excreted by kidneys.

Contraindications and Precautions: Phenytoin is contraindicated in patients with hypersensitivity to hydantoin products. It is also contraindicated in patients with seizures due to hypoglycemia, sinus bradycardia, complete or incomplete heart block, and Adams-Stokes syndrome. Use with caution in patients with impaired hepatic or renal function; alcoholism,
hypotension, heart block, brachycardia, severe myocardial insufficiency, impending or frank heart failure; elderly, debilitated, gravely ill patients; diabetes mellitus, hyperglycemia; respiratory depression.

**Adverse/Side Effects:** CNS: Nystagmus, drowsiness, ataxia, dizziness, mental confusion, tremors, insomnia, headache, seizures • CV: Bradycardia, hypotension, cardiovascular collapse, ventricular fibrillation, phlebitis • Eye: Photophobia, conjunctivitis, diplopia, blurred vision • GI: Nausea, vomiting, constipation, epigastric pain, dysphagia, loss of taste, weight loss, hepatitis, liver necrosis • Metabolic: Fever, hyperglycemia, glycosuria, weight gain, edema • Skin: Maculopapular, urticarial, or morbilliform rash; bullous, exfoliative, or purpuric dermatitis; keratosis; neonatal hemorrhage • Other: Acute renal failure, acute pneumonitis, pulmonary fibrosis, lymphadenopathy.

**Interactions:** Alcohol decreases phenytoin effects; other anticonvulsants may increase or decrease phenytoin levels. Phenytin may decrease absorption and increase metabolism of oral anticoagulants; phenytoin increases metabolism of corticosteroids, oral contraceptives, and nisoldipine, thus decreasing their effectiveness. Amiodarone increases phenytoin levels; antituberculosis agents decrease phenytoin levels.

**Prehospital Considerations**
- **IV administration:** Give by direct IV 50 mg or fraction thereof over 1 min (25 mg/min in elderly or when used as anti-dysrhythmic). Usually, phenytoin is not given as a continuous infusion.
- **During IV phenytoin administration,** observe injection site frequently to prevent infiltration. Local soft tissue irritation may be serious, leading to erosion of tissues. Elderly women, especially those with peripheral vascular disease, seem to be at high risk.
To minimize local venous irritation, each IV injection is followed with an injection of sterile saline through the same in-place catheter or needle.

To reduce side effects with IV administration, lower doses than the usual adult range are given to geriatric, severely ill, or debilitated patients and to those with liver damage, and the flow rate is reduced to 50 mg over a 2–3-min period.

A slightly yellowed injectable solution may be used safely. Precipitation may be caused by refrigeration, but slow warming to room temperature restores clarity. Do not administer unclear solution.

Store phenytoin at 15–30°C (59–86°F) in tightly closed container. Protect from light.

Margin between toxic and therapeutic IV doses is relatively small. Continuously monitor vital signs and symptoms during IV infusion and for an hour afterward. Watch for respiratory depression. If patient is elderly or has cardiac disease, constant observation and a cardiac monitor are necessary.

Observe patient closely for neurologic side effects. Have on hand oxygen, atropine, vasopressor, assisted ventilation, and seizure precaution equipment (mouth gag, nonmetal airway, suction apparatus).

Phenytoin must not be diluted with, or administered through, dextrose-containing solutions (i.e., D₅W).

**PHYSOSTIGMINE**

**Class:** Parasympathomimetic  
**Trade Name:** Antilirium  
**Therapeutic Actions/Pharmacodynamics:** Physostigmine inhibits cholinesterase from breaking down acetylcholine. This results in increased acetylcholine levels at the receptor sites and a prolonging of parasympathetic effects. It is used as an antidote for anticholinergic overdose from drugs such as...
atropine and scopolamine and from plants containing anti-cholinergic agents such as belladonna. Since toxic levels of tricyclic antidepressants also cause cholinesterase inhibition, physostigmine is sometimes given for tricyclic overdoses. Tricyclic overdoses can cause serious cardiac conduction disturbances, resulting in a variety of dysrhythmias. By inhibiting cholinesterase, physostigmine causes a decrease in cardiac automaticity and conductivity, mucus secretion, and pupillary constriction by directly affecting the autonomic ganglia.

**Emergency Uses:** To reverse CNS and cardiac effects of tricyclic antidepressant overdose, to reverse CNS toxic effects of atropine, scopolamine, and similar anticholinergic drugs. **Adult dose:** 0.5–3 mg IV (not faster than 1 mg/min); repeat as needed. **Pediatric dose:** 0.01–0.03 mg/kg IV; may repeat every 15–20 min to maximum total dose of 2 mg.

**Pharmacokinetics**

*Absorption:* Readily absorbed from mucous membranes, muscle, subcutaneous tissue; onset is 3–8 min; duration is 0.5–5.0 hr; half-life is 15–40 min.

*Distribution:* Crosses blood-brain barrier.

*Metabolism:* Metabolized in plasma by cholinesterases.

*Elimination:* Excretion not fully understood; small amounts excreted in urine.

**Contraindications and Precautions:** Physostigmine is contraindicated in asthma, diabetes mellitus, gangrene, cardiovascular disease, and narrow-angle glaucoma. If excessive parasympathetic actions are seen, such as increased salivation, emesis, or bradycardia, reduce the dosage or administer atropine to antagonize the effects.

**Adverse/Side Effects:** Acute toxicity: Cholinergic crisis
- **CNS:** Restlessness, hallucinations, twitching, tremors, sweating, weakness, ataxia, convulsions, collapse, headache
- **Respiratory:** Respiratory paralysis, pulmonary edema. **CV:** Bradycardia, hypotension
- **Eye:** Constricted pupils, twitching
of eyelids, lacrimation, dimness and blurring of vision • GI: Increased urination and defecation.

**Interactions:** Anticholinergic drugs such as atropine, antidepressants, antihistamines, and phenothiazines can antagonize the effects of physostigmine.

**Prehospital Considerations**
- Physostigmine is rarely required in the prehospital setting.
- Physostigmine is given by direct IV undiluted at a slow rate, no more than 1 mg/min. Rapid administration and over-dosage can cause a cholinergic crisis.
- Monitor vital signs and state of consciousness in patients receiving drug for atropine poisoning. Since physostigmine is usually rapidly destroyed, patient can lapse into delirium and coma within 1 to 2 hr; repeat doses may be required.
- Monitor closely for side effects related to CNS and for signs of sensitivity to physostigmine. Have atropine sulfate readily available for clinical emergency.
- When used parenterally or orally, the following symptoms indicate need to discontinue drug: excessive salivation, emesis, frequent urination, or diarrhea. Excessive sweating or nausea may be eliminated by dose reduction.

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**PRLIDOXIME**

**Class:** Cholinesterase reactivator

**Trade Names:** 2-PAM, Protopam Chloride

**Therapeutic Actions/Pharmacodynamics:** Pralidoxime reactivates the cholinesterase inhibited by phosphate esters by displacing the enzyme from its receptor sites. The free enzyme then can resume its function of degrading accumulated acetylcholine, thereby restoring normal neuromuscular transmission. Since it is more active against effects of anticholinesterase at the skeletal neuromuscular junction than at autonomic effector...
sites or in CNS respiratory center, atropine must be given concomitantly to block effects of acetylcholine and accumulation in these sites. Pralidoxime also detoxifies some organophosphates by direct chemical reaction. It is saved for severe organophosphate poisonings to reverse the respiratory depression and skeletal muscle paralysis.

**Emergency Use:** As antidote in treatment of poisoning by organophosphate. *Adult dose:* 1–2 g in 250–500 mL NS infused over 15–30 min; or 1–2 g IM/SC if IV not feasible. *Pediatric dose:* 20–40 mg/kg IV/IM/SC.

**Pharmacokinetics**

*Absorption:* Peak effect in 5–15 min IV; 10–20 min IM; half-life: 0.8–2.7 hr.

*Distribution:* Distributed throughout extracellular fluids; crosses blood-brain barrier slowly if at all.

*Metabolism:* Probably metabolized in liver.

*Elimination:* Rapidly excreted in urine.

**Contraindications and Precautions:** Pralidoxime is contraindicated in poisonings by the carbamate insecticide Sevin, inorganic phosphates, or organophosphates having no anticholinesterase activity. Do not use in patients with asthma, peptic ulcer, severe cardiac disease, or in patients receiving aminophylline, theophylline, morphine, succinylcholine, reserpine, or phenothiazines. Rapid IV administration may result in tachycardia, laryngospasm, and muscle rigidity. If used with large atropine doses, the effects of atropine may be seen much earlier than expected. Excitement and manic behavior may occur in patients immediately following recovery from unconsciousness.

**Adverse/Side Effects:** CNS: Dizziness, headache, drowsiness, muscular weakness • Eye: Blurred vision, diplopia, impaired accommodation • CV: Tachycardia, hypertension • GI: Nausea • With rapid IV: Tachycardia, laryngospasm, muscle rigidity.

**Interactions:** Respiratory depressants can potentiate the effects of pralidoxime. These include narcotics, phenothiazines, anti-
histamines, and alcohol. Pralidoxime should not be used with theophylline preparations.

**Prehospital Considerations**

- The initial treatment of organophosphate poisoning is atropinization. Following adequate atropinization, and in severe overdoses, pralidoxime should be considered.
- Always protect yourself and other rescuers when caring for the victims of organophosphate poisoning.
- Infusion should be stopped or IV rate reduced if hypertension occurs.
- It is difficult to differentiate toxic effects of organophosphates or atropine from toxic effects of pralidoxime. Be alert for these signs and report them immediately: reduction in muscle strength, onset of muscle twitching, changes in respiratory pattern, altered level of consciousness, increases or changes in heart rate and rhythm.
- Excitement and manic behavior reportedly may occur following recovery of consciousness. Observe necessary safety precautions.
- Pralidoxime is relatively short-acting. In patients with myasthenia gravis, overdosage with pralidoxime may convert cholinergic crisis into myasthenic crisis.

**PROCAINAMIDE**

**Class:** Antidyssrhythmic  
**Trade Names:** Procan, Procanbid, Promine, Pronestyl  
**Therapeutic Actions/Pharmacodynamics:** Procainamide is an amide analog of procaine hydrochloride with cardiac actions similar to those of quinine. It is a Class Ia antidysrhythmic agent that depresses the excitability of the myocardium to electrical stimulation, and reduces conduction velocity in the atria, ventricles, and His-Purkinje system. Procainamide increases
the duration of the refractory period, especially in the atria. It also produces a slight change in contractility of cardiac muscle and cardiac output and suppresses automaticity of His-Purkinje ventricular muscle. It can produce peripheral vasodilation and hypotension, especially with IV use.

**Emergency Uses:** To convert ventricular fibrillation and pulseless ventricular tachycardia refractory to lidocaine to sinus rhythm. **Adult dose:** 20–30 mg/min IV infusion up to 17 mg/kg loading dose. Maintenance dose is 1–4 mg/min. **Pediatric dose:** 15 mg/kg IV/IO over 30–60 min.

**Pharmacokinetics**

**Absorption:** Onset is 10–30 min; peak effect in 15–60 min; duration is 3–6 hr; half-life is 3 hr.

**Distribution:** Distributed to CSF, liver, spleen, kidney, brain, and heart; crosses placenta; distributed into breast milk.

**Metabolism:** Metabolized in liver to N-acetylprocainamide (NAPA), an active metabolite (30–60% metabolized to NAPA).

**Elimination:** Excreted in urine.

**Contraindications and Precautions:** Procainamide is contraindicated in myasthenia gravis; hypersensitivity to procainamide or procaine; complete AV block, second and third degree AV block unassisted by pacemaker. Use with caution in patients with hypotension, cardiac enlargement, CHF, MI, coronary occlusion, ventricular dysrhythmia from digitalis intoxication, hepatic or renal insufficiency, electrolyte imbalance, bronchial asthma.

**Adverse/Side Effects:** CNS: Dizziness, psychosis • CV: Severe hypotension, pericarditis, ventricular fibrillation, AV block, tachycardia, flushing • GI: Nausea, vomiting, diarrhea, anorexia • Hematologic: thrombocytopenia • Hypersensitivity: Fever, muscle and joint pain, angioneurotic edema, maculopapular rash, pruritus • Other: Pleuritic pain, pleural effusion, erythema, skin rash, myalgia, fever.

**Interactions:** Other antidysrhythmics add to therapeutic and
toxic effects; anticholinergic agents compound anticholinergic effects; antihypertensives add to hypotensive effects; cimetidine may increase procainamide and NAPA levels with increase in toxicity.

**Prehospital Considerations**

- When procainamide is given direct IV, dilute each 100 mg with 10 mL of D$_5$W or sterile water for injection. When procainamide is given by IV infusion, add 1 g of procainamide to 250–500 mL of D$_5$W solution. Yields 4 mg/mL in 250 mL or 2 mg/mL in 500 mL.
- Procainamide administration by IV infusion pump requires constant monitoring to maintain desired flow rate. Keep patient in supine position. Be alert to signs of too rapid administration: irregular pulse, tight feeling in chest, flushed face, headache, loss of consciousness, shock, cardiac arrest.
- Procainamide solution is stable for 24 hr at room temperature and for 7 days under refrigeration at 2–8°C (36–46°F). Avoid freezing the solution. Refrigeration will retard color changes in solution. Slight yellowing does not alter drug potency, but discard solution if it is markedly discolored or precipitated.
- Patients at particular risk for adverse effects are those with severe heart, hepatic, or renal disease and hypotension.
- Monitor the patient’s ECG and BP continuously during IV drug administration.
- IV drug is temporarily discontinued when dysrhythmia is interrupted; severe toxic effects are present; QRS complex is excessively widened (greater than 50%); PR interval is prolonged; or BP drops 15 mm Hg or more. Obtain rhythm strip and notify physician.
PROCHLORPERAZINE

Class: Antiemetic
Trade Name: Compazine, Prochlorparazine (Can), Prorazin (Can), Stemetil (Can)

Therapeutic Actions/Pharmacodynamics: Prochlorperazine is a phenothiazine derivative with actions, contraindications, and interactions similar to those of chlorpromazine. It has greater extrapyramidal effects and antiemetic potency but fewer sedative, hypotensive, and anticholinergic effects than chlorpromazine. It does not prevent vertigo and motion sickness like many of the other phenothiazines. In addition, prochlorperazine has weak anticholinergic properties.

Emergency Uses: To relieve severe nausea and vomiting; to manage acute psychosis. Adult dose: 5–10 mg IV/IM. Pediatric dose: More than 10 kg or older than 2 yr: 0.13 mg/kg IV/IM/PR (rarely used).

Pharmacokinetics
Absorption: Onset is 10–20 min IV/IM, 60 min PR; duration is up to 12 hr IM, 3–4 hr PR.
Distribution: Crosses placenta; distributed into breast milk.
Metabolism: Metabolized in liver.
Elimination: Excreted in urine.

Contraindications and Precautions: Prochlorperazine is contraindicated in patients with hypersensitivity to phenothiazines and in patients who are comatose or severely depressed. Use with caution in patients with previously diagnosed breast cancer, and in children with acute illness or dehydration.

Adverse/Side Effects: CNS: Drowsiness, dizziness, extrapyramidal reactions (akathisia, dystonia, or parkinsonism), persistent tardive dyskinesia, acute catatonia, sedation • CV: Hypotension, tachycardia • Eyes: Photosensitivity, blurred vision • Skin: Cholestatic jaundice.
Interactions: Alcohol and other CNS depressants increase CNS depression. Phenobarbital increases the metabolism of prochlorperazine. Concomitant administration of phenylpropanolamine poses the possibility of sudden death. Tricyclic antidepressants intensify its hypotensive and anticholinergic effects and decrease the seizure threshold, so the anticonvulsant dosage may need to be increased.

Prehospital Considerations

- The incidence of extrapyramidal symptoms appears to be higher with prochlorperazine than with other phenothiazines. Have diphendydramine available prior to administration.
- Dosage for elderly, emaciated patients and for children should be advanced slowly.
- Avoid skin contact with oral concentrate or injection solution because of possibility of contact dermatitis.
- IM injection in adults should be made deep into the upper outer quadrant of the buttock. Do not mix IM solution in the same syringe with other agents. Follow agency policy regarding IM injection site for children.
- IV administration: Give direct IV by diluting in D5W, NS, or other compatible diluent to a concentration of 1 mg/mL and give at a maximum rate of 5 mg/min.
- Slight yellowing does not appear to alter potency; however, markedly discolored solutions should be discarded. Protect drug from light; do not freeze. Store at temperature between 15 and 30°C (59 and 86°F) unless otherwise instructed by manufacturer.
- Postoperative patients who have received prochlorperazine may have depressed cough reflex and should be carefully positioned to prevent aspiration of vomitus.
- Most elderly and emaciated patients and children, especially those with dehydration or acute illness, appear to be particularly susceptible to extrapyramidal effects. Be alert to onset
of symptoms: in early therapy watch for pseudoparkinson’s and acute dyskinesia.

- Keep in mind that the antiemetic effect may mask toxicity of other drugs or make it difficult to diagnose conditions with a primary symptom of nausea, such as intestinal obstruction and increased intracranial pressure.
- It has been reported that although the patient is not responsive during acute catatonia (side effect), everything that happens during the episode can be recalled. Approach patient accordingly.
- Exposure to high environmental temperature, to sun’s rays, or to a high fever associated with serious illness places this patient at risk for heat stroke. Be alert to signs: red, dry, hot skin; full bounding pulse; dilated pupils; dyspnea; confusion; temperature over 40.6°C (105°F); elevated BP. Inform physician and institute measures to reduce body temperature rapidly.
- Prochlorperazine is rarely used in children because of their tendency to develop extrapyramidal system symptoms with even low dosages.

**PROMETHAZINE**

**Class:** Antiemetic

**Trade Names:** Anergan, Histantil (Can), Pentazine, Phenameth, Phenergan, Phenoject-50, Promethegan, Prorex, Prothazine, V-Gan

**Therapeutic Actions/Pharmacodynamics:** Promethazine is a long-acting derivative of phenothiazine with marked antihistaminic activity and prominent sedative, amnesic, antiemetic, and anti-motion sickness actions. Unlike other phenothiazine derivatives, it is relatively free of extrapyramidal side effects; however, in high doses it carries same potential for toxicity. In common with other antihistamines, exerts antiserotonin, anticholinergic, and local anesthetic action. Antiemetic action
thought to be due to depression of CTZ in medulla.

Emergency Uses: To relieve nausea and vomiting, motion sickness; to potentiate the effects of analgesics; to induce sedation. Adult dose: 12.5–25.0 mg IV/IM/PR. Pediatric dose: 0.5 mg/kg IV/IM/PR.

Pharmacokinetics
Absorption: Onset is 20 min PR/IM; 5 min IV; duration is 2–8 hr.
Distribution: Crosses placenta.
Metabolism: Metabolized in liver.
Elimination: Slowly excreted in urine and feces.

Contraindications and Precautions: Promethazine is contraindicated in patients with hypersensitivity to phenothiazines, nursing mothers, newborn or premature infants, acutely ill or dehydrated children. Use with caution in patients with impaired hepatic function, cardiovascular disease, asthma, acute or chronic respiratory impairment (particularly in children), hypertension; elderly or debilitated patients.

Adverse/Side Effects: Acute toxicity: Deep sleep, coma, convulsions, cardiorespiratory symptoms, extrapyramidal reactions, nightmares (in children), CNS stimulation, abnormal movements, respiratory depression. Toxic potential as for other phenothiazines • CNS: Sedation drowsiness, confusion, dizziness, disturbed coordination, restlessness, tremors • CV: Transient mild hypotension or hypertension • GI: Anorexia, nausea, vomiting, constipation • Other: Photosensitivity, irregular respiration, blurred vision, urinary retention; dry mouth, nose, or throat.

Interactions: Alcohol and other CNS depressants add to CNS depression and anticholinergic effects.

Prehospital Considerations
• Inspect parenteral drug before preparation. Discard if it is darkened or contains precipitate.
• IM injection is made deep into large muscle mass. Aspirate carefully before injecting drug. Intra-arterial injection can
cause arterial or arteriolar spasm, with resultant gangrene. Subcutaneous injection (also contraindicated) can cause chemical irritation and necrosis. Rotate injection sites and observe daily.

- IV administration: IV promethazine in concentrations of 25 mg/mL or less may be given by direct IV undiluted over 2 min. More concentrated preparations should be diluted in NS to yield no more than 25 mg/mL.
- Store in tight, light-resistant container at 15–30°C (59–86°F) unless otherwise directed.
- Antiemetic action may mask symptoms of unrecognized disease and signs of drug overdosage as well as dizziness, vertigo, or tinnitus associated with toxic doses of aspirin or other ototoxic drugs.
- Patients in pain may develop involuntary movements of upper extremities following parenteral administration. These symptoms usually disappear after pain is controlled.
- Respiratory function should be monitored in patients with respiratory problems, particularly children. Promethazine may suppress cough reflex and cause thickening of bronchial secretions.
- Have diphenhydramine available in case extrapyramidal symptoms appear.

**PROPAFANONE**

**Class:** Antidysrhythmic  
**Trade Name:** Rythmol  
**Therapeutic Actions/Pharmacodynamics:** Propafanone is a class IC antidysrhythmic drug with a direct stabilizing action on myocardial membranes. It reduces spontaneous automaticity. The rate of single and multiple PVCs is decreased by appropriate dose and concentration of propafanone. In addition, it suppresses ventricular tachycardia. Propafanone, like other class IC
antidysrhythmics, exerts a negative inotropic effect on the myocardium. It also has nonselctive beta-blocking properties.

**Emergency Uses:** To convert ventricular and supraventricular dysrhythmias in patients without structural heart disease. *Adult dose:* 150–300 mg PO every 8 hr. IV dose is 1–2 mg/kg administered at 10 mg/min.

**Pharmacokinetics**
*Absorption:* Readily absorbed from GI tract; peak effect in 3.5 hr; half-life is 5–8 hr.
*Distribution:* 97% protein bound, highest concentrations in the lung. Crosses placenta, distributed into breast milk.
*Metabolism:* Extensively metabolized in the liver.
*Elimination:* 18.5–38% of dose excreted in urine as metabolites.

**Contraindications and Precautions:** Propafenone is contraindicated in patients with uncontrolled CHF, cardiogenic shock, sinoatrial, AV or intraventricular disorders (e.g., sick sinus node syndrome, AV block) without a pacemaker; bradyarrhythmias, marked hypotension, bronchospastic disorders; electrolyte imbalances; hypersensitivity to propafenone; non-life-threatening dysrhythmias, chronic bronchitis, emphysema, nursing mothers. Use with caution in patients with CHF, AV block, hepatic/renal impairment; in elderly patients; and during pregnancy. Safety and efficacy in children have not been established.

**Adverse/Side Effects:**
*CNS:* Blurred vision, dizziness, paresthesias, fatigue, somnolence, vertigo, and headache • *CV:* arrhythmias, ventricular tachycardia, hypotension, bundle branch block, AV block, complete heart block, sinus arrest, CHF, bradycardia • *GI:* Nausea, abdominal discomfort, constipation, vomiting, dry mouth, taste alterations, cholestatic hepatitis • *Other:* Rash.

**Interactions:** Amiodarone and quinidine increase the levels and toxicity of propafenone. May increase levels and toxicity
of tricyclic antidepressants, cyclosporine, digoxin, beta blockers, theophylline, and warfarin; may increase levels of both propafenone and diltiazem. Phenobarbital decreases levels of propafenone.

**Prehospital Considerations**
- IV use of propafenone is not approved in the United States.
- Dosage is usually initiated with 150 mg every 8 hr and may be increased at 3–4 days intervals to a maximum of 300 mg every 8 hr.
- Dosage increments should be more gradual in elderly patients or in those with previous extensive myocardial damage.
- Dosage reduction should be considered with significant widening of the QRS complex or development of second- or third-degree AV block.
- With severe liver dysfunction, significant dose reduction is warranted.
- Monitor cardiovascular status frequently (e.g., ECG, holter monitor) to determine effectiveness of drug and development of new or worsened arrhythmias.
- Development of second- or third-degree AV block after initiation of therapy requires dosage reduction or discontinuation of the drug.
- Advise patients to report any of the following: chest pain, palpitations, blurred or abnormal vision, dyspnea, or signs and symptoms of infection.
- Advise patients on concurrent warfarin therapy to report unusual bleeding or bruising.
- Instruct patients to monitor radial pulse daily and report decreased heart rate or development of an abnormal heartbeat.
- Alert elderly or debilitated patients to possibility of developing dizziness and need for caution with ambulation.
PROPRANOLOL

Class: Beta blocker

Trade Names: Apo-Propranolol (Can), Betachron, Deralin (Aus), Detensol (Can), Inderal, Novopranol (Can)

Therapeutic Actions/Pharmacodynamics: Propranolol is a nonselective beta blocker of both cardiac and bronchial adrenoreceptors that competes with epinephrine and norepinephrine for available beta-receptor sites. It blocks cardiac effects of beta-adrenergic stimulation; as a result, reduces heart rate, myocardial irritability (Class II antidysrhythmic) and force of contraction, depresses automaticity of sinus node and ectopic pacemaker, and decreases AV and intraventricular conduction velocity. In higher doses, it exerts direct quinidine-like effects that depress cardiac function. Propranolol lowers both supine and standing blood pressures in hypertensive patients. Its hypotensive effect is associated with decreased cardiac output and suppressed renin activity, as well as beta blockade. It also decreases platelet aggregability. The mechanism of antimigraine action unknown but thought to be related to inhibition of cerebral vasodilation and arteriolar spasms.

Emergency Uses: To convert ventricular fibrillation and pulseless ventricular tachycardia refractory to lidocaine and bretylium; to convert selected supraventricular tachydysrhythmias.

Adult dose: 1–3 mg slow IV (over 2–5 min), not to exceed 1 mg/min. May repeat dose in 2 min to total dose of 0.1 mg/kg.

Pediatric dose: 0.01 mg/kg slow IV.

Pharmacokinetics

Absorption: Onset in less than 2 min; peak effect in 15 min; duration of 2–6 hr; half-life is 2.3 hr.

Distribution: Widely distributed including CNS, placenta, and breast milk.

Metabolism: Almost completely metabolized in liver.

Elimination: 90–95% excreted in urine as metabolites; 1–4% excreted in feces.
Contraindications and Precautions: Propranolol is contraindicated in patients with greater than first-degree heart block, CHF, right ventricular failure secondary to pulmonary hypertension, sinus bradycardia, cardiogenic shock, significant aortic or mitral valvular disease, bronchial asthma or bronchospasm, severe COPD, allergic rhinitis during pollen season. Avoid concurrent use with adrenergic-augmenting psychotropic drugs or within 2 wk of MAO inhibition therapy. Safe use during pregnancy (category C) and in nursing mothers not established. Use with caution in patients with peripheral arterial insufficiency; history of systemic insect sting reaction; patients prone to nonallergenic bronchospasm (e.g., chronic bronchitis, emphysema); major surgery; renal or hepatic impairment; diabetes mellitus; patients prone to hypoglycemia; myasthenia gravis; Wolff-Parkinson-White syndrome.

Adverse/Side Effects: Allergic: Erythematous, psoriasis-like eruptions; pruritus; fever; pharyngitis; respiratory distress
  • CNS: Drug-induced psychosis, sleep disturbances, depression, confusion, agitation, giddiness, light-headedness, fatigue, vertigo, syncope, weakness, drowsiness, insomnia, vivid dreams, visual hallucinations, delusions, reversible organic brain syndrome
  • CV: Palpitation, profound bradycardia, AV heart block, cardiac standstill, hypotension, angina pectoris, tachydyssrhythmia, acute CHF, peripheral arterial insufficiency resembling Raynaud’s disease, myotonia, paresthesia of hands
  • Eye/ear: Dry eyes (gritty sensation), visual disturbances, conjunctivitis, tinnitus, hearing loss, nasal stuffiness
  • GI: Dry mouth, cheilostomatitis, nausea, vomiting, heartburn, diarrhea, constipation, flatulence, abdominal cramps, mesenteric arterial thrombosis, ischemic colitis. Hematologic: Transient eosinophilia, hypoglycemia, hyperglycemia, hypocalcemia (patients with hyperthyroidism)
  • Respiratory: Dyspnea, laryngospasm, bronchospasm
  • Skin: Hyperkeratoses of scalp, palms, feet; nail changes; dry skin
  • Other: Pancreatitis, weight gain, impotence or decreased libido, cold extremities, leg fatigue, arthralgia.
**Interactions:** Phenothiazines have additive hypotensive effects. Beta-adrenergic agonists (e.g., albuterol) antagonize effects. Atropine and tricyclic antidepressants block bradycardia. Diuretics and other hypotensive agents increase hypotension. High doses of tubocurarine may potentiate neuromuscular blockade. Cimetidine decreases clearance, increases effects. Antacids may decrease absorption.

**Prehospital Considerations**
- IV administration: For direct IV, give each 1 mg over 1 min either undiluted or diluted in 10 mL of 5% dextrose. For intermittent infusion, further dilute in 50 mL of normal saline solution and give over 15–20 min.
- Preserve in tightly closed, light-resistant containers at 15–30°C (59–86°F).
- Take apical pulse and BP before administering drug.
- Withhold drug if heart rate is less than 60 bpm or systolic BP is greater than 90 mm Hg.
- Careful medical history and physical examination are essential to rule out allergies, asthma, and other obstructive pulmonary disease. Propranolol can cause bronchiolar constriction even in normal subjects.
- Bradycardia is the most common adverse cardiac effect especially in patients with digitalis intoxication and Wolff-Parkinson-White syndrome.
- When propranolol is administered IV, ECG, and BP must be carefully monitored. Reduction in sympathetic stimulation caused by beta-blocking action can result in cardiac standstill.
- Adverse reactions generally occur most frequently following IV administration; however, incidence is also high following oral use in the elderly and in patients with impaired renal function. Reactions may or may not be dose related and commonly occur soon after therapy is initiated.
PROSTAGLANDIN E₁

Class: Vasodilator
Trade Name: Prostin VR Pediatric

Therapeutic Actions/Pharmacodynamics: Prostaglandin E₁, also called alprostadil, is a compound derived from fatty acids that is found in several body cells. It causes vasodilation, inhibits platelet aggregation, and stimulates intestinal and uterine smooth muscle. Immediately following birth, the ductus arteriosus begins to close as blood is diverted to the pulmonary circulation. Complete closure usually occurs within one to several weeks. Some neonates and infants may suffer from one of several congenital heart diseases. Certain congenital disorders depend upon a patent ductus arteriosus to prevent hypoxemia or systemic hypoperfusion. Infants with congenital heart disease may develop hypoxemia and congestive heart failure as the ductus closes. In these cases, IV administration of prostaglandin E₁ can keep the ductus open until a surgical repair can be made.

Prostaglandin E₁ can also be used to “ripen” the cervix and induce labor due to its effects on uterine smooth muscle.

Emergency Uses: To maintain patency of the ductus arteriosus in infants with cyanotic congenital heart disease who are dependent upon a patent ductus for life. Pediatric dose: Prostaglandin E₁ is administered IV or IO at 0.05–0.10 µg/kg per min. Prostaglandin E₁ is used to ripen the cervix in anticipation of delivery and induces labor. Adult dose: A gel is prepared by pharmacy staff that is placed over the cervix in diaphragm. This is replaced every 2–3 hr. The patient should be monitored for fetal heart rate and contractions.

Pharmacokinetics Absorption: Slow through vaginal mucosa. No absorption for IV dosing. Approximately 68% of circulating prostaglandin E₁ is metabolized in one pass through the lungs, and the metabolites are excreted through the kidney. Excretion is complete within 24 hr.
Contraindications and Precautions: Prostaglandin E₁ is contraindicated in any pregnancy in which the fetus is not ready for extrauterine life. Apnea occurs in 10–12% of patients. Constant respiratory monitoring, including pulse oximetry, should be utilized.

Adverse/Side Effects: CV: Flushing, bradycardia, hypotension, tachycardia, CHF, conduction disturbances, and dysrhythmias • CNS: Seizures, hyperpyrexia, cerebral bleeding, hyperextension of the neck, hypothermia, jitteriness, and lethargy • GI: Diarrhea and gastric regurgitation • Respiratory: Apnea (usually appears in first hr and most common in infants weighing less than 2 kg at birth; bradypnea, wheezing, hypercapnea, respiratory depression, and tachypnea.

Interactions: Prostaglandin E₁ should be monitored throughout infusion and stopped immediately if worrisome symptoms develop.

Prehospital Considerations
• IV prostaglandin E₁ will be almost exclusively used in pediatric critical transport situations involving an infant with a congenital heart condition.
• The IV infusion can be prepared by placing 500 µg in D5W or NS.
• Smallest effective dose should be used.
• The drug should be kept refrigerated prior to use.
• Carefully monitor cardiac status, including ECG, blood pressure, pulse and respiratory rate through course of therapy.
• Be sure to administer supplemental oxygen to infants who are dependent on a patent ductus.

RACEMIC EPINEPHRINE
Class: Sympathomimetic
Trade Names: MicroNEFRIN, Vaponephrine
Therapeutic Actions/Pharmacodynamics: Racemic epinephrine is slightly different chemically from the epinephrine compounds that have been discussed previously. Compounds that differ only in their chemical arrangement are called isomers. This particular form is frequently used in children to treat croup. Racemic ephnephrine stimulates both alpha- and beta-adrenergic receptors, with a slight preference for the beta2 receptors in the lungs. This results in bronchodilation and a decrease in mucus secretion. It also has some effect in relieving the subglottic edema associated with croup when administered by inhalation.

Emergency Use: To relieve subglottic edema in croup (laryngotracheobronchitis). Adult dose: 0.25–0.75 mL of 2.25 solution in 2 mL normal saline via nebulizer. Pediatric dose: Same as adult.

Contraindications and Precautions: Racemic epinephrine is contraindicated in patients with a hypersensitivity to the drug, those with potential for tachydysrhythmias and hypertension, and epiglottitis.

Adverse/Side Effects: CNS: Restlessness, tremors, anxiety, nervousness, dizziness, headache • CV: Tachycardia, hypertension, palpitation, angina, dysrhythmias • Respiratory: Bronchial edema/inflammation, paradoxical bronchospasm • GI: Nausea, vomiting.

Prehospital Considerations
• Monitor vital signs prior to, during, and following administration.
• Many patients will experience rebound worsening 30–60 min after the initial treatment and the effects of the drug have worn off. For this reason, transport all patients to the hospital. Most hospitals have the policy that all children who have received racemic epinephrine will be admitted for at least 24 hr for observation.
SODIUM BICARBONATE (NaHCO₃)

Class: Electrolyte

Trade Name: Sodium bicarbonate

Therapeutic Actions/Pharmacodynamics: Sodium bicarbonate is a short-acting, potent, systemic antacid. Given IV, it immediately raises the pH of blood plasma by buffering excess hydrogen ions (acidosis). In a short time, the plasma alkali reserve is increased and excess sodium and bicarbonate ions are excreted in urine, thus rendering the urine less acidic. This effect plays an important role in treating certain drug overdoses, particularly tricyclic antidepressants and barbiturates, by speeding excretion of the drug from the body. The role of sodium bicarbonate is limited in cardiac arrest. Because ventilation is an effective tool in managing respiratory acidosis, sodium bicarbonate should only be considered in a prolonged cardiac arrest after adequate airway and ventilation have been accomplished. It is considered acceptable if the arrested patient has a pre-existing hyperkalemia, a pre-existing bicarbonate-responsive acidosis, or a tricyclic antidepressant overdose.

Emergency Uses: To alkalinize the urine to enhance excretion of drug overdose (tricyclic antidepressants, barbiturates); to correct severe acidosis refractory to hyperventilation; known hyperkalemia. Adult dose: 1 mEq/kg IV, may repeat at half dose every 10 min. Pediatric dose: Same as adult. Can be given IO.

Pharmacokinetics

Absorption: Immediate absorption if given IV; onset is less than 15 min, duration is 1−2 hr.

Elimination: Excreted in urine within 3−4 hr.

Contraindications and Precautions: There are no absolute contraindications to using sodium bicarbonate in the above situations. When administered in large quantities, it can cause a metabolic alkalosis. Always calculate the dose based on the patient’s weight.
**Sodium Nitroprusside**

**Adverse/Side Effects:** Sodium bicarbonate may inhibit oxygen release secondary to a shift in oxyhemoglobin saturation. It also may produce a paradoxical acidosis that can depress cerebral and cardiac function. Sodium bicarbonate may cause extracellular alkalosis, which may reduce the concentration of ionized calcium, decrease plasma potassium, induce a left shift on the oxyhemoglobin dissociation curve, and induce malignant arrhythmias. Severe tissue damage if extravasated.

**Interactions:** Most catecholamines and vasopressors (dopamine, epinephrine) can be deactivated by alkaline solutions like sodium bicarbonate. When administered with calcium chloride, a precipitate may form that will clog the IV line.

**Prehospital Considerations**
- Infusion should be stopped immediately if extravasation occurs. Severe tissue damage has followed tissue infiltration.
- Always flush the IV line following sodium bicarbonate administration, especially in cardiac arrest.

**SODIUM NITROPRUSSIDE**

**Class:** Nitrate

**Trade Names:** Nipride, Nitropress

**Therapeutic Action/Pharmacodynamics:** Sodium Nitroprusside is a potent, rapidly acting hypotensive agent with effects similar to those of the nitrates. It acts directly on vascular smooth muscle to produce peripheral vasodilation, with consequent marked lowering of arterial BP, associated with slight increase in heart rate, mild decrease in cardiac output, and moderate lowering of peripheral vascular resistance.

**Emergency Uses:** To reduce blood pressure in an acute hypertensive crisis. **Adult dose:** 0.5–10 µg/kg/min IV infusion. **Pediatric dose:** Same as adult.
**Pharmacokinetics**

*Absorption:* Onset is less than 1 min; peak effect 1–5 min; duration is 1–10 min; half-life is 2.7–7 days.

*Metabolism:* Rapidly converted to cyanogen in erythrocytes and tissue, which is metabolized to thiocyanate in liver.

*Elimination:* Excreted in urine primarily as thiocyanate.

**Contraindications and Precautions:** Sodium nitroprusside is contraindicated in patients with compensatory hypertension, as in atriovenous shunt or coarctation of aorta, and for control of hypertension in patients with inadequate cerebral circulation. Safe use during pregnancy (category C) not established. Use with caution in patients with hepatic insufficiency, hypothyroidism, severe renal impairment, hyponatremia.

**Adverse/Side Effects:** CNS: Headache, dizziness, apprehension, restlessness, muscle twitching • CV: Profound hypotension, retrosternal discomfort, palpitation, increased or transient lowering of pulse rate, bradycardia, tachycardia, ECG changes • GI: Nausea, retching, abdominal pain • Other: Nasal stuffiness, diaphoresis. • Overdosage or prolonged use (more than 48 hr): thiocyanate toxicity: profound hypotension, tinnitus, blurred vision, fatigue, metabolic acidosis, pink skin color, absence of reflexes, faint heart sounds, loss of consciousness.

**Interactions:** The effects of nitroprusside can be potentiated when administered with other antihypertensive agents.

**Prehospital Considerations**

- Solutions must be freshly prepared with D5W and used no later than 4 hr after reconstitution.
- IV nitroprusside is diluted by dissolving 50 mg in 2–3 mL of D5W and then further diluted in 250 mL D5W (200 µg/mL) or 500 mL D5W (100 µg/mL).
- No other drug should be added to sodium nitroprusside infusion.
- Following reconstitution, solutions usually have faint brown-
ish tint; if solution is highly colored, do not use it. Promptly wrap container with aluminum foil or other opaque material to protect drug from light.
- Administer by infusion pump or similar device that will allow precise measurement of flow rate required to lower BP.
- Protect drug from light, heat, and moisture; store at 15–30°C (59–86°F) unless otherwise directed.
- Constant monitoring is required to titrate IV infusion rate to BP response.
- Adverse effects are usually relieved by slowing the IV rate or by stopping drug; they may be minimized by keeping patient supine.
- If BP begins to rise after drug infusion rate is decreased or infusion is discontinued, notify physician immediately.

SOTALOL
Classes: Beta blocker; antidysrhythmic
Trade Name: Betapace

Therapeutic Actions/Pharmacodynamics: Sotalol is a non-selective beta-blocking agent with class II and class III antidysrhythmic properties. It slows the heart rate, decreases AV nodal conduction, and increases AV nodal refractoriness. Sotalol produces significant reductions in both systolic and diastolic blood pressure. It is used both orally and intravenously for ventricular and supraventricular dysrhythmias.

Emergency Uses: To convert ventricular and supraventricular dysrhythmias. Adult dose: 1–1.5 mg/kg IV at a rate of 10 mg/min. The oral dose is 80 mg PO bid or 160 mg PO QD taken prior to meals. Pediatric dose: Not indicated.

Pharmacokinetics
Absorption: Slowly and completely absorbed from GI tract. Negligible first-pass metabolism. Absorption of sotalol may be reduced by food, especially milk and milk products. Peak effect
is 2–3 hr; duration is 24 hr; half-life is 7–18 hr.  

**Distribution:** Drug is hydrophilic and will enter the CSF slowly (about 10%). Crosses placental barrier. Distributed in breast milk. Not appreciably protein bound.  

**Metabolism:** Does not undergo significant hepatic enzyme metabolism and no active metabolites have been identified.  

**Elimination:** Excreted by glomerular filtration in the urine with 75% of the drug excreted unchanged within 72 hr.  

**Contraindications and Precautions:** Sotalol is contraindicated in patients with bronchial asthma, sinus bradycardia, second- and third-degree heart block, long QT syndromes, cardiogenic shock, uncontrolled CHF, chronic bronchitis, emphysema, hypersensitivity to sotalol. Use with caution in patients with CHF, electrolyte disturbances, recent MI, diabetes, sick sinus rhythms, renal impairment.  

**Adverse / Side Effects:** CV: AV block, hypotension, aggravation of CHF (although the incidence of heart failure may be lower than for other beta blockers), life-threatening ventricular arrhythmias, including polymorphous ventricular tachycardia or *torsade de pointes*, bradycardia, dyspnea, chest pain, palpitation, bleeding (less than 2%) • CNS: Headache, fatigue, dizziness, weakness, lethargy, depression, lassitude • GI: Nausea, vomiting, diarrhea, dyspepsia, dry mouth • GU: Impotence, decreased libido • Metabolic: Hyperglycemia • Other: Visual disturbances, respiratory complaints, and rash.  

**Interactions:** Sotalol antagonizes the effects of beta agonists. Amiodarone may lead to symptomatic bradycardia and sinus arrest. Astemizole may prolong QT interval leading to dysrhythmias. The hypoglycemic effects of oral hypoglycemic agents may be potentiated. May cause resistance to epinephrine in anaphylactic reactions. Should be used with caution with other antidysrhythmic agents. Drug–food: absorption of sotalol may be reduced by food, especially milk and milk products.
Prehospital Considerations

- IV sotalol is not available in the United States.
- IV sotalol is limited by its need to be infused slowly. This may be impractical and has uncertain efficacy in emergent situations, particularly under compromised circulatory conditions.
- Smallest effective dose should be used for patients with non-allergic bronchospasms.
- Discontinuation of sotalol should not be abrupt. Dose should gradually be reduced over 1−2 wk.
- Carefully monitor cardiac status, including ECG, through course of therapy. Special caution is warranted when sotalol is used concurrently with other antidysrhythmics, digoxin, or calcium channel blockers.
- Carefully monitor patients with bronchospastic disease (e.g., bronchitis, emphysema) for inhibition of bronchodilation.
- Monitor diabetics for loss of glycemic control. Beta blockade reduces the release of endogenous insulin in response to hyperglycemia and may blunt symptoms of acute hypoglycemia (e.g., tachycardia, BP changes).

STREPTOKINASE

**Class:** Thrombolytic

**Trade Names:** Kabikinase, Streptase

**Therapeutic Actions/Pharmacodynamics:** Streptokinase is a derivative of the beta-hemolytic streptococci. It promotes thrombolysis by activating the conversion of plasminogen to plasmin, the enzyme that degrades fibrin, fibrinogen, and other procoagulant proteins into soluble fragments. This fibrinolytic activity is effective both outside and within the formed thrombus/embolus. It also decreases blood and plasma viscosity and erythrocyte aggregation tendency, thus increasing perfusion of collateral blood vessels.
**Emergency Uses:** To reduce infarct size in acute MI by thrombolysis. *Adult dose:* 1.5 million units IV over 1 hr. *Pediatric dose:* Not used.

As a thrombolytic for clots in deep veins (DVT) and pulmonary embolism. *Adult dose:* 250,000 units IV over 30 min loading dose. Maintenance dose is 100,000 units/hr for 48–72 hr. *Pediatric dose:* Not used.

**Pharmacokinetics**

*Absorption:* Onset is less than 1 hour; peak effect in 80 min; duration is 2–36 hr; half-life is 83 min.

*Metabolism:* Rapidly cleared from circulation by antibodies.

*Elimination:* Does not cross placenta, but antibodies do.

**Contraindications and Precautions:** Streptokinase is absolutely contraindicated in patients with active internal bleeding, suspected aortic dissection, traumatic CPR (rib fractures, pneumothorax), history of recent stroke (within 6 months), recent (within 2 months) intracranial or intraspinal surgery or trauma, intracranial tumors, uncontrolled hypertension, pregnancy, or severe allergic reactions to either anistreplase or streptokinase. Use with caution in patients over age 75, and in those with recent major surgery (within 10 days), cerebral vascular disease, recent GI or GU bleeding, recent trauma, hypertension, hemorrhagic ophthalmic conditions, and current use of oral anticoagulants.

**Adverse/Side Effects:** Allergic: Major (12%) (bronchospasm, periorbital swelling, angioneurotic edema, anaphylaxis); mild (urticaria, itching, headache, musculoskeletal pain, flushing, nausea, pyrexia) • Hematologic: Phlebitis, bleeding or oozing at sites of percutaneous trauma; prolonged systemic hypocoagulability; spontaneous bleeding (GU, GI, retroperitoneal); unstable blood pressure; reperfusion atrial or ventricular dysrhythmias.

**Interactions:** Anticoagulants, NSAIDS increase the risk of bleeding; aminocaproic acid reverses the action of streptokinase.
Prehospital Considerations

- IV preparation: SK is reconstituted with 5 mL 0.9% NaCl injection (preferred) or 5 mL 5% dextrose injection. Roll or tilt vial; avoid shaking to prevent foaming or increase in flocculation. Reconstituted solution may be carefully diluted again, avoiding shaking or agitation of the solution. Slight flocculation does not interfere with drug action; discard solution with large amount of flocculi.
- Observe infusion site frequently. If phlebitis occurs, it can usually be controlled by diluting the infusion solution.
- Reconstituted solution should be stored at 2−4°C (36−39°F). Discard after 24 hr. Store unopened vials at 15−30°C (59−86°F).
- Thrombi more than 7 days old respond poorly to SK therapy; therefore, IV infusion is started as soon as possible after the thrombotic event.
- Spontaneous bleeding occurs about twice as often with SK as with heparin. Protect patient from invasive procedures. IM injections are contraindicated. Also prevent undue manipulation during thrombolytic therapy to prevent bruising.
- Monitor for excessive bleeding every 15 min for the first hour of therapy, every 30 min for second to eighth hour, then every 8 hr.
- Report signs of potential serious bleeding: gum bleeding, epistaxis, hematoma, spontaneous ecchymoses, oozing at catheter site, increased pulse, pain from internal bleeding. SK infusion should be interrupted, then resumed when bleeding stops.
- Report promptly symptoms of a major allergic reaction; therapy will be discontinued and emergency treatment instituted. Minor symptoms (e.g., itching, nausea) respond to concurrent antihistamine or corticosteroid treatment or both without interruption of SK administration.
- Check pulse frequently. Be alert to changes in cardiac rhythm, especially during intracoronary instillation. Dysrhythmias signal need to stop therapy at once.
• Monitor BP. Mild changes can be expected, but report substantial changes (greater than 25 mm Hg). Therapy may be discontinued.
• Patients who have had streptokinase before should not receive the drug a second time because of the possibility of a severe allergic reaction.

SUCCINYLCHOLINE

**Class:** Depolarizing neuromuscular blocker

**Trade Names:** Anectine, Quelicin, Scoline (Aus), Sucostrin

**Therapeutic Actions/Pharmacodynamics:** Succinylcholine is a synthetic, ultra-short-acting depolarizing neuromuscular blocking agent with high affinity for acetylcholine (ACh) receptor sites. Its initial transient contractions and fasciculations are followed by sustained flaccid skeletal muscle paralysis produced by state of accommodation that develops in adjacent excitable muscle membranes. It is rapidly hydrolyzed by plasma pseudocholinesterase. It may increase vagal tone initially, particularly in children and with high doses, and subsequently produce mild sympathetic stimulation. Following IV injection, muscle paralysis begins at the eyelids and jaw, then progresses to the limbs, the abdomen, and diaphragm. There is no effect on level of consciousness.

**Emergency Uses:** To facilitate endotracheal intubation. *Adult dose:* 1.0–1.5 mg/kg IV/IM. *Pediatric dose:* 1.0–2.0 mg/kg IV/IM.

**Pharmacokinetics**

**Absorption:** Onset: 0.5–1 min IV; 2–3 min IM. Duration: 2–3 min IV; 10–30 min IM.

**Distribution:** Crosses placenta in small amounts.

**Metabolism:** Metabolized in plasma by pseudocholinesterases.

**Elimination:** Excreted in urine.
Contraindications and Precautions: Succinylcholine is contraindicated in patients with hypersensitivity to succinylcholine, family history of malignant hyperthermia, penetrating eye injuries, or narrow-angle glaucoma. Safe use in pregnancy (category C) not established. Use with caution in patients with severe burns or severe crush injuries as cardiac and ventricular dysrhythmias have been reported; electrolyte imbalances; renal, hepatic, pulmonary, metabolic, or cardiovascular disorders; fractures, spinal cord injury, severe liver disease, severe anemia, dehydration; collagen disorders, porphyria, intraocular surgery, glaucoma.

Adverse/Side Effects: CNS: Muscle fasciculations, profound and prolonged muscle relaxation, muscle pain • CV: Bradycardia, tachycardia, hypotension, hypertension, dysrhythmias, sinus arrest • Respiratory: Respiratory depression, bronchospasm, hypoxia, apnea • Other: Malignant hyperthermia, increased IOP, excessive salivation, enlarged salivary glands, myoglobinemia, hyperkalemia; decreased tone and motility of GI tract (large doses).

Interactions: Aminoglycosides, colistin, cyclophosphamide, cyclopropane, echothiophate iodide, halothane, lidocaine, magnesium salts, methotrimeprazine, narcotic analgesics, organophosphamide insecticides, MAO inhibitors, phenothiazines, procaine, procaainamide, quinidine, quinine, propranolol may prolong neuromuscular blockade. Digitalis glycosides may increase risk of cardiac dysrhythmias.

Prehospital Considerations
- Only freshly prepared solutions should be used; succinylcholine hydrolyzes rapidly with consequent loss of potency.
- IM injections are made deeply, preferably high into deltoid muscle.
- IV preparation (for prolonged paralysis): IV succinylcholine may be diluted, 1 g in 1 L of D5W or NS, and given by intermittent or continuous infusion at a rate not to exceed 10 mg/min. A nondepolarizing agent, such as vecuronium or pancuronium, is preferred for prolonged neuromuscular blockade.
• IV administration: IV succinylcholine chloride may be given by direct IV undiluted over 10–30 seconds.
• Facilities for emergency endotracheal intubation and positive pressure ventilation with oxygen should be immediately available.
• Tachyphylaxis (reduced response) may occur after repeated doses.
• Adverse effects are primarily extensions of pharmacologic actions.
• Monitor vital signs and keep airway clear of secretions.
• All resuscitation equipment should be immediately available before administering a neuromuscular blocker.

**THIAMINE**

**Class:** Vitamin

**Trade Names:** Betamin (Aus), Beta-Sol (Aus), Biamine

**Therapeutic Actions/Pharmacodynamics:** Thiamine is a water-soluble vitamin and member of the B-complex group. It functions as an essential coenzyme in carbohydrate metabolism. Thiamine is not produced by the body but must be obtained through the diet. It is required for the conversion of pyruvic acid to acetyl-coenzyme-A. Without thiamine, a significant amount of the energy available in glucose cannot be obtained. The brain, particularly, is extremely sensitive to thiamine deficiency. Chronic alcoholic intake interferes with the absorption, intake, and use of thiamine, thus a significant number of alcoholics have thiamine deficiency. Two serious neurologic conditions, Wernicke’s syndrome and Korsakoff’s psychosis, can result from thiamine deficiency.

**Emergency Uses:** To treat coma of unknown origin, especially if alcohol is involved, and delerium tremens. *Adult dose:* 50–100 mg IV/IM. *Pediatric dose:* 10–25 mg IV/IM.
Pharmacokinetics
Distribution: Widely distributed, including into breast milk.
Elimination: Excreted in urine.
Adverse/Side Effects: CNS: Restlessness, weakness • CV:
Cardiovascular collapse • Respiratory: Pulmonary edema •
Other: Feeling of warmth, urticaria, pruritus, sweating, nausea,
tightness of throat, angioneurotic edema, cyanosis, GI hemorrhage, anaphylaxis • Following rapid IV administration: Slight fall in BP.
Prehospital Considerations
• IV thiamine may be given by direct IV undiluted at a rate of 100 mg over 5 min. May also be added to IV solutions and infused at ordered rate.

VASOPRESSIN
Classes: Hormone; vasopressor
Trade Name: Pitressin
Therapeutic Actions/Pharmacodynamics: Vasopressin is a polypeptide hormone extracted from animal posterior pituitaries. It possesses pressor and antidiuretic (ADH) principles, but is relatively free of oxytocic properties. Vasopressin produces concentrated urine by increasing tubular reabsorption of water (ADH activity), thus preserving up to 90% water. It may increase sodium and decrease potassium reabsorption but plays no causative role in edema formation. Small doses may produce anginal pain; large doses may precipitate MI, decrease heart rate and cardiac output, and increase pulmonary arterial pressure and BP. In unnaturally high doses (higher than those needed for antidiuretic effects) vasopressin acts as a non-adrenergic vasoconstrictor. It acts by direct stimulation of smooth muscle (V1) receptors. It can be used as an alternative to epinephrine during CPR.
Emergency Uses: To increase peripheral vascular resistance during CPR (as an alternative to epinephrine) or after epinephrine has been used. Adult dose: 40 units IV (single dose only). Vasopressin is used to control bleeding from esophageal varices. It is administered by IV infusion at 0.2–0.4 units/min.

Pharmacokinetics
Absorption: Duration is 30–60 min IV infusion; half-life is 10–20 min.
Distribution: Extracellular fluid.
Metabolism: Metabolized in liver and kidneys.
Elimination: Excreted in urine.

Contraindications and Precautions: Vasopressin is contraindicated in patients with chronic nephritis accompanied by nitrogen retention; ischemic heart disease, PVCs, advanced arteriosclerosis; during first stage of labor. Use with caution in patients with epilepsy; migraine; asthma; heart failure, angina pectoris; any state in which rapid addition to extracellular fluid may be hazardous; vascular disease; preoperative and postoperative polyuric patients; renal disease; goiter with cardiac complications; and in elderly patients and children.

Adverse/Side Effects: Infrequent with low doses. Large doses: blanching of skin, abdominal cramps, nausea (almost spontaneously reversible), hypertension, bradycardia, minor arrhythmias, premature atrial contraction, heart block, peripheral vascular collapse, coronary insufficiency, MI.

Interactions: Alcohol, demeclocycline, epinephrine, heparin, lithium, phenytoin may decrease antidiuretic effects of vasopressin; guanethidine, neostigmine increase vasopressor actions; chlorpropamide, clofibrate, carbamazepine, thiazide diuretics may increase antidiuretic activity.

Prehospital Considerations
- Conclusive evidence supporting the use of vasopressin in cardiac arrest is lacking. Its use in refractory VF is class IIb
Vecuronium's use in asystole, PEA, and prolonged cardiac arrest is indeterminate (neither recommended nor forbidden).

- It may be useful in septic shock. It is a class IIb when used when standard therapy (inotropic agents and vasoconstrictor drugs commonly used) is inadequate.
- When used in vasopressor doses, as in the treatment of bleeding esophageal varices, it is common practice to administer IV nitroglycerin to help maintain mesenteric and intestinal perfusion.

VECURONIUM

**Class:** Nondepolarizing skeletal muscle relaxant

**Trade Name:** Norcuron

**Therapeutic Actions/Pharmacodynamics:** Vecuronium is an intermediate-acting nondepolarizing skeletal muscle relaxant structurally similar to pancuronium. Unlike older neuromuscular blocking agents, it demonstrates negligible histamine release and therefore has minimal direct effect on the cardiovascular system. It is similar to atracurium in having unique metabolic and excretion pathways. In common with other drugs of this class, vecuronium inhibits neuromuscular transmission by competitive binding with acetylcholine to motor endplate receptors. It is given only after induction of general anesthesia.

**Emergency Use:** To facilitate endotracheal intubation. *Adult dose:* 0.08–0.10 mg/kg IV. *Pediatric dose (1 yr or older):* Same as adult.

**Pharmacokinetics**

*Absorption:* Onset is less than 1 min; peak effects in 3–5 min; duration is 25–40 min; half-life is 30–80 min.

*Distribution:* Well distributed to tissues and extracellular fluids; crosses placenta; distribution into breast milk unknown.

*Metabolism:* Rapid nonenzymatic degradation in bloodstream.
Elimination: 30–35% excreted in urine, 30–35% in bile.

Contraindications and Precautions: Vecuronium is contraindicated in patients with a hypersensitivity to the drug. Safe use during pregnancy (category C), in nursing mothers, and in neonates not established. Use with caution in patients with severe hepatic disease; impaired acid-base or fluid/electrolyte balance; severe obesity; adrenal or neuromuscular disease (myasthenia gravis); patients with slow circulation time (cardiovascular disease, old age, edematous states); malignant hyperthermia.

Adverse/Side Effects: CNS: Skeletal muscle weakness • Respiratory: Respiratory depression • Other: Malignant hyperthermia.

Interactions: General anesthetics increase neuromuscular blockade and duration of action. Aminoglycosides, bacitracin, polymyxin B, clindamycin, lidocaine, parenteral magnesium, quinidine, quinine, trimethaphan, and verapamil increase neuromuscular blockade. Diuretics may increase or decrease neuromuscular blockade. Lithium prolongs duration of neuromuscular blockade. Narcotic analgesics increase possibility of additive respiratory depression. Succinylcholine increases onset and depth of neuromuscular blockade. Phenytoin may cause resistance to or reversal of neuromuscular blockade.

Prehospital Considerations

• Monitor vital signs at least every 15 min until stable, then every 30 min for the next 2 hr. Also monitor airway patency until assured that patient has fully recovered from drug effects. Note rate, depth, and pattern of respirations. Obese patients and patients with myasthenia gravis or other neuromuscular disease may pose ventilation problems.

• Evaluate patients for recovery from neuromuscular blocking (curare-like) effects as evidenced by ability to breathe naturally or take deep breaths and cough, to keep eyes open, and
to lift head keeping mouth closed and by adequacy of hand grip strength. Notify physician if recovery is delayed.

- Note that recovery time may be delayed in patients with cardiovascular disease, edematous states, and in the elderly.
- All resuscitation equipment and supplies must be readily available before administering a neuromuscular blocker.
- Vecuronium is often used as the primary neuromuscular blocker in some EMS systems as it is nondepolarizing and does not require refrigeration.

VERAPAMIL

Class: Calcium channel blocker

Trade Names: Anpec (Aus), Calan, Cordilox (Aus), Isoptin, Novo-Veramil (Can), Verelan

Therapeutic Actions/Pharmacodynamics: Verapamil inhibits calcium ion influx through slow channels into cell of myocardial and arterial smooth muscle. It dilates the coronary arteries and arterioles and inhibits coronary artery spasm; thus, myocardial oxygen delivery is increased (antianginal effect). It also decreases and slows SA and AV node conduction (antidysrhythmic effect) without effect on normal arterial action potential or intraventricular conduction. By vasodilation of peripheral arterioles, the drug decreases total peripheral vascular resistance and reduces arterial BP at rest. Verapamil may slightly decrease heart rate.

Emergency Uses: To convert paroxysmal supraventricular tachycardia (PSVT) refractory to adenosine, to convert atrial fibrillation and atrial flutter. Adult dose: 2.5–5.0 mg slow IV. May repeat at double dose in 15–30 min as needed. Do not exceed 30 mg in 30 min. Pediatric dose: Newborn to 1 yr: 0.1–0.2 mg/kg (not to exceed 2 mg) IV. Age 1–15 yr: 0.1–0.3 mg/kg (not to exceed 5 mg) IV.
Pharmacokinetics

Absorption: Onset is 5 min; peak effect in 5–15 min; duration is 10–60 min; half-life is 2–8 hr.

Distribution: Widely distributed, including CNS; crosses placentas; present in breast milk. Metabolism: Metabolized in liver.

Elimination: 70% excreted in urine; 16% in feces.

Contraindications and Precautions: Verapamil is contraindicated in severe hypotension (diastolic greater than 90 mm Hg), cardiogenic shock, cardiomegaly, digitalis toxicity, second- or third-degree AV block, Wolff-Parkinson-White syndrome including atrial flutter and fibrillation, accessory AV pathway, left ventricular dysfunction, severe CHF, sinus node disease, and sick sinus syndrome (except in patient with functioning ventricular pacemaker). Safe use during pregnancy (category C), in nursing mothers, or in children (oral) not established. Use with caution in patients with Duchenne’s muscular dystrophy; hepatic and renal impairment; MI followed by coronary occlusion or aortic stenosis.

Adverse/Side Effects: CNS: Dizziness, vertigo, headache, fatigue, sleep disturbances, depression, syncope • CV: Hypotension, congestive heart failure, bradycardia, severe tachycardia, peripheral edema, AV block • GI: Nausea, abdominal discomfort, constipation • Other: Pruritus, flushing, pulmonary edema, muscle fatigue, diaphoresis, elevated liver enzymes.

Interactions: Beta blockers increase risk of CHF, bradycardia, or heart block. Increases serum levels and toxicity of digoxin, carbamazepine, cyclosporine, lithium. Potentiates hypotensive effects of other antihypertensive agents. Verapamil effects antagonized by calcium chloride.

Prehospital Considerations

• IV injection: IV verapamil may be given by direct IV diluted in 5 mL of sterile water for injection at a rate of 10 mg/min.
• Inspect parenteral drug preparation before administration. Solution should be clear and colorless.
• Establish baseline data and periodically monitor BP, pulse, and hepatic and renal function.
• Transient asymptomatic hypotension may accompany IV bolus. Instruct patient to remain in recumbent position for at least 1 hr after dose is given to diminish subjective effects of hypotension.
• If IV verapamil is given concurrently with digitalis, monitor for AV block or excessive bradycardia.
• The incidence of adverse reactions is highest with IV administration, in the elderly, in patients with impaired renal function, and in patients of small stature. Drug action may be prolonged in these patients. Continuous ECG monitoring during IV administration is essential.
Home Medications

Home Medication Classes
List of Home Medications
### Home Medication Classes

**ACE inhibitor**
- Prevents the conversion of angiotensin I into angiotensin II. Prescribed for hypertension and CHF.

**Adrenergic Antagonist**
- Stimulates the binding of nor-epinephrine and epinephrine at receptor sites. Prescribed for a wide variety of conditions.

**Alpha₁ antagonist**
- Blocks effects of alpha₁ receptors in the peripheral blood vessels. Prescribed for hypertension and BPH.

**Alpha₂ agonist**
- Stimulates the alpha₂ receptors in the brain to inhibit presynaptic adrenergic terminals. Prescribed for hypertension.

**Analgesic**
- Broad term referring to drugs that relieve pain.

**Anorexiant**
- Used to control appetite and cause weight loss.

**Angiotensin II antagonist**
- Blocks the effects of angiotensin II at receptor sites. Prescribed for hypertension.

**Antialcohol**
- Creates unpleasant effects when alcohol is consumed. Used as a deterrent in chronic alcoholism.

**Anti-ALS**
- Used to treat amyotrophic lateral sclerosis.

**Antibiotic**
- Used to fight bacterial infection. Prescribed for a variety of infections.
<table>
<thead>
<tr>
<th><strong>Anticholinergic</strong></th>
<th>Blocks acetylcholine at neuro-receptor site. Prescribed for a variety of conditions.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anticonvulsant</strong></td>
<td>Broad term referring to drugs used to control seizures.</td>
</tr>
<tr>
<td><strong>Anticoagulant</strong></td>
<td>Interrupts the clotting cascade. Prescribed to treat deep venous thrombosis (DVT), pulmonary embolism (PE), ischemic stroke, myocardial infarction (MI).</td>
</tr>
<tr>
<td><strong>Antidepressant</strong></td>
<td>Broad term referring to drugs used to treat depression.</td>
</tr>
<tr>
<td><strong>Antidiarrheal</strong></td>
<td>Inhibits bowel motility. Prescribed for diarrhea.</td>
</tr>
<tr>
<td><strong>Antidiuretic</strong></td>
<td>Inhibits diuresis.</td>
</tr>
<tr>
<td><strong>Antidysrhythmic</strong></td>
<td>Broad term referring to drugs that control and regulate the cardiac rhythm.</td>
</tr>
<tr>
<td><strong>Antiemetic</strong></td>
<td>Broad term referring to drugs used to control vomiting.</td>
</tr>
<tr>
<td><strong>Antihistamine</strong></td>
<td>Blocks the effects of histamine at receptor sites. H₁ blockers are prescribed for allergies. H₂ blockers are prescribed for ulcers, reflux, acid indigestion.</td>
</tr>
<tr>
<td><strong>Antihyperlipidemic</strong></td>
<td>Lowers serum cholesterol levels. Prescribed to prevent atherosclerosis.</td>
</tr>
<tr>
<td><strong>Antihypertensive</strong></td>
<td>Broad term referring to drugs that lower the blood pressure.</td>
</tr>
<tr>
<td><strong>Antimigraine</strong></td>
<td>Used to treat migraine headaches.</td>
</tr>
<tr>
<td><strong>Antineoplastic</strong></td>
<td>Kills cancer cells by a variety of mechanisms. Prescribed to treat a variety of cancers.</td>
</tr>
<tr>
<td><strong>Antiparkinson</strong></td>
<td>Increases stimulation of dopamine receptors in the brain. Prescribed to manage symptoms of Parkinson’s disease.</td>
</tr>
<tr>
<td><strong>Antiplatelet</strong></td>
<td>Decreases the formation of platelet aggregates. Prescribed to prevent MI and stroke.</td>
</tr>
<tr>
<td><strong>Antipsychotic</strong></td>
<td>Broad term referring to drugs used to treat a variety of psychiatric conditions.</td>
</tr>
<tr>
<td><strong>Antireflux</strong></td>
<td>Controls esophageal reflux.</td>
</tr>
<tr>
<td><strong>Antiseizure</strong></td>
<td>Controls seizure activity in the brain.</td>
</tr>
<tr>
<td><strong>Antituberculosis</strong></td>
<td>Kills the tuberculosis bacteria. Prescribed for suspected and confirmed tuberculosis.</td>
</tr>
<tr>
<td><strong>Antitussive</strong></td>
<td>Suppresses the cough reflex mechanism. Prescribed for cough.</td>
</tr>
<tr>
<td><strong>Antiulcer</strong></td>
<td>Reduces the secretion of acids. Prescribed for ulcers.</td>
</tr>
<tr>
<td><strong>Antivertigo</strong></td>
<td>Broad term referring to drugs used to control vertigo and motion sickness.</td>
</tr>
<tr>
<td><strong>Antiviral</strong></td>
<td>Treats viruses by a variety of mechanisms. Prescribed for a variety of viral infections, particularly HIV.</td>
</tr>
</tbody>
</table>
Anxiolytic Alleviates anxiety.
Barbiturate Causes sedative-hypnotic effect. Prescribed for seizures, anxiety.
Benzodiazepine Causes sedative-hypnotic effect. Prescribed for seizures, anxiety, muscle relaxant.
Beta blocker Blocks the effects of epinephrine at beta receptor sites. Prescribed for angina, hypertension, tachydyssrhythmias.
Bronchodilator Dilates the bronchiole smooth muscles by a variety of mechanisms.
Calcium channel blocker Inhibits the influx of calcium into the cell. Prescribed for angina, hypertension, tachydyssrhythmias.
Cardiac glycoside Lowers heart rate while increasing the force of contraction. Prescribed for tachydyssrhythmias, congestive heart failure.
Cholinesterase inhibitor Prolongs the effects of acetylcholine by inhibiting its breakdown. Prescribed for myasthenia gravis, some types of poisoning, and glaucoma.
Cholinergic Stimulates binding of acetylcholine with muscarinic receptors, particularly in
bladder and GI tract.
Prescribed to increase urination and peristalsis.

CNS stimulant
Increases CNS depolarization, prescribed for fatigue, narcolepsy, obesity, attention deficit hyperactivity disorder (ADHD), drowsiness.

Decongestant
Relieves upper respiratory tract congestion.

Digestant
Mimics the pancreatic enzymes and aids in digestion of food.

Diuretic
Stimulates the kidneys to produce more urine. Prescribed for hypertension, and congestive heart failure.

Estrogen hormone
Replaces estrogen in postmenopausal women.

Glucocorticoid
Reduces the inflammatory process. Prescribed for asthma, and other causes of inflammation.

Hormone
Simulates the action of various glands. Prescribed for a variety of conditions that affect the endocrine system.

Hypnotic
Induces sleep.

Immunosupressant
Used with transplant surgery to decrease incidence of rejection. Also to treat some cancers.
<table>
<thead>
<tr>
<th>Medication Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infertility agent</td>
<td>Promotes the maturation of ovarian follicles. Prescribed to treat infertility.</td>
</tr>
<tr>
<td>Insulin preparation</td>
<td>Facilitates the diffusion of glucose into the cells. Prescribed for insulin-dependent diabetes mellitus.</td>
</tr>
<tr>
<td>Laxative</td>
<td>Alters the consistency of stool. Prescribed for constipation.</td>
</tr>
<tr>
<td>Leukotriene antagonist</td>
<td>Blocks the effects of leukotrienes at receptor sites. Prescribed for asthma.</td>
</tr>
<tr>
<td>MAO inhibitor</td>
<td>Inhibits breakdown of monoamine oxidase (MAO). Prescribed for depression.</td>
</tr>
<tr>
<td>Mast cell stabilizer</td>
<td>Prevents mast cells from secreting histamine.</td>
</tr>
<tr>
<td>Methylxanthine</td>
<td>Bronchodilates by inhibiting the breakdown of beta-agonists. Prescribed for asthma.</td>
</tr>
<tr>
<td>Mood stabilizer</td>
<td>Prescribed for bipolar disorder (manic-depressive).</td>
</tr>
<tr>
<td>Mucolytic</td>
<td>Decreases the viscosity of respiratory secretions. Prescribed for upper respiratory infection, pneumonia, cystic fibrosis.</td>
</tr>
<tr>
<td>Narcotic analgesic</td>
<td>Contains opium or its derivative. Prescribed for moderate or severe pain relief.</td>
</tr>
<tr>
<td>Class</td>
<td>Description</td>
</tr>
<tr>
<td>---------------------------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>NSAID</td>
<td>Nonsteroidal anti-inflammatory drug; prescribed for pain, fever, inflammation, arthritis.</td>
</tr>
<tr>
<td>Oral hypoglycemic</td>
<td>Some stimulate the pancreas to produce insulin. Others alter the absorption of glucose from the GI tract. Prescribed for non-insulin-dependent diabetes mellitus.</td>
</tr>
<tr>
<td>Phenothiazine</td>
<td>Antipsychotic medications that block dopamine receptors in the brain; prescribed for schizophrenia, depression, bipolar disorder, and nausea.</td>
</tr>
<tr>
<td>Progestin hormone</td>
<td>Replaces progesterone in postmenopausal women. Prescribed as oral contraceptive and to treat amenorrhea, endometriosis, and uterine bleeding.</td>
</tr>
<tr>
<td>Psychotherapeutic</td>
<td>Broad term referring to drugs used to treat a variety of psychiatric conditions.</td>
</tr>
<tr>
<td>SSRI</td>
<td>Selective serotonin reuptake inhibitors. Block reuptake of serotonin. Prescribed for depression and as an antipsychotic.</td>
</tr>
<tr>
<td>Skeletal muscle relaxant</td>
<td>Blocks the release of calcium from sarcoplasmic reticulum. Prescribed for muscle spasms.</td>
</tr>
<tr>
<td>Medicine Type</td>
<td>Description</td>
</tr>
<tr>
<td>-------------------------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Stool softener</td>
<td>Alters the consistency of stool. Used as a laxative.</td>
</tr>
<tr>
<td>Thyroid hormone</td>
<td>Synthetic replica of the natural thyroid hormone.</td>
</tr>
<tr>
<td>Tricyclic antidepressant</td>
<td>Blocks reuptake of norepinephrine and serotonin. Prescribed for depression.</td>
</tr>
<tr>
<td>Uricosuric</td>
<td>Used to treat gout.</td>
</tr>
<tr>
<td>Vasodilator</td>
<td>Dilates the blood vessels.</td>
</tr>
</tbody>
</table>
List of Home Medications

Following is a list of the most commonly prescribed medications and their class. If additional information is required concerning a drug, consult the *Physician’s Desk Reference* or a similar source. In the left column, trade names are capitalized, and generic names are in lower case.

- acarbose: Oral hypoglycemic
- Accolate: Leukotriene antagonist
- Accupril: ACE inhibitor
- acebutolol: Beta blocker
- Aceon: ACE inhibitor
- acetaminophen: Analgesic
- Acetazolam: Diuretic
- acetazolamide: Diuretic
- acetohexamide: Oral hypoglycemic
- acetylcysteine: Mucolytic
- Achro-mycin: Antibiotic
- Actidil: Antihistamine
- Actron: NSAID
- Acular: NSAID
- acycloguanosine: Antiviral
- acyclovir: Antiviral
- Adalat: Calcium channel blocker
- Adapin: Tricyclic antidepressant
- Aerosorb-Dex: Glucocorticoid
- Aerosporin: Antibiotic
- Agrylin: Antiplatelet
- Airbron: Mucolytic
- Akineton: Anticholinergic (antiparkinson)
- Alazine: Antihypertensive
- Albamycin: Antibiotic
- albendazole: Antibiotic
- Albenza: Antibiotic
<table>
<thead>
<tr>
<th>Drug</th>
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Arthropan
Asendin
Atabrine
Atacand
Atarax
Atenolol
Ativan
Atolone
Atorvastatin
Atromid
Atrovent
Avapro
Aventyl
Avlosulfon
Axid
Azactam
Azatadine
Azathioprine
Azithromycin
Aztreonam
Bacampicillin
Baclofen
Bactocill
Bactrim
Banflex
Barbased
Baychol
Beclohasone
Beeclomvent
Beconase
Benadryl
Benazepril
Bendrofluamide
NSAID
Tricyclic antidepressant
Antibiotic
Angiotensin II blocker
Antihistamine
Beta blocker
Benzodiazepine
Glucocorticoid
Antihyperlipidemic
Antihyperlipidemic
Anticholinergic bronchodilator
Angiotensin II blocker
Tricyclic antidepressant
Antibiotic
Antulcer
Antibiotic
Antihistamine
Immunosuppressant
Antibiotic
Antibiotic
Skeletal muscle relaxant
Barbiturate
Antihyperlipidemic
Glucocorticoid
Glucocorticoid
Glucocorticoid
Antihistamine
ACE inhibitor
Diuretic
Benemid
Bensylate
Bentyl
Benuryl
benzonatate
benzphetamine
benzthiazide
benztropine
bepridil
Betactol
Betapace
Betapen
Bethanechol
Betimol
Biaxin
Biperiden
Bishydroxycoumarin
bisoprolol
bitolterol
Blocadren
Bonamine
Bonine
Brethaire
Brethine
Bricanyl
Bromphen
brompheniramine
Bronkodyl
Bronkometer
Bronkosol
Bucalgin
Buclizine
Budesonide
Bumetanide

Uricosuric (antigout)
Anticholinergic (antiparkinson)
Antispasmodic
Uricosuric (antigout)
Antitussive
CNS stimulant
Diuretic
Anticholinergic (antiparkinson)
Calcium channel blocker
Beta blocker
Beta blocker
Antibiotic
Cholinergic
Beta blocker
Antiemetic
Antiemetic
Adrenergic bronchodilator
Adrenergic bronchodilator
Adrenergic bronchodilator
Antihistamine
Antihistamine
Methylxanthine bronchodilator
Adrenergic bronchodilator
Adrenergic bronchodilator
Antivertigo
Antivertigo
Glucocorticoid
Diuretic
Bumex  Diuretic
bumprion  Antidepressant
Buspar  Anxiolytic
buspirone  Anxiolytic
busulfan  Antineoplastic
butabarbital  Barbiturate
Butalan  Barbiturate
Butisol  Barbiturate
Calan  Calcium channel blocker
candesartan  Angiotensin II antagonist
capcicitabine  Antineoplastic
Capoten  ACE inhibitor
captopril  ACE inhibitor
Carafate  Antiulcer
carbamazepine  Anticonvulsant
Carbatrol  Anticonvulsant
carbenicillin  Antibiotic
Carbex  MAO inhibitor
carbidopa  Anticholinergic (antiparkinson)
Carbolith  Mood stabilizer
Cardene  Calcium channel blocker
Cardioquin  Antidysrhythmic
Cardizem  Calcium channel blocker
Cardura  Alpha_1 blocker
carisoprodol  Skeletal muscle relaxant
carteolol  Beta blocker
Cartrol  Beta blocker
carvedilol  Antihypertensive
Catapres  Alpha_2 agonist
Ceeclor  Antibiotic
Cedax  Antibiotic
cefaclor  Antibiotic
cefadroxil  Antibiotic
Cefanex  Antibiotic
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cefdinir Antibiotic
cefixime Antibiotic
cefpodoxime Antibiotic
cefproxi Antibiotic
ceftibuten Antibiotic
cefuroxime Antibiotic
Cefzil Antibiotic
Celebrex NSAID
celecoxib NSAID
Celexa SSRI antidepressant
Celontin Anticonvulsant
cephalexin Antibiotic
cephradine Antibiotic
Cephulac Laxative
Ceporex Antibiotic
Cerespan Vasodilator
cerivastatin Antihyperlipidemic
Cesamet Antiemetic
Cetirizine Antihistamine
Chibroxin Antibiotic
chloral hydrate Anxiolytic
chlorambucil Antineoplastic
clordiazepoxide Benzodiazepine
Chloronase Oral hypoglycemic
chlorothiazide Diuretic
 Chlorpromamyl Phenothiazine
chlorpromazine Phenothiazine
chlorpropamide Oral hypoglycemic
chlorprothixene Phenothiazine
chlorthalidone Diuretic
chlortrianisene Estrogen hormone
chlorzoxazone Skeletal muscle relaxant
Choledyl Methylxanthine bronchodilator
Cholestabyl Antihyperlipidemic
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HOME MEDICATIONS

Cogentin  Anticholinergic (antiparkinson)
Cognex  Cholinesterase inhibitor
Colace  Stool softener
colchicine  Uricosuric (antigout)
Colestid  Antihyperlipidemic
colestipol  Antihyperlipidemic
colistimethate  Antihyperlipidemic
Coly-Mycin  Antibiotic
Compazine  Phenothiazine
Conjec  Antihistamine
Cophene  Antihistamine
Coreg  Antihypertensive
Corgard  Beta blocker
Coronex  Nitrate
cortisone  Glucocorticoid
Cortistan  Glucocorticoid
Cortone  Glucocorticoid
Cotazym  Digestant
Coumadin  Anticoagulant
Cozaar  Angiotensin II blocker
Crinone  Progesterone hormone
Crixivan  Antiviral
cromolyn sodium  Mast cell stabilizer
Cronetal  Antiemetic
cyclizine  Antiemetic
cyclobenzaprine  Skeletal muscle relaxant
cycloserine  Antituberculosis
cyclosporine  Immunosuppressant
Cycoflex  Skeletal muscle relaxant
Cycrin  Progesterone hormone
Cylert  CNS stimulant
cyproheptadine  Antihistamine
Cytadren  Antineoplastic
Cytomel  Thyroid hormone
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Dexampex  CNS stimulant
Dexasone  Glucocorticoid
Dexchlor  Antihistamine
dexchlorpheniramine  Antihistamine
Dex-edrine  CNS stimulant
Dexone  Glucocorticoid
dextroamphetamine  CNS stimulant
dextrothyroxine  Antihyperlipidemic
DGSS  Stool softener
DiaBeta  Oral hypoglycemic
Diabinese  Oral hypoglycemic
Diachlor  Diuretic
Diamox  Diuretic
Diapil  Antidiuretic
Diaqua  Diuretic
diazepam  Benzodiazepine
Dibenzyline  Alpha1 blocker
diclofenac  NSAID
dicloxacillin  Antibiotic
dicumarol  Anticoagulant
dicyclomine  Anticholinergic; antispasmodic
didanosine  Antiviral agent
Didrex  CNS stimulant
diethylproprion  Anorexiant
Diflucan  Antibiotic
diflunisal  NSAID
digoxin  Cardiac glycoside
Dilacor  Calcium channel blocker
Dilantin  Anticonvulsant
Dilatrate  Nitrate
Dilaudid  Narcotic analgesic
Dilor  Methylxanthine bronchodilator
diltiazem  Calcium channel blocker
Dimelor  Oral hypoglycemic
dimenhydrinate  Antihistamine
Dimetane  Antihistamine
Dinate  Antihistamine
Dio-Sul  Stool softener
Diovan  Angiotensin II antagonist
Dipentum  Antiinflammatory
diphenhydramine  Antihistamine
diphenidol  Antivertigo
dirithromycin  Antibiotic
Disalcid  NSAID
Disodium Cromoglycate  Mast cell stabilizer
Disonate  Stool softener
disopyramide  Antidysrhythmic
disulfiram  Antialcohol agent
Ditropan  Anticholinergic
Diurardin  Diuretic
Diulo  Diuretic
Dieuse  Diuretic
Diuril  Diuretic
Dixaril  Alpha₂ agonist
Dizmiss  Antiemetic
docusate  Stool softener
Dolobid  NSAID
Dolophine  Narcotic analgesic
Dommanate  Antihistamine
donepezil  Cholinesterase inhibitor
Dopamet  Antihypertensive
Dopar  Anticholinergic (antiparkinson)
Doral  Benzodiazepine
Dorilute  Barbiturate
dornase alpha  Mucolytic
Doryx  Antibiotic
doxazosin  Alpha₁ blocker
doxepin  Tricyclic antidepressant
Doxy
Doxychel
doxycycline
Dramamine
Dramanate
Dramilin
Dramocen
droperidol
DSCG
Duosol
Duotrate
Duraclon
Duralith
Durapam
Duraquin
Duretic
Duricef
Duvoid
Dycill
Dyflex
Dyl-line
Dymenate
Dynabac
Dynacirc
Dynapen
dyphylline
Dyrening
Edecrin
Efavirenz
Efedron
Effexor
Elavil
Eldepryl
Elixophyllin

Antibiotic
Antibiotic
Antibiotic
Antihistamine
Antihistamine
Antihistamine
Antihistamine
Antiemetic
Mast cell stabilizer
Stool softener
Nitrate
Alpha2 agonist
Mood stabilizer
Benzodiazepine
Antidysrhythmic
Diuretic
Antibiotic
Cholinergic
Antibiotic
Methylxanthine bronchodilator
Methylxanthine bronchodilator
Antihistamine
Antibiotic
Calcium channel blocker
Antibiotic
Methylxanthine bronchodilator
Diuretic
Diuretic
Antiviral
Decongestant
SSRI antidepressant
Tricyclic antidepressant
MAO inhibitor
Methylxanthine bronchodilator
Eltroxin Thyroid hormone
Emcyt Antineoplastic
Emex Cholinergic
Emitrip Tricyclic antidepressant
E-Mycin Antibiotic
enalapril ACE inhibitor
Endep Tricyclic antidepressant
Enduron Diuretic
Enovil Tricyclic antidepressant
enoxacin Antibiotic
ephedrine Decongestant
Epitol Anticonvulsant
Epivir Antiviral
Equanil Anxiolytic
Erythrocin Antibiotic
erythromycin Antibiotic
Esidrix Diuretic
Eskalith Mood stabilizer
estazolam Benzodiazepine
Estinyl Estrogen hormone
Estrace Estrogen hormone
Estraderm Estrogen hormone
estradiol Estrogen hormone
estramustine Antineoplastic
Estring Estrogen hormone
estrogens Estrogen hormone
estropipate Estrogen hormone
ethacrynic acid Diuretic
ethambutol Antituberculosis agent
Ethaquin Vasodilator
Ethatab Vasodilator
ethaverine Vasodilator
ethchlorvynol Barbiturate
ethinyl estradiol Estrogen hormone
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ethionamide  Antituberculosis agent
Ethmozine  Antidysrhythmic
Ethon  Diuretic
ethosuximide  Anticonvulsant
Etibi  Antituberculosis
etodolac  NSAID
Euglucon  Oral hypoglycemic
Eulexin  Antineoplastic
Euthroid  Thyroid hormone
Evista  Estrogen hormone
Exna  Diuretic
famciclovir  Antiviral agent
Famvir  Antiviral agent
Fareston  Antineoplastic
felbamate  Anticonvulsant
Felbatol  Anticonvulsant
Feldene  NSAID
felodipine  Calcium channel blocker
Femara  Antineoplastic
Feminone  Estrogen hormone
Fempatch  Estrogen hormone
fenofibrate  Antihyperlipidemic
fexofenadine  Antihistamine
Fivent  Mast cell stabilizer
Flagyl  Antibiotic
flavoxate  Anticholinergic
flecainide  Antidysrhythmic
Flexeril  Skeletal muscle relaxant
Flexon  Skeletal muscle relaxant
Flomax  Alpha1 blocker
Florinef  Glucocorticoid
Floxin  Antibiotic
fluconazole  Antibiotic
flucytosine  Antibiotic
<table>
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guanfacine Antihypertensive
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Halcion Benzodiazepine
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Haldrone Glucocorticoid
haloperidol Antipsychotic
haloprogin Antibiotic
Halotex Antibiotic
HCTZ Diuretic
Hexadrol Glucocorticoid
Hexalen Antineoplastic
hexamethylamine Antineoplastic
Hiprex Antibiotic
Histantil Phenothiazine
Hivid Antiviral agent
Honval Estrogen hormone
Humalog Insulin preparation
Humatin Antibiotic
Humulin Insulin preparation
Hycodan Narcotic analgesic
hydralazine Antihypertensive
Hydrate Antihistamine
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Alpha1 blocker
Antihistamine
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Antibiotic
Digestant
Nitrate
Antimigraine
Antidiarrheal
Tricyclic antidepressant
Tricyclic antidepressant
Immunosuppressant
Antiemetic
NSAID
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Mast cell stabilizer
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metolazone    Diuretic
metoprolol    Beta blocker
Metrodin      Infertility agent
metronidazole Antibiotic
Mevacor       Antihyperlipidemic
Mevinolin     Antihyperlipidemic
Mexate        Antineoplastic
mexiletine    Antidysrhythmic
Mexitil       Antidysrhythmic
Mezlin        Antibiotic
mezlocillin   Antibiotic
Micronase     Oral hypoglycemic
Micronor      Progesterone hormone
Midamor       Diuretic
midodrine     Alpha1 agonist
Miltown       Anxiolytic
Minipress     Alpha1 blocker
Minitran      Nitrate
Minocin       Antibiotic
minocycline   Antibiotic
minoxidil     Vasodilator
Mirapex       Anticholinergic (antiparkinson)
mirtazapine   Antidepressant
misoprostol   Antiulcer
mitotane      Antineoplastic
Moban         Phenothiazine
Mobenol       Oral hypoglycemic
Modane Soft   Stool softener
moexipril     ACE inhibitor
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Monitan       Beta blocker
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Nasalcrom   Mast cell stabilizer
Naturetin   Diuretic
Nauseatol   Antihistamine
Navane      Phenothiazine
Nebcin      Antibiotic
nedocromil  Mast cell stabilizer
nefazodone  SSRI antidepressant
NegGram     Antibiotic
nelfinavir   Antiviral agent
Nembutal    Barbiturate
neomycin    Antibiotic
Neoral      Immunosuppressant
neostigmine Cholinesterase inhibitor
Neosynephrine Decongestant
Neothyline  Methylxanthine bronchodilator
Neotrexin   Antineoplastic
Nephronex   Antibiotic
Neurotin    Anticonvulsant
nevirapine  Antiviral agent
Niazhate    Diuretic
nicardipine Calcium channel blocker
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Percocet Narcotic analgesic
Percodan Narcotic analgesic
pergolide Antiparkinson
Pergonal Infertility drug
Periactin Antihistamine
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Perindopril ACE inhibitor
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Prozine  Phenothiazine
pseudoephedrine  Decongestant
Pulmicort  Glucocorticoid
Pulmozyme  Mucolytic
Purinethol  Antineoplastic
Purinol  Uricosuric (antigout)
pyrazinamide  Antituberculosis agent
Pyribenzamine  Antihistamine
quazepam  Benzodiazepine
Questran  Antihyperlipidemic
quetiapine  SSRI antipsychotic
Quibron  Methylxanthine bronchodilator
Quiess  Antihistamine
quinacrine  Antibiotic
Quinaglute  Antidysrhythmic
quinapril  ACE inhibitor
Quinidex  Antidysrhythmic
quinidine  Antidysrhythmic
raloxifene  Estrogen hormone
ramipril  ACE inhibitor
rantiidine  Antiulcer
Raxar  Antibiotic
Reactine  Antihistamine
Reglan  Cholinergic
Regulax  Stool softener
Regutol  Stool softener
Rela  Skeletal muscle relaxant
Relafen  NSAID
Remeron  Antidepressant
Renese  Diuretic
Renormax  ACE inhibitor
repaglinide  Oral hypoglycemic
Requip  Anticholinergic (antiparkinson)
Rescriptor  Antiviral
reserpine  Antihypertensive
Respbid  Methylxanthine bronchodilator
Restoril  Benzodiazepine
Retrovir  Antiviral
Rheumatrex  Antineoplastic
Rhinocort  Glucocorticoid
Rhodis  NSAID
ribavirin  Antiviral
rifabutin  Antituberculosis
Rifadin  Antituberculosis
rifampin  Antituberculosis
rifapentine  Antituberculosis
Rilutek  Anti-ALS
riluzole  Anti-ALS
Rimactane  Antituberculosis
rimantadine  Antiviral
Risperdal  Antipsychotic
risperidone  Antipsychotic
Ritalin  CNS stimulant
ritonavir  Antiviral
Rivotril  Benzodiazepine
rizatriptan  Antimigraine
Robaxin  Skeletal muscle relaxant
Robicillin  Antibiotic
Robidone  Narcotic analgesic
Robimycin  Antibiotic
Robinul  Antiulcer
Robitet  Antibiotic
Rofact  Antituberculosis
Rogaine  Vasodilator
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Ro-sulfiram  Antialcohol
Roxicet  Narcotic analgesic
Roxicodone  Narcotic analgesic
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253
sildenafil  Vasodilator
simvastatin  Antihyperlipidemic
Sinemet  Anticholinergic (antiparkinson)
Sinequan  Tricyclic antidepressant
Singulair  Leukotriene blocker
Sinusol  Antihistamine
Slo-Bid  Methylxanthine bronchodilator
Slo-Phyllin  Methylxanthine bronchodilator
Solazine  Phenothiazone
Solfoton  Barbiturate
Solium  Benzodiazepine
Soma  Skeletal muscle relaxant
Somophyllin  Methylxanthine bronchodilator
Sonazine  Phenothiazone
Soprodol  Skeletal muscle relaxant
Sorbitrate  Nitrate
sotalol  Beta Blocker
Spancap  CNS stimulant
sparfloxacin  Antibiotic
Sparine  Phenothiazone
Spectrobid  Antibiotic
spirapril  ACE inhibitor
spironolactone  Diuretic
Sporanox  Antibiotic
Staticin  Antibiotic
stavudine  Antiviral agent
Stelazine  Phenothiazone
Stilphostrol  Estrogen hormone
Stimate  Antidiuretic hormone
sucralfate  Antiulcer
Sudafed  Decongestant
Sular  Calcium channel blocker
sulfamethoxazole  Antibiotic
sulfapyrazone  Uricosuric (antigout)
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Tilade              Mast cell stabilizer
timolol             Beta blocker
Tizanidine          Skeletal muscle relaxant
tobramycin          Antibiotic
Tobrex              Antibiotic
tocainamide         Antidysrhythmic
Tofranil            Tricyclic antidepressant
tolazamide          Oral hypoglycemic
tolbutamide         Oral hypoglycemic
tolcapone           Anticholinergic (antiparkinson)
Tolcentin           NSAID
Tolinase            Oral hypoglycemic
tolmetin            NSAID
tolterodine         Anticholinergic
Tonocard            Antidysrhythmic
Topamax             Anticonvulsant
topiramate          Anticonvulsant
Toprol              Beta blocker
Toradol             NSAID
Torecan             Antiemetic
toremifene          Antineoplastic
Tornalate           Adrenergic bronchodilator
torsemide           Diuretic
Totacillin          Antibiotic
tramadol            Narcotic analgesic
Trandate            Beta blocker
trandolapril        ACE inhibitor
Tranxene            Benzodiazepine
tramicycprone       MAO inhibitor
Travamine           Antihistamine
trazodone           Antidepressant
Trecator-SC         Antituberculosis
Trental             Antiplatelet
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verapamil  Calcium channel blocker  
Vercyte  Antineoplastic  
Verelan  Calcium channel blocker  
Viagra  Vasodilator  
Vibramycin  Antibiotic  
Vicodin  Narcotic analgesic  
Videx  Antiviral  
Vimicon  Antihistamine  
Viokase  Digestant  
Viracept  Antiviral agent  
Viramune  Antiviral agent  
Virazole  Antiviral agent  
Viroptic  Antiviral agent  
Visken  Beta blocker  
Vistacon  Antihistamine  
Vistacrom  Mast cell stabilizer  
Vistaril Oral  Antihistamine  
Vivactil  Tricyclic antidepressant  
Vivelle  Estrogen hormone  
Vivol  Benzodiazepine  
Vivox  Antibiotic  
Volmax  Adrenergic bronchodilator  
Voltaren  NSAID  
Voltrol  Antivertigo  
warfarin  Anticoagulant  
Warfilone  Anticoagulant  
Wellbutrin  Antidepressant  
Wymox  Antibiotic  
Wytensin  Antihypertensive  
Xanax  Benzodiazepine  
Xeloda  Antineoplastic  
xylometazoline  Decongestant  
zafirlukast  Leukotriene antagonist
Zagam: Antibiotic
zalcitabine: Antiviral agent
Zanaflex: Skeletal muscle relaxant
Zantac: Antiulcer
Zapex: Benzodiazepine
Zarontin: Anticonvulsant
Zaroxolyn: Diuretic
Zebeta: Beta blocker
Zerit: Antiviral agent
Zestril: ACE inhibitor
zidovudine: Antiviral agent
zileuton: Leukotriene antagonist
Zinacef: Antibiotic
Zithromax: Antibiotic
Zocor: Antihyperlipidemic
Zofran: Antiemetic
zolmitriptan: Antimigraine
Zoloft: SSRI antidepressant
zolpidem: Anxiolytic
Zomig: Antimigraine
Zonalon: Tricyclic antidepressant
Zovirax: Antiviral agent
Zyban: Antidepressant
Zydol: Narcotic analgesic
Zyflo: Leukotriene antagonist
Zyloprim: Uricosuric (antigout)
Zyprexa: SSRI antipsychotic
Zyrtec: Antihistamine