



Drug Formulary

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Acetaminophen (APAP)

- Class** Analgesic, Antipyretic.
- Action** Equivalent to aspirin in both analgesic and antipyretic effects. Unlike aspirin, acetaminophen has little effect on platelet function, no effect on homeostasis, and is not known to produce gastric bleeding. Acetaminophen is not an NSAID, as it has no anti-inflammatory properties. Its function was largely a mystery until the early 1990's when it was found that it acted on a variant of cyclooxygenase called COX3 that is only expressed in the central nervous system. Because it does not work on COX1 and COX2 (like ASA) it does not cause the downstream effects on platelets or the immune system.
- Pharmacokinetics** Absorption is rapid, peak 1-2h, duration 3-4h, $\frac{1}{2}$ life 1-3h. APAP is processed in the Liver.
- Contraindications** Use in caution with children afflicted with arthritic or rheumatoid conditions. Use in caution with known thrombocytopenia.
- Adverse effects** N/V, abdominal pain,
- Indications** Fever with or without seizures or Pain.
- Dosing** **Per Protocols: M-09, M-16, PM-03, PM-06, PM-07**

Adenosine

- Class** Antidysrhythmic
- Action** Slows AV node conduction, interrupts reentry pathways. Adenosine works in a variety of receptors grouped into a group called P1 receptors. The true mechanism is somewhat unclear. Adenosine works through the activation of cAMP and coupled G-proteins to cause its cardiac effects.
- Pharmacokinetics** Immediate onset and peak, half-life 10s.
- Contraindications** Known hypersensitivity. Sick Sinus Syndrome. Second or third degree AV block. Use with caution in patients with severe asthma.
- Adverse effects** Flushing, CP, HA, N/V, hypotension
- Indications** Symptomatic (poor perfusion) narrow complex tachycardia w/ pulse
- Dosing** **Per Protocols: C-04, PC-02**

Albuterol

- Class** Sympathomimetic Bronchodilator
- Action** Beta2 adrenergic. Smooth muscle relaxant. Minimal Beta1 effects.
Reduces mucous secretion and edema via histamine inhibition.
- Pharmacokinetics** Onset 5-15m, peak 1-1.5h, duration 3-6h, half-life 3h.
- Contraindications** Known hypersensitivity.
- Adverse effects** Tachycardia, palpitations, peripheral vasodilation, tremors, HA, sore throat, dry mouth, PVCs, N/V.
- Indications** Wheezing due to bronchospasm
- Dosing** **Per Protocols: M-02, PM-01, PR-03, R-04, SO-01, T-04**

Amiodarone

- Class** Antidysrhythmic
- Action** Prolongs the duration of the action potential and refractory period of all Cardiac fibers. Depresses the Phase 0 slope by causing a sodium blockade. Causes a Beta block as well as a weak calcium channel blockade. Therefore it decreases the SA nodes rate of firing, suppresses automaticity, interrupts reentrant pathways and prolongs PR, QRS and QT intervals. Relaxes vascular smooth muscle, decreases peripheral vascular resistance, and increases coronary contractility.
- Pharmacokinetics** Rapid onset, serum concentrations drop to 10% w/in 30-45 minutes.
- Contraindications** Cardiogenic shock, bradycardia, second/third degree block
- Adverse effects** Vasodilation (usually not associated with decreased cardiac output secondary to the negative inotropic effects), hypotension, bradycardia, AV block, increased QT interval, V-Tach
- Indications** Ventricular Arrhythmias or Wide Complex Tachycardia with or without a pulse
- Dosing** **Per Protocols: C-05, CA-03, PCA-03**

Amyl Nitrite

- Class** Vasodilator
- Action** The mechanism of action may be b inducing low levels of methemoglobinemia. Another postulated mechanism is by acting through nitric oxide synthetase. Airway management and provision of supplemental oxygen increase efficacy.
- Pharmacokinetics** Amyl nitrite vapours are absorbed rapidly through the pulmonary alveoli, manifesting therapeutic effects within one minute after inhalation. The drug is metabolised rapidly, probably by hydrolytic denitration; approximately one third of the inhaled amyl nitrite is excreted in the urine.
- Contraindications** Relative Contraindications:
Significant hypotension
Methemoglobinemia >40%
Carbon monoxide poisoning
Absolute Contraindication:
Known Allergy to Medication
- Adverse effects** Headache, Hypotension, Reflex tachycardia, Hypoperfusion (shock)
- Indications** Patients with significant cyanide, cyanogenic compound, or sulfide poisoning.
- Dosing** Per Protocols: SO-05, SO-09

Aspirin

- Class** Analgesic, Antipyretic, NSAID, platelet inhibitor
- Action** Inhibits the formation of prostaglandins associated with pain, fever, and inflammation. Inhibits platelet aggregation by acetylating cyclooxygenase permanently disabling it so that it cannot synthesize prostaglandins and Thromboxanes. Since Thromboxane A2 is important in clotting its absence does not allow blood to clot effectively.
- Pharmacokinetics** Onset 5-30m, peak in 15m-2h, duration is 1-4h.
- Contraindications** Allergy, ulcer, GI bleeding
- Precaution** Patients with known ASA or NSAIDs sensitive Asthma (defer to OLMC)
- Adverse effects** N/V, diarrhea, heartburn, GI bleeding
- Indications** Cardiac type Chest Pain
- Dosing** **Per Protocol: C-01**

Atropine Sulfate

- Class** Parasympatholytic
- Action** Competitive antagonist that selectively blocks all muscarinic responses to acetylcholine. Blocks vagal impulses, thereby increasing SA node discharge, thereby enhancing AV conduction and cardiac output. Potent anti-secretory effects caused by the blocking of acetylcholine at the muscarinic site. Atropine is also useful in the treatment of the symptoms associated with nerve agent poisoning.
- Pharmacokinetics** Rapid onset, peak in 2-4m IV, half-life 2-3h.
- Contraindications** A-Fib, A-Flutter, second degree type II or third degree block. Tachycardia, glaucoma. Use with caution in suspected AMI.
- Adverse effects** Pupil dilation, tachycardia, V-Tach, V-Fib, HA, dry mouth
- Indications** Bradycardia and Organophosphate poisoning
- Dosing** **Per Protocols: C-02, M-14, PC-01**
Per Clinical Procedure: CP-24

Calcium Gluconate

- Class** Electrolyte
- Action** Calcium binds to troponin that connects tropomyosin to actin covering myosins binding site to actin. While this binding site is covered, muscular contraction cannot occur. The exposure of this binding site in the presence of ATP (chemical energy) allows the muscle to contract. Enhancing muscular contraction. Contraction in the heart is therefore increased as is smooth muscles (vascular) contraction. Calcium in its ionic (dissolved) state carries a very positive charge. This charge causes the membrane to be stable if the calcium is too low neurons and cardiac cells have a decreased threshold for activation resulting in tetany. Calcium Gluconate is less potent and less irritating to veins than Calcium Chloride.
- Pharmacokinetics** Onset and peak are immediate
- Contraindications** V-Fib, renal/cardiac insufficiency, patients taking digatalis.
- Adverse effects** Tingling sensation in IV site, hypotension, syncope, cardiac arrest.
- Indications** Calcium Channel Overdose, Beta Blocker Overdose, Hydrofluoric Acid Exposure, Hyperkalemia
- Dosing** **Per Protocols: C-02, CA-02, CA-03, PC-01, PCA-02, SO-08**

Chlorohexadine Gluconate

- Class** skin antiseptic
- Action** Disinfect/clean skin with an antiseptic that contains a minimum of a 2% chlorhexidine-based preparation before sharp appliance insertion.
- Pharmacokinetics** Found to possess a high level of antibacterial activity, low mammalian toxicity and a strong affinity for binding to the skin. The antimicrobial action of CHG is attributed to the disruption of the microbial cell membrane and precipitation of cell contents. Provides residual effect up to 2 days
- Contraindications** None for System use.
- Precaution** Allow the antiseptic to remain on the site and to air dry before catheter insertion, approximately 30 seconds. Chlorohexidine is a skin antiseptic and is not approved for use on environmental surfaces.
- Adverse effects** Handle with care. In case of eye contact, immediately flush eye with plenty of water, if irritation persists, seek medical attention.
- Indications** Necessary skin preparation prior to the application of a sharp appliance including, but not limited to venous catheters, intraosseous infusion needles, lancets, and the delivery of medications or immunizations through syringes either intramuscular, dermal, or subcutaneous.
- Dosing** **Per Clinical Procedures: CP-05, CP-10, CP-28, CP-34, CP-37, CP-38**

Dextrose 50%

- Class** Carbohydrate. Dextrose (aka. glucose) is one of the basic building blocks of all sugars. Glucose is a monomer and is therefore readily processed in the blood. Through glycolysis glucose is turned into pyruvate giving off a small amount of chemical energy (ATP). Pyruvate is further processed through the Citric Acid Cycle (Kreb's Cycle) yielding even more energy (GTP, FADH₂ and NADH) and CO₂. The GTP, FADH₂ and NADH are then converted into a large amount of ATP through the use of a specialized cell membrane and the ability of Oxygen to receive extra protons and carbon to form water and CO₂. Insulin turns excess glucose into glycogen when blood sugars are high. Glucose is a large molecule that forms a ring, this structure is incapable of being absorbed into a cell without a mediator (insulin) and therefore increases damage to epithelium as it floats through the blood stream. It also causes an osmotic pressure as concentrations vary across membranes. The pressure is less with D5 and D10 therefore they are used in pediatrics.
- Action** Principal form of glucose used by the body
- Pharmacokinetics** Rapid absorption in bloodstream
- Contraindications** Use with caution in patients with suspected increased ICP.
- Adverse effects** Patients may complain of warmth, pain, or burning at the injection site. Extravasation causes necrosis.
- Indications** Cardiac Arrest or altered mentation with Glucose level < 50 or Newly Born with heart rate < 60
- Dosing** **Per Protocols: CA-02, M-03, OB-03, PC-01, PM-02, PCA-02**

Diazepam

- Class** Anticonvulsant/sedative, benzodiazepine
- Action** Enhances the action of GABA. GABA is an inhibitory neurotransmitter in the brain (in adults, the glutamate pathway may not be well developed in children and GABA may excite in this population). Glutamate is the excitatory neurotransmitter. By enhancing the GABA effect the brain typically slows transmissions and suppresses the spread of seizure activity throughout the brain. It does not appear to abolish the abnormal focus. Potent skeletal muscle relaxant.
- Pharmacokinetics** Onset 1-5m IV, 15-30m IM. Peak 15m IV, 30m IM. $\frac{1}{2}$ life 20-50m.
- Contraindications** Coma, hypotension.
- Adverse effects** Respiratory depression (especially when pushed fast), lightheadedness, ataxia, neural depression, confusion, slurred speech, amnesia.
- Indications** Seizures
- Dosing** **Per Protocols: M-17, PM-07**

Diltiazem

- Class** Diltiazem hydrochloride is a calcium ion cellular influx inhibitor (slow channel blocker or calcium antagonist).
- Action** Calcium channel blockers are drugs that block the entry of calcium into the muscle cells of the heart and arteries. The entry of calcium is critical for the conduction of the electrical signal that passes from muscle cell to muscle cell of the heart, and signals the cells to contract. It also is necessary in order for the muscle cells to contract and thereby pump blood. In the arteries, the entry of calcium into muscle cells causes contraction of the cells and thereby dilates (widens) the arteries. Thus, by blocking the entry of calcium, calcium channel blockers reduce electrical conduction within the heart, decrease the force of contraction (work) of the muscle cells, and dilate arteries. Dilation of the arteries reduces blood pressure and thereby the effort the heart must exert to pump blood. Combined with decreases in the force of contraction, this leads to a reduced requirement for oxygen by the heart. Dilation of the arteries provides more oxygen-carrying blood to the heart. The combination of reduced demand for oxygen and increased delivery of oxygen prevents angina or heart pain. (Angina occurs when the heart is not getting enough oxygen relative to the amount of work it is doing.) In addition, calcium channel blockers slow electrical conduction through the heart and thereby correct abnormal rapid heartbeats.
- Pharmacokinetics** Diltiazem hydrochloride is extensively metabolized by the liver and excreted by the kidneys and in bile.
- Contraindications** Diltiazem is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, (3) patients with hypotension (less than 90 mm Hg systolic), (4) patients who have demonstrated hypersensitivity to the drug, and (5) patients with acute myocardial infarction and pulmonary congestion.
- Precaution** Cardiac Conduction: Diltiazem prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction
- Adverse effects** Headache, constipation, rash, nausea, flushing, edema, drowsiness, low blood pressure, and dizziness.
- Indications** Atrial Fibrillation with RVR, Paroxysmal Supraventricular Tachycardia
- Dosing** **Per Protocol C-03, C-04**

Diphenhydramine

- Class** Antihistamine, Ethanolamine, Anticholinergic
- Action** Diphenhydramine blocks the effects of Histamine (H1 histamine) on the H1 receptor site through a competitive competition for the peripheral H1 site. When diphenhydramine is bound the H1 site cannot be stimulated preventing the effects of histamines (swelling, etc...). As an H1 blocker diphenhydramine blocks the effects of H1 on its receptor in the cortex as well this causes a change in the cortex neural potassium channels causing neurons in the cortex to have a higher threshold to depolarize. This results in an increase in sedation as a result of the H1 block. As an antihistamine, diphenhydramine one of the most effective antihistamines.
- Pharmacokinetics** Onset of 15m IV, peak 1-4h, ½ life 2-10h.
- Contraindications** Known allergy.
- Adverse effects** Potent anticholinergic agent. Mydriasis, Photophobia, ataxia, tachycardia
- Indications** Hives/Rash or Adult dystonic reaction
- Dosing** **Per Protocols: M-02, M-15, PM-01**

Dopamine

- Class** Sympathomimetic, Catecholamine
- Action** Naturally occurring hormone and preceptor to Norepinephrine. This catecholamine has different effects at different doses due to the sensitivity of receptors at different sites being related to the concentration of dopamine present. At low doses (2-5 mcg/kg/min) Dopamine increases the perfusion of the mesenteric arteries and the kidneys. Low doses can be used to try and perfuse an ischemic bowel or a failing kidney. Has a direct action on alpha and beta-adrenergic receptors. As doses are increased (5-10 mcg/kg/min), beta receptors are stimulated increasing force of contraction as well as heart rate and conduction. As dopamine becomes more concentrated (10-20 mcg/kg/min) the less sensitive peripheral alpha receptors become activated this causes a increase in vascular constriction that increases as the drug becomes more concentrated until at 20 mcg/kg/min the effects are mainly on the peripheral vasculature.
- Pharmacokinetics** Onset <5m, duration <10m, ½ life 2m.
- Contraindications** Pheochromocytoma (adrenal tumors), tachydysrhythmias, HTN
- Adverse effects** Tachydysrhythmias, VF, VT, AMI, N/V, HA.
- Indications** Hypotension unresponsive to fluid therapy
- Dosing** **Per Protocols: C-02, CA-04, M-11, PM-04, PT-03, T-07**

Enalaprilat (Vasotec)

- Class** Enalaprilat (a.k.a. – Enalapril) injection is a sterile aqueous solution for intravenous administration. Enalaprilat is an angiotensin converting enzyme (ACE) inhibitor.
- Action** Intravenous enalaprilat, or oral enalapril, after hydrolysis to enalaprilat, inhibits ACE in human subjects and animals. ACE is a peptidyl dipeptidase that catalyzes the conversion of angiotensin I to the vasoconstrictor substance, angiotensin II. Angiotensin II also stimulates aldosterone secretion by the adrenal cortex. Inhibition of ACE results in decreased plasma angiotensin II, which leads to decreased vasopressor activity and to decreased aldosterone secretion.
- Pharmacokinetics** A clinical response is usually seen within 15 minutes. Peak effects after the first dose may not occur for up to four hours after dosing, although most of the effect is usually apparent within the first hour. This drug is known to be substantially excreted by the kidney.
- Contraindications** Enalaprilat injection is contraindicated in patients who are hypersensitive to any component of this product and in patients with a history of angioedema related to previous treatment with an angiotensin converting enzyme inhibitor and in patients with hereditary, idiopathic angioedema, known pregnancy or Hypotension.
- Precaution** The risk of toxic reactions of this drug may be greater in patients with impaired renal function.
- Adverse effects** Hypertensive patients at risk of excessive hypotension include those with the following concurrent conditions or characteristics: heart failure, hyponatremia, high dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology.
- Indications** Enalaprilat injection is indicated for the treatment of Hypertension in CHF.
- Dosing** **Per Protocol: R-03**

Epinephrine

- Class** Sympathomimetic
- Action** Naturally occurring catecholamine obtained from animal adrenal glands. Acts on alpha and beta adrenergic receptors. The most potent alpha agonist. Beta1: Strengthens myocardial contraction, increase sys BP (may decrease dia BP), increases HR and cardiac output. Beta2: Dilates bronchial smooth muscle and inhibits mucous secretion. Alpha: Constricts bronchiole arterioles, inhibits histamine release, constricts arterioles in the skin, mucous membranes, and kidneys but dilates those in the skeletal muscle. Action is through a natural hormonal mechanism.
- Pharmacokinetics** Onset<2m IV, 3-10m SQ. Peak 5m IV, 20m SQ. Duration 5-10m IV, 20-30m SQ.
- Contraindications** Tachydysrhythmias, coronary artery disease.
- Adverse effects** HA, N/V, tachydysrhythmias, AMI, diaphoresis, anxiety, palpitations.
- Indications** Allergic Reaction/Anaphylaxis, Reactive Airway Disease, PEDI Bradycardia, Cardiac Resuscitation
- Dosing** **Per Protocols: CA-02, CA-03, M-02, PC-01, PM-01, PR-03, PCA-02, PCA-03, R-04, SO-01**
Per Clinical Procedure: CP-24

Fentanyl Citrate

- Class** Narcotic analgesic
- Action** The principal actions of therapeutic value are analgesia and sedation.
- Pharmacokinetics** Opioid (narcotic, CNS-acting) analgesics are derivatives of opium. These drugs modify the perception of pain and provide a sense of euphoria by binding to specific opiate receptors throughout the central nervous system. Many of the characteristics of particular opioids relate to the receptor to which they bind. Fentanyl is classified as a full agonists and binds to mu receptor sites, blocks pain impulses, and produces maximum pain control. Onset immediate, peak 3-5m, duration 30-60m.
- Contraindications** Fentanyl is not indicated for MAOI use, asthma, myasthenia gravis, evidence of hypoperfusion.
- Adverse effects** Fentanyl may cause muscle rigidity, particularly involving the muscles of respiration. In addition, skeletal muscle movements of various groups in the extremities, neck and external eye have been reported during induction of anesthesia with fentanyl; these reported movements have, on rare occasions, been strong enough to pose patient management problems. This effect is related to the dose and speed of injection and its incidence can be reduced by slower administration and lower doses titrated to effect. As with other narcotic analgesics, the most common serious adverse reactions reported to occur with fentanyl are respiratory depression, apnea, rigidity, and bradycardia; if these remain untreated, respiratory arrest, circulatory depression or cardiac arrest could occur. Other adverse reactions that have been reported are hypertension, hypotension, dizziness, blurred vision, nausea, emesis, laryngospasm, and diaphoresis.
- Indications** Acute pain management
- Dosing** **Per Protocols: C-01, M-16, PM-06, PT-01, T-02, T-03**

Furosemide

Class Loop Diuretic

Action Rapid-acting sulfonamide loop diuretic with antihypertensive properties. Decreases renal vascular resistance and increases renal perfusion. Inhibits resorption of Na⁺ and Cl⁻ in the Loop of Henle and also in the proximal and distal tubules. Fluid then follows the Na⁺ and is extracted with the Na⁺. Furosemide is extremely potent when a patient receives it for the first time. Furosemide is not potassium sparing and patients on Lasix should be on a Potassium supplement or hypokalemia may result. Also acts as a venous dilator, reducing preload, therefore cardiac workload.

Pharmacokinetics Onset 5-10m, peak diuresis effect 20-60m, duration 6h, ½ life 30m.

Contraindications Dehydration, hypokalemia, hepatic coma, SBP<100.

Adverse effects Hypokalemia, hypotension, dehydration, urinary urgency.

Indications CHF/Pulmonary Edema and Crush > 4 hrs (Adult only)

Dosing **Per Protocols: T-03**

Glucagon

- Class** Hormone
- Action** Causes a breakdown of stored blood glycogen to glucose and inhibits glycogen synthesis. Glucagon acts by binding to glucagon receptor sites and stimulating a secondary messenger through the increase of adenylate cyclase. Beta stimulation causes an increase in the adenylate cyclase. Therefore glucagon has been known to have beta like effects just as Beta drugs such as Epinephrine are known to stimulate Glycogenolysis in the liver.
- Pharmacokinetics** Onset 5-20m, peak 30m, duration 1-1.5h. $\frac{1}{2}$ life 30m.
- Contraindications** Not efficacious in poorly nourished individuals as they have no glycogen stores.
- Adverse effects** N/V, HA
- Indications** Hypoglycemia < 50 if unable to obtain IV access for D50
- Dosing** **Per Protocols: C-02, CA-02, M-03, PC-01, PM-02, PCA-02**

Haloperidol

- Class** Antipsychotic
- Action** The precise mechanism of action has not been clearly established.
- Pharmacokinetics** This drug is known to be substantially excreted by the kidney.
- Contraindications** Severe toxic central nervous system depression or comatose states from any cause and in individuals who are hypersensitive to this drug or have Parkinson's disease.
- Precaution** Elderly Patients with Dementia-Related Psychosis.
- Adverse effects** Tachycardia, hypotension, and hypertension have been reported. QT prolongation and/or ventricular arrhythmias have also been reported, in addition to ECG pattern changes compatible with the polymorphous configuration of torsade de pointes, and may occur more frequently with high doses and in predisposed patients. Symptoms of dystonia, prolonged abnormal contractions of muscle groups, may occur in susceptible individuals during the first few days of treatment. Dystonic symptoms include: spasm of the neck muscles, sometimes progressing to tightness of the throat, swallowing difficulty, difficulty breathing, and/or protrusion of the tongue. While these symptoms can occur at low doses, they occur more frequently and with greater severity with high potency and at higher doses of first generation antipsychotic drugs. An elevated risk of acute dystonia is observed in males and younger age groups. The risk of toxic reactions of this drug may be greater in patients with impaired renal function.
- Indications** Haloperidol is used to treat certain mental/mood disorders (e.g., schizophrenia, schizoaffective disorders). It can also help prevent suicide in people who are likely to harm themselves. It also reduces aggression and the desire to hurt others. It can decrease negative thoughts and hallucinations. Haloperidol can also be used to treat uncontrolled movements and outbursts of words/sounds related to Tourettes disorder. Haloperidol is also used for severe behavior problems in hyperactive children when other treatments or medications have not worked. Haloperidol is a psychiatric medication (antipsychotic-type) that works by helping to restore the balance of certain natural substances in the brain (neurotransmitters).
- Dosing** **Per Protocol: M-05**

Hurricane/Cetacaine Spray

Class Topical anesthetic; Contains 14-20% Benzocaine

Action Blocks conduction of impulses at the sensory nerve endings.

Pharmacokinetics Benzocaine is an ester, a compound made from the organic acid PABA (para-aminobenzoic acid) and ethanol.

Pain is caused by the stimulation of nerve endings. When the nerve endings are stimulated, sodium enters the nerve ending, which causes an electrical signal to build up in the nerve. Once the electrical signal becomes big enough, it is able to travel to the brain, which then interprets this as pain.

Esters of PABA work as a chemical barrier, stopping the sodium from being able to enter the nerve ending.

Contraindications Known sensitivity to Benzocaine anesthetics.

Adverse effects Benzocaine is a well-known cause of methemoglobinemia. Because it may be used in topical creams with a concentration as much as 20%, it is not difficult to administer a dose sufficient to cause this problem.

Indications To facilitate nasal intubation attempts in patients with a gag reflex.

Dosing **Per Clinical Procedure: CP-44**

Ibuprofen

- Class** Non-Steroidal Anti-Inflammatory Drug (NSAID)
- Action** Ibuprofen possesses analgesic and antipyretic activities. Its mode of action, like that of other NSAIDs, is not completely understood, but may be related to prostaglandin synthetase inhibition, by blocking the enzyme in your body that makes prostaglandins. Decreasing prostaglandins helps to reduce pain, swelling, and fever.
- Pharmacokinetics** Ibuprofen is rapidly absorbed. Peak serum ibuprofen levels are generally attained one to two hours after administration. Ibuprofen is rapidly metabolized and eliminated in the urine. The excretion of ibuprofen is virtually complete 24 hours after the last dose. The serum half-life is 1.8 to 2.0 hours.
- Contraindications** In patients with known hypersensitivity and should not be given to patients who have experienced asthma, urticaria, or allergic-type reactions after taking aspirin or other NSAIDs. Severe, rarely fatal, anaphylactic-like reactions to NSAIDs have been reported in such patients. Contraindicated for the treatment of peri-operative pain in the setting of coronary artery bypass graft (CABG) surgery.
- Precaution** Late Pregnancy, aspirin-sensitive asthma, coagulation disorders or patients receiving anticoagulants should be carefully monitored.
- Adverse effects** Heart attack, stroke, high blood pressure, heart failure from body swelling (fluid retention), kidney problems including kidney failure, bleeding and ulcers in the stomach and intestine, low red blood cells (anemia), life-threatening skin reactions, life-threatening allergic reactions, liver problems including liver failure, asthma attacks in people who have asthma.
- Indications** Ibuprofen is a non-steroidal anti-inflammatory drug (NSAID), which relieves pain and swelling (inflammation). It is used to treat headaches, muscle aches, backaches, dental pain, menstrual cramps, arthritis, or athletic injuries. This medication is also used to reduce fever and to relieve minor aches and pains due to the common cold or flu.
- Dosing** **Per Protocol: M-09**

Ipratropium Bromide

- Class** Parasympatholytic Bronchodilator
- Action** Anticholinergic agent, chemically closely related to atropine and has the same actions as Atropine. Acts directly on the smooth muscle and decreases secretions. Reduces the vagally mediated reflex bronchospasm caused by inhaled irritants.
- Pharmacokinetics** 10% of inhaled dose reaches lower airway, 0.5% reaches systemic distribution. Peak 1.5-2h, duration 4-6h, $\frac{1}{2}$ life 1.5-2h.
- Contraindications** Narrow-angle Glaucoma, Hypersensitivity to Atropine or allergy to soy products
- Adverse effects** Dry mouth, HA, cough, dries secretions
- Indications** Obstructive Airway Disease, Reactive Airway Disease
- Dosing** **Per Protocols: PR-03, R-04, T-04**

Lidocaine

- Class** Antidysrhythmic, Sodium channel blocker
- Action** Raises the threshold for ventricular contractions and lowers the threshold for defibrillation and cardioversion. Suppresses automaticity in the His-purkinje system and by elevating the electrical stimulation threshold of ventricular contractions. This is accomplished by blocking the rapid influx of Na⁺ during the initial phase of depolarization. Typically shortens the action potential and the refractory period secondary to a blockade of sodium channels that usually (in procainamide's blockade) continue to function normally through phase 2 of the action potential. Lidocaine functions well in hyperkalemic and acidotic states therefore it works well on ischemic tissues.
- Pharmacokinetics** Onset 3m, peak 5-7m, duration 10-20m, ½ life 1.5-2h.
- Contraindications** CHF, shock, use caution in the elderly.
- Adverse effects** Seizures, slurred speech, AMS
- Indications** Pain Management for IO Infusion, Cardiac Arrest and Post Resuscitation Care
- Dosing** **Per Protocols: C-05, CA-03, M-08, PCA-03, U-02**
Per Clinical Procedures: CP-24, CP-37, CP-38

Magnesium Sulfate

- Class** Electrolyte
- Action** Molecularly Mg⁺ is very similar to Ca as it has the same electron valence. Because of this it chemically very similarly to Ca⁺ and in some reactions in the body. Ca⁺ is significantly larger than Mg⁺ therefore Mg⁺ does not adequately replace it in cases that are not purely chemical. Because of these qualities Mg⁺ can prevent Ca⁺ from binding to Troponin and prevent muscles from contracting as described in the action for “Calcium Gluconate”. Because of its extremely positive charge it also blocks neuromuscular transmission by changing the electric potentials threshold.
- Pharmacokinetics** Onset immediate, duration 30m
- Contraindications** Renal disease, AV block, previous myocardial damage.
- Adverse effects** Hypotension, asystole, cardiac arrest, respiratory/CNS depression, flushing, sweating.
- Indications** Obstetrical Emergencies/ Seizures (adult only), Reactive Airway Disease, Toxic Exposure (Hydrofluoric Acid), Pulseless Arrest, Tachycardia w/ pulse (adults only).
- Dosing** **Per Protocols: C-05, CA-03, OB-02, PR-03, PCA-03, R-04**

Methylprednisolone

- Class** Glucocorticosteroid
- Action** Adrenal Corticosteroid with fewer sodium and water retention effects than hydrocortisone. Methylprednisolone alters the body's immune response. Swelling is reduced because it prevents the white blood cells traveling to the area.
- Pharmacokinetics** $\frac{1}{2}$ life of 2.5-3.5h.
- Contraindications** None for anaphylaxis.
- Adverse effects** Peptic ulcer, hyperglycemia, hypokalemia, impaired ability to fight infection, in the prolonged use the side effects are so numerous they are the subject of several books.
- Indications** Allergic Reaction/Anaphylaxis, Reactive Airway Disease
- Dosing** **Per Protocols: M-02, PM-01, PR-03, R-04, SO-01**

Midazolam

- Class** Sedative, Benzodiazepine
- Action** As a Benzodiazepine this drug functions on GABA similarly to the action of “Diazepam”. Midazolam is a short-acting muscle-relaxant, anticonvulsant, in addition to these effects Midazolam also has anterograde amnestic effects, it is therefore preferred prior to cardioversion.
- Pharmacokinetics** Onset 3-5m IV, 6-14 IN, peak 20-60, duration < 2h, ½ life 1-4h.
- Contraindications** Shock, acute narrow glaucoma, coma
- Adverse effects** Resp. depression, hypotension, bradycardia, HA, N/V
- Indications** Seizures, Violent Patient/Chemical Sedation, Sedation for Electrical therapy, Hyperthermia (Environmental) and Induced Hypothermia.
- Dosing** **Per Protocols: C-02, C-03, C-04, C-05, CA-04, M-05, M-06, M-07, M-10, M-15, M-17, PC-02, PC-03, PM-07**

Naloxone

- Class** Narcotic Antagonist
- Action** Competitive antagonist for opioids competing for opiate receptor sites in the brain. Displaces narcotic molecules from opiate receptors through this competition. Higher doses are needed to overcome overdoses of opiates that have a higher affinity for the opiate receptor in the brain.
- Pharmacokinetics** Onset <2m, peak <2m, duration 2-20m, ½ life 60-90m.
- Contraindications** Neonates with narcotic-addicted mothers.
- Adverse effects** Withdrawal symptoms.
- Indications** narcotic overdose
- Dosing** **Per Protocols: M-03, OB-03, PM-02**
Per Clinical Procedure: CP-24

Nitroglycerin

- Class** Nitrate
- Action** Potent vasodilator with antianginal, anti-ischemic, and antihypertensive effects. Relaxes vascular smooth muscle by an unknown mechanism. Decreases peripheral vascular resistance, preload, and afterload.
- Pharmacokinetics** Onset 1-3m SL, 30m transdermal. Peak 5-10m SL. Duration is 20-30m SL, 3-6h transdermal.
- Contraindications** Hypotension, hypovolemia, severe bradycardia or tachycardia, use of erectile dysfunction drugs within past 24hrs up to 48 hours depending on use of extended release medications.
- Adverse effects** Hypotension, HA, syncope, tachycardia.
- Indications** Chest Pain, CHF/Pulmonary Edema
- Dosing** **Per Protocols: C-01, R-03**

Ondansetron

- Class** Antiemetic, 5-HT₃, receptor antagonist
- Action** Ondansetron is a selective 5-HT₃ receptor antagonist. While its mechanism of action has not been fully characterized, Ondansetron is not a dopamine-receptor antagonist. Serotonin receptors of the 5-HT₃ type are present both peripherally on vagal nerve terminals and centrally in the chemoreceptor trigger zone of the area postrema. It is not certain whether Ondansetron's antiemetic action is mediated centrally, peripherally, or in both sites. The released serotonin may stimulate the vagal afferents through the 5-HT₃ receptors and initiate the vomiting reflex.
- Pharmacokinetics** Rapid onset, half-life 3-4 hours
- Contraindications** If the patient is sensitive to or has ever had an allergic reaction to ondansetron hydrochloride, do not give Zofran. If drugs similar to Zofran (for instance, Anzemet or Kytril) have caused a reaction, Zofran may cause one too. If your patient has phenylketonuria (an excess of the amino acid phenylalanine) Zofran also contains this substance.
- Adverse effects** Blurred vision or temporary blindness, fever, slow heart rate, trouble breathing, anxiety, agitation, shivering, feeling light-headed or fainting
- Indications** Nausea and/or Vomiting
- Dosing** Per Protocols: M-08, M-13, M-10, PM-05

Oral Glucose Dextrose 40%

- Class** Carbohydrate. Dextrose (aka. glucose) is one of the basic building blocks of all sugars. Glucose is a monomer and is therefore readily processed in the blood. Through glycolysis glucose is turned into pyruvate giving off a small amount of chemical energy (ATP). Pyruvate is further processed through the Citric Acid Cycle (Kreb's Cycle) yielding even more energy (GTP, FADH₂ and NADH) and CO₂. The GTP, FADH₂ and NADH are then converted into a large amount of ATP through the use of a specialized cell membrane and the ability of Oxygen to receive extra protons and carbon to form water and CO₂.
- Action** Principal form of glucose used by the body readily absorbed via the digestive tract.
- Pharmacokinetics** Rapid absorption in bloodstream
- Contraindications** Patients that are unconscious or unable to control their airway, not recommended for patients < 2 years of age.
- Adverse effects** Airway compromise during administration
- Indications** Patients with blood glucose level < 50 with altered mentation and who can control their airway and are able to swallow.
- Dosing** **Per Protocols: M-03, PM-02**

Otrivin (Afrin)

- Class** Decongestant (topical)
- Action** A direct-acting sympathomimetic amine. Xylometazoline acts on alpha-adrenergic receptors in the nasal mucosa to produce vasoconstriction, resulting in decreased blood flow and decreased nasal congestion.
- Pharmacokinetics** rapid onset, up to 10 hours duration
- Contraindications** known hypersensitivity to this Medication
- Precaution** Patients sensitive to other nasal decongestants may be sensitive to this medication also.
- Adverse effects** Coronary artery disease or Heart disease, including angina or hypertension (condition may be exacerbated due to drug-induced cardiovascular effects)
- Indications** Nasal preparation prior to Nasal Tracheal Intubation attempt
- Dosing** **Per Clinical Procedure: CP-44**

Pralidoxime

- Class** Cholinesterase Reactivator
- Action** Organophosphates inhibit cholinesterase by phosphorylation of the enzyme. Pralidoxime reactivates the cholinesterase by removing the phosphoryl group that is bound to the ester group. In this reaction both the organophosphate and the pralidoxime are mutually inactivated. These products undergo rapid metabolism, leading to the removal of the organophosphate.
- Pharmacokinetics** Binds nerve agent and breaks agent-enzyme bond
Less effective after nerve agent aging occurs
- Soman (GD) ages in 2 minutes, Other nerve agents age over 24-48 hours
- Contraindications** There are no known absolute contraindications for the use of pralidoxime. Relative contraindications include known hypersensitivity to the drug and other situations in which the risk of its use clearly outweighs possible benefit.
- Adverse effects** Blurred or double vision; difficulty in focusing your eyes; difficulty in speaking; difficult or rapid breathing; dizziness; fast heartbeat; muscle stiffness or weakness; pain at the place of injection (after injection into a muscle) drowsiness; headache; nausea.
- Indications** Toxic Exposure — Organophosphates/Nerve Agents
- Dosing** **Per Protocol: M-14**

Proparacaine Hydrochloride

- Class** is a topical anesthetic prepared as a sterile aqueous ophthalmic solution.
- Action** a rapid acting local anesthetic suitable for ophthalmic use. The onset of anesthesia usually begins within 30 seconds and lasts a relatively short period of time. The main site of anesthetic action is the nerve cell membrane where proparacaine interferes with the large transient increase in the membrane permeability to sodium ions that is internally produced by a slight depolarization of the membrane. As the anesthetic action progressively develops in a nerve, the threshold for electrical stimulation gradually increases and the safety factor for conduction decreases; when this action is sufficiently well developed, block of conduction is produced. The exact mechanism whereby proparacaine and other local anesthetics influence the permeability of the cell membrane is unknown; however, several studies indicate that local anesthetics may limit sodium ion permeability through the lipid layer of the nerve cell membrane. This limitation prevents the fundamental change necessary for the generation of the action potential.
- Contraindications** patients with known hypersensitivity
- Precaution** Proparacaine hydrochloride should be used cautiously and sparingly in patients with known allergies, cardiac disease, or hyperthyroidism. The long-term toxicity of proparacaine is unknown, prolonged use may possibly delay wound healing. Although exceedingly rare with ophthalmic application of local anesthetics, it should be borne in mind that systemic toxicity (manifested by central nervous system stimulation followed by depression) may occur.
- Adverse effects** CNS depression, Allergic reaction
- Indications** anesthetic used for eye procedures related to eye injury or contamination
- Dosing** **Per Protocol: M-08**

Pyridoxine

- Class** Vitamin B6
- Action** Vitamin B6 acts as a coenzyme necessary for the metabolism of fats, proteins and carbohydrates. It helps with the functioning of the immune and nervous systems and the formation red blood cells. Asthma, diabetes and PMS (premenstrual syndrome) sufferers may benefit from this vitamin, plus it may also lower cholesterol and help prevent heart disease. Along with tryptophan, vitamin B6 is used to produce niacin.
- Contraindications** None in the Toxic Exposure of Hydrazine
- Precaution** Pyridoxine is safe for most people. In some people, pyridoxine might cause nausea, vomiting, stomach pain, loss of appetite, headache, tingling, sleepiness, and other side effects.
- Indications** Toxic Exposure of Hydrazine
- Dosing** **Per Protocol: SO-07**

Sodium Bicarbonate

- Class** Electrolyte
- Action** Short-acting, potent, systemic antacid. Immediately raises the pH of blood plasma by buffering excess hydrogen ions. This occurs because the Na⁺ (sodium) and the HCO₃⁻ (bicarbonate ion) separate in solution. While separate the negative charge on the bicarbonate is able to accept (and will prefer over sodium) hydrogen ions. The HCO₃⁻ then becomes H₂CO₃ which the body will turn into water and CO₂. In tricyclic overdoses the Na⁺ ion is important also in its use to attempt to overcome the sodium blockade that occurs.
- Pharmacokinetics** Onset immediate, duration 1-2h.
- Contraindications** None on an indicate condition.
- Adverse effects** Metabolic acidosis, hypokalemia, fluid overload.
- Indications** Overdose, Hyperkalemic Arrest, Neonatal Resuscitation (OLMC), Burns, Crush Injuries
- Dosing** **Per Protocols: C-05, CA-02, CA-03, M-15, PC-01, PCA-02, T-03, SO-04**

Sodium Nitrite

- Class** Nitrates and Nitrites
- Action** The mechanism of action may be by inducing low levels of methemoglobinemia. Another postulated mechanism is by acting through nitric oxide synthetase. Airway management and provision of supplemental oxygen increase efficacy.
- Pharmacokinetics** Sodium nitrite antagonizes acetylcholine, epinephrine, and histamine effects; sodium nitrite potentiates hypotensive effects and/or anticholinergic effects of tricyclic antidepressants, antihistamines, and meperidine and related CNS depressants; ethanol increases the toxicity of amyl nitrite.
- Contraindications** Relative Contraindications:
Significant hypotension
Methemoglobinemia >40%
Carbon monoxide poisoning
Absolute Contraindications:
Known Allergy to Medication
- Adverse effects** Cardiovascular: Tachycardia, syncope, cyanosis, hypotension (associated with rapid infusion), flushing
Central nervous system: Dizziness, headache
Gastrointestinal: Nausea, vomiting
Miscellaneous: Methemoglobin formation
- Indications** Significant cyanide, cyanogenic compound, or sulfide poisoning
- Dosing** **Per Protocols: SO-05, SO-09**

Sodium Thiosulfate

- Class** Crystalline Salt
- Action** Sodium thiosulfate provides sulfane sulfur which is needed by the hepatic enzyme rhodanese to change cyanide into thicyanate that is then excreted in the urine. The availability of sulfane sulfur is a rate-limiting step for this reaction. Endogenous supplies of sulfane sulfur (mostly in sulfur-containing amino acids) are insufficient during cyanide or cyanogenic compound poisoning.
- Pharmacokinetics** When administered intravenously, it is distributed in the extracellular fluid and then rapidly excreted via the urine.
- Contraindications** **Relative Contraindications:**
An unprotected, at risk airway. This is because many patients will vomit and could aspirate.
Absolute Contraindications:
NONE
- Adverse effects** Cardiovascular: Hypotension
Central nervous system: Coma, CNS depression secondary to thiocyanate intoxication, psychosis, confusion
Dermatologic: Contact dermatitis
Local: Local irritation
Neuromuscular & skeletal: Weakness
Otic: Tinnitus
- Indications** Significant cyanide or cyanogenic compound poisoning
- Dosing** **Per Protocol: SO-05**

Terbutaline Sulfate

- Class** beta-adrenergic agonist bronchodilator available as a sterile, nonpyrogenic, aqueous solution in vials, for subcutaneous administration.
- Action** Terbutaline is a beta-adrenergic receptor agonist. In vitro and in vivo pharmacologic studies have demonstrated that terbutaline exerts a preferential effect on beta2-adrenergic receptors. While it is recognized that beta2-adrenergic receptors are the predominant receptors in bronchial smooth muscle, data indicate that there is a population of beta2-receptors in the human heart, existing in a concentration between 10% to 50%. The precise function of these receptors has not been established. Controlled clinical studies have shown that terbutaline relieves bronchospasm in acute and chronic obstructive pulmonary disease by significantly increasing pulmonary flow rates (e.g., an increase of 15% or more in FEV1). After subcutaneous administration of 0.25 mg of terbutaline, a measurable change in expiratory flow rate usually occurs within 5 minutes, and a clinically significant increase in FEV1 occurs within 15 minutes. The maximum effect usually occurs within 30 to 60 minutes, and clinically significant bronchodilator activity may continue for 1.5 to 4 hours. The duration of clinically significant improvement is comparable to that observed with equimilligram doses of epinephrine.
- Contraindications** Terbutaline sulfate injection is contraindicated in patients known to be hypersensitive to sympathomimetic amines or any component of this drug product.
- Precaution** Terbutaline, as with all sympathomimetic amines, should be used with caution in patients with cardiovascular disorders, including ischemic heart disease, hypertension, and cardiac arrhythmias; in patients with hyperthyroidism or diabetes mellitus; and in patients who are unusually responsive to sympathomimetic amines or who have convulsive disorders.
- Adverse effects** Significant changes in systolic and diastolic blood pressure have been seen and could be expected to occur in some patients after use of any beta-adrenergic bronchodilator. Immediate hypersensitivity reactions and exacerbations of bronchospasm have been reported after terbutaline administration.
- Indications** Respiratory distress in patients > 36 Kg during remote rescue events with significant extraction time and transport delay
- Dosing** **Per Protocol: SO-01**

Vecuronium Bromide

- Class** Non-depolarizing neuromuscular blocker
- Action** The relaxation of skeletal muscles which facilitates endotracheal intubation and mechanical ventilation.
- Pharmacokinetics** Binds to receptors and prevents acetylcholine (Ach) from stimulating receptors. It competes with Ach for nicotinic receptor binding sites. The blockade is competitive, hence muscle paralysis occurs gradually.
- Contraindications** None
- Adverse effects** Prolonged paralysis
- Indications** To facilitate invasive cooling procedure.
- Dosing** **Per Protocol: CA-04**

Xylocaine Gel

- Class** anesthetic
- Action** stabilizes the neuronal membrane by inhibiting the ionic fluxes required for the initiation and conduction of impulses thereby, effecting local anesthetic action. Local anesthetics of the amide type are thought to act within the sodium channels of the nerve membrane.
- Pharmacokinetics** After application local anesthesia is achieved within 5 minutes. Duration of anesthesia is approximately 20 - 30 minutes.
- Contraindications** Lidocaine HCl is contraindicated in patients with a known history of hypersensitivity to local anesthetics of the amide type.
- Indications** Nasal preparation prior to Nasal Tracheal Intubation attempt
- Dosing** **Per Clinical Procedure: CP-32, CP44**