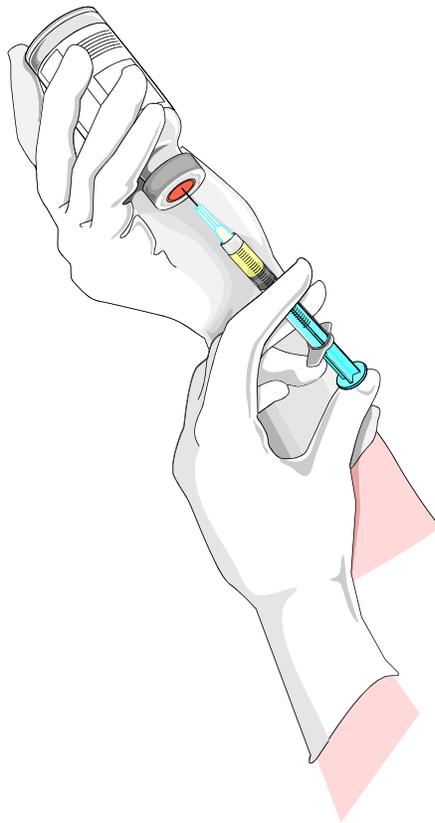


ADVANCED LIFE SUPPORT

Drug Guide for Paramedics



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Medicine and pharmacology is a constantly changing science and not all therapies are clearly established. New research changes drug and treatment therapies daily. The author and publisher of this document have used his best efforts to provide information that is up-to-date and accurate and is generally accepted within medical standards at the time of publication. However, as medical science is constantly changing and **human error is always possible**, the author and publisher does not warrant that the information in this document is accurate or complete, nor is the author responsible for omissions or errors in the document or for the results of using this information. The reader should confirm the information in this document from other sources prior to use. In particular, all drug doses, indications, and contraindications should be confirmed. In addition, *the drug indications and dosages described in this guide are based on general guidelines and principles of drug administration and do not replace or supersede your Medical Directives or Standing Orders.*

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Seven “Rights” of Drug Administration

1. Right Patient?

- Is this patient right for this drug?
- Is this drug contraindicated because of medical history, allergies, drug interaction, presenting condition, heart rate, blood pressure, mental status, etc?

2. Right Drug?

- some drugs come in similar ampoules, vials or nebulas (e.g. epinephrine and morphine, naloxone and midazolam) – *always* check the drug when you pull it out of the kit for the name, dose, concentration and for fluid clarity and expiry date. Check the drug again before administering it.
- syringes with left over medication must be labelled with the drug name and concentration per ml.
- A narcotic should be checked by two people prior to administration (except when alone in the back of the ambulance)

3. Right Dose?

- double check dosage calculation - have partner do the same when practical
- Is your estimation of the patient’s weight reasonable?
- The Broselow® tape is recommended for weight estimation in paediatrics

4. Right Time?

- Follow dosing intervals listed in Medical Directives.
- Remember that repeated doses of a drug may have an added effect.
- Timing may be critical to maintain a therapeutic drug level

5. Right Route?

- Which route is most appropriate for this patient – e.g. SC, IM or IV.
- In the case of anaphylaxis for example, SC may be acceptable in the early stages, however, once shock has set in, the IM route is better.

6. Right to know?

- patient has a right to be informed about the drug; What the drug is and what it does; benefits and risks; a right to sound medical advice

7. Right to refuse

- You **must** obtain permission from the patient for any intervention
- The patient has the right to refuse treatment at any time
 - e.g. it’s not uncommon for patients to refuse ASA for various reasons
 - e.g. some patients will refuse Adenosine because it causes them great physical distress or they know it hasn’t worked for them in the past.
- Assess patient’s “capacity” in the event of a refusal and use the Base Hosp. Physician and/or supervisor for assistance

Adenosine (Adenocard)

- Classification:
- antiarrhythmic
- Pharmacodynamics:
- naturally occurring nucleoside that stimulates specific adenosine receptors. This results in activation of acetylcholine sensitive potassium channels (efflux of potassium) and blockade of calcium influx in the SA node, atrium and AV node. The cells become hyperpolarized and this blunts SA node discharge, slows AV conduction and increases the AV node refractory period. AV nodal conduction may be completely blocked.
 - interrupts AV nodal re-entry and other AV node dependant tachyarrhythmias
- Pharmacokinetics: Intravenous
- ONSET
- 20-30 seconds
- PEAK
- unknown
- HALF-LIFE
- 6-10 seconds
- DURATION
- 1-2 minutes
- Indications:
- conversion of supraventricular tachycardia (SVT) / paroxysmal supraventricular tachycardia (PSVT) including that associated with Wolff-Parkinson-White syndrome
- Contraindications:
- hypersensitivity, 2nd or 3rd degree AV block, sick sinus syndrome
- Precautions:
- may worsen bronchospasm in asthmatics and some patients with COPD
 - flushing and chest pain may occur briefly after administration.
- drug to drug:*
- higher than normal doses of Adenosine may be required for patients on xanthines (eg. theophylline).
 - lower than normal doses (i.e. 3 mg or less) should be used for patients on dipyridamole (Persantine®) as this drug potentiates Adenosine.
 - the effects of Adenosine are prolonged in patients taking Carbamazepine (anti-convulsant) and in heart transplant recipients (denervated hearts)
- Adverse effects
- crushing chest pain, flushing, SOB, N/V, lightheadedness, dizziness, syncope, etc
 - explain to the patient they will likely experience some of the above symptoms

Dosage:

- 6 mg IV bolus (FAST!) -followed by an immediate 20-30 cc of NS or R/L flush - run ECG strip as drug is being given
- 12 mg IV bolus (FAST!) -followed by an immediate 20-30 cc of NS or R/L flush - may be repeated in 1-2 minutes if the first dose is ineffective.
- Note: Adenosine must be given very quickly and in the IV site closest to the central circulation (e.g. antecubital, external jugular, central line). It should always be immediately followed by a 20-30 cc flush of NS or R/L to make sure that all of the drug is cleared from the IV tubing and delivered to the intended site

Pediatric

- 0.1 - 0.2 mg/kg rapid push (flush with 2-20 cc IV fluid depending weight of child)

SPECIAL NOTES:

- has a $\geq 90\%$ successful conversion of PSVT rate when the full dose is given (Crankin et al, 1989; Garrat et al, 1989; DiMarco et al, 1990)
- has an extremely short half life of 10 seconds or less – consequently, as many as 40% of patients may revert back into PSVT
- Once the drug is given, the patient may be experience a period of asystole of 3-15 seconds. A variety of other rhythms may also appear on the ECG (e.g. second or third-degree heart block). Because of the drug's short half life, these effects are generally self-limiting
- *Sometimes rapid Atrial Fibrillation is difficult to distinguish from a regular SVT. If that occurs turn the volume up on the cardiac monitor. This will provide an auditory clue that the rhythm is irregularly irregular. Map out the R-R interval to see if the rhythm is regular (SVT) or irregular (A. Fib.). Use the patient's history and medications as a guide – i.e. the elderly patient on digoxin and coumadin is more likely to be in an atrial fib. The younger patient is more likely to be in an SVT.*
- *Transport of the patient should not be delayed as other treatments/drugs may be required in hospital should SVT/PSVT recur*

Amiodarone

Classification:

- antiarrhythmic

Pharmacodynamics:

- considered a class III antiarrhythmic
- also possesses electrophysiologic characteristics of all 4 Vaughan Williams classes
- Like Class I drugs, amiodarone blocks sodium channels at rapid pacing frequencies
- like class II drugs, it exerts sympatholytic activity through beta-adrenoreceptor (weak) antagonism
- Class II type effects negative chronotropic effect in nodal tissues
- **class III effect:** lengthens the cardiac action potential – prolongs the QT interval
- In addition to blocking sodium channels, amiodarone blocks myocardial potassium channels, which contributes to slowing of conduction and prolongation of refractoriness
- antisympathetic action and block of calcium and potassium channels are responsible for the negative dromotropic effects on the sinus node and for the slowing of conduction and prolongation of refractoriness in the atrioventricular (AV) node.

Pharmacokinetics:

ONSET

- ? minutes

PEAK

- 10-15 minutes

HALF-LIFE

- ?

DURATION

- ?

Indications:

- ventricular fibrillation
- ventricular tachycardia
- SVT
- atrial fibrillation

Contraindications:

- Hypersensitivity
- Cardiogenic shock
- Marked sinus bradycardia
- 2nd or 3rd degree AV block

Precautions:

- Poor liver function

Warning

- Hypotension of the most common side effect during IV infusion (~ 39% of patients in one trial) Slow the infusion.
- Thyroid dysfunction – may cause hypo or hyperthyroidism (negligible for IV administration)

- Pulmonary interstitial abnormalities (1/1 000 patients treated with amiodarone i.v. in clinical studies developed pulmonary fibrosis)
 - May cause prolongation of the QT interval (>500ms may lead to Torsade de Pointes)
- Drug to drug interactions
- Amiodarone interacts with numerous other drugs, however these effects may be more relevant to oral administration in some cases
 - risk of life-threatening cardiac arrhythmias, including torsades de pointes, may be increased in a patient taking Vardenafil
 - Amiodarone may increase serum concentrations of disopyramide, flecainide, procainamide, quinidine
 - May potentiate the effects of warfarin, Dabigatran, beta-blockers, calcium channel blockers (eg, diltiazem, verapamil), Digoxin, Clozapine, Cyclosporine, Dextromethorphan, Fentanyl, Fingolimod, loratadine, trazodone
 - May cause QTc prolongation with or without torsades de pointes when combined with Azole antifungals (eg, itraconazole), fluoroquinolones (eg, moxifloxacin), macrolide antibiotics (eg, azithromycin, telithromycin), Quinupristin/Dalfopristin
 - Cimetidine and protease inhibitors (e.g. ritonavir) may increase the plasma concentrations of Amiodarone
 - May diminish the effects of Clopidogrel
 - Effects of amiodarone may be reduced by the concomitant use of Cholestyramine
- Dosage:
- 150-300mg loading dose
- Pediatric
- 5mg/kg in cardiac arrest
- Special Notes:
- if the patient becomes hypotensive or bradycardic during the infusion of amiodarone, slow down the infusion or discontinue

Aspirin (ASA)

- Classification:
- antiplatelet
 - antithrombotic
 - aspirin also falls under many other functional classifications
- Pharmacodynamics:
- inhibits the formation of thromboxane A₂ which is a potent platelet aggregate and vasoconstrictor
- Pharmacokinetics:
- ONSET
- 15-30 minutes
- PEAK
- 1-2 hours
- HALF-LIFE
- 2-3 hours (low dose)
- DURATION
- 4-6 hours
- Indications:
- chest pain or atypical symptoms consistent with cardiac ischemia/AMI
- Contraindications:
- allergy to aspirin or other non-steroidal anti-inflammatory (NSAIDS) agents. This includes many non-aspirin/non-Tylenol pain relievers such as Advil
 - asthma
 - recent head injury, stroke or acute bleeding (significant) of any kind
- Precautions:
- recent internal bleeding (within last 3 months)
 - known bleeding diseases
 - patients currently taking anticoagulant agent(s)
 - recent surgery
 - possibility of pregnancy
- Dosage:
- 160 - 325 mg
 - Have the patient chew ASA before swallowing
- Pediatric
- none
- Special Notes:
- As an antithrombotic, ASA helps to limit the size of the infarction. It does not reduce the size of the infarction as thrombolytics do.
 - higher doses of aspirin (> 325 mg) may suppress the production of prostacyclin which is a prostaglandin with antiplatelet and vasodilatory properties. Therefore, higher doses of aspirin counteract the beneficial affect of the lower doses.

Atropine

Classification:

- anticholinergic
- antimuscurinic

Pharmacodynamics:

- parasympatholytic (inhibits stimulation from the parasympathetic nervous system)
- vagolytic (inhibits stimulation from the vagus nerve)
- inhibits vagal stimulation - allowing the sympathetic nervous system to dominate
- by allowing the sympathetic nervous system to dominate, impulse generation at the SA node and conduction through the AV node *should* increased

Pharmacokinetics:

ONSET

- 2-4 minutes

PEAK

- 2-4 minutes

HALF-LIFE

- 13-40 hours

DURATION

- 4-6 hours

Indications:

- restoration of cardiac rate in the presence of bradydysrhythmias
- sinus bradycardia, less than 50 bpm - accompanied by hemodynamic compromise
- sinus arrest
- acceptable in the setting of bradydysrhythmias secondary to AV blocks
- treatment of organophosphate exposure/ingestion (high dose)
- antidote for poisoning by certain species of mushrooms (e.g. *Amanita muscaria*)

Contraindications:

- hypersensitivity to anticholinergics
- tachycardia

Precautions:

- hepatic or renal insufficiency
- COPD - dries secretions/mucous plugging

Drug to Drug

- Antimuscurinic effects will be ↑ in patients taking Dysopyramide

Dosage:

- 0.5 mg IV push - initial dose
- Repeated q 3-5 min. To a max. of 3mg

Pediatric

- 0.02 mg/kg (give no less than 0.1 mg)

Special Notes:

- Atropine is no longer recommended for routine use in asystole or bradycardic PEA
- must be given in the correct dose and quickly - given in too low of a dose or too slowly may paradoxically slow the heart rate
- considered controversial in the setting of 2nd degree type II AV block. It may paradoxically slow the heart rate if the block is infranodal.
- not likely to be effective in ventricular escape rhythms as there is minimal parasympathetic innervation in the ventricles, however some
- atropine is unlikely to be effective in patients who have had cardiac transplantation as transplanted hearts lack vagal innervation
- Atropine also causes pupil dilation, therefore, assessment of pupils in the setting of asystole or PEA after Atropine has been administered may be unreliable

D₅₀W (Dextrose 50% in water)

- Classification:
- carbohydrate substrate
- Pharmacodynamics:
- immediate source of glucose and H₂O for nutrient deprived cells
 - transient osmotic diuretic
- Pharmacokinetics:
- ONSET
- immediate
- PEAK
- immediate
- HALF-LIFE
- unknown
- DURATION
- unknown
- Indications:
- suspected or known hypoglycemia
 - altered level of responsiveness NYD
 - coma or seizure NYD
- Contraindications:
- none
- Precautions:
- extravasation causes tissue necrosis
 - use with caution for alcoholics – consider pre-medicating with thiamin
 - consider consultation with BHP before administration if cerebral bleed is suspected
- Dosage:
- 25 g (50 ml of 50% sol.) prn
- Pediatric
- 0.2 g/kg of a 10% sol. for neonates (2 ml/kg)
 - 0.5 g/kg of a 25% sol. for 1 y/o or less (2 ml/kg)
 - 0.5 g/kg of a 50% sol. for ≥ 2 y/o (1 ml/kg)
 - Provincial medical directives may differ
- Special Notes:
- D₅₀W may precipitate Wernicke's encephalopathy in thiamin deficient patients (e.g. alcoholics)
 - pediatric: Dextrose is diluted in infants and neonates because the osmolarity of more concentrated solutions can cause intraventricular (cerebral) hemorrhage

Diazepam (Valium)

Classification:

- anticonvulsant
- sedative, anxiolytic, amnesic

Pharmacodynamics:

- binds to benzodiazepine receptor sites on CNS cells. This promotes the interaction between gamma-aminobutyric acid (GABA) and its receptor on neurons. When GABA interacts with its receptor site, the neuron becomes permeable to Chloride which is a negatively charged ion. An influx of chloride occurs making the inside of the cell more negative (hyperpolarized) and thus the cell takes longer to reach threshold and depolarize – suppresses the spread of seizure activity by raising the seizure threshold
- skeletal muscle relaxation (for muscle spasm)

Pharmacokinetics:

ONSET

- 1-5 minutes

PEAK

- 15 minutes

HALF-LIFE

- 20-50 hours

DURATION

- 15-60 minutes

Note: All benzodiazepines are metabolized by the liver.

Diazepam is converted by the cytochrome P450 enzymes in the liver to desmethyldiazepam (major) and oxazepam (minor). The fact that it has active metabolites makes it a longer lasting sedative than Midazolam.

Indications:

- treatment of prolonged seizures (greater than three-five minutes) or recurrent seizures
- sedation prior to electrical therapies (e.g. synchronized cardioversion, external cardiac pacing – use with caution in patients who are borderline unstable)

Contraindications:

- allergy or known hypersensitivity to benzodiazepines
- acute narrow-angle glaucoma (due to an anticholinergic effect)
- myasthenia Gravis
- hypoglycemic seizures - be sure to check BGL in the seizing patient

Precautions:

- may cause hypotension (Valium is mixed in propylene glycol, which is a vasodilator). Benzodiazepines also inhibit the neuronal re-uptake of Adenosine. The ↑ in circulating Adenosine outside the CNS might also explain why Diazepam has peripheral vasodilatory effects.
- may depress respirations (particularly in high dose: e.g. 10 mg in adults) -be prepared to assist ventilations

- impaired liver or kidney function
- patient who has ingested alcohol

drug to drug

- increased risk of toxicity in patients taking cimetidine, disulfiram, oral contraceptives. Decreased effects of diazepam when given to patients taking theophyllines, ranitidine

Dosage:

Status seizures

- 5 mg over 1 minute - repeat x 1 prn for status seizures - secure airway pr

Sedation

- 2-5 mg aliquots, given slow (over 1-2 minutes) for sedation - max. 30 mg

Pediatric

- 0.2 mg/kg IV, PR or IO (max. 20 mg) is the standard dosage for pediatric patients

Special Notes:

- also a common prescription drug
- OD on valium is unlikely to cause respiratory/cardiac arrest unless combined with alcohol or other drugs
- may precipitate when diluted with other solutions - **DO NOT dilute/mix with any other solution**

Dimenhydrinate (Gravol)

- Classification:
- antiemetic, antivertigo, anti-motion sickness agent
 - antihistamine, anticholinergic
- Pharmacodynamics:
- H₁ receptor antagonist
 - depresses hyperstimulated labyrinthine function
 - may block synapses in the vomiting center
- Pharmacokinetics: Intravenous
- ONSET
- Immediate
- PEAK
- 1-2 hours
- HALF-LIFE
- Unknown
- DURATION
- 3-6 hours
- Indications:
- prevention and treatment of motion sickness, nausea and vomiting, vertigo
 - N/V associated with AMI
- Contraindications:
- hypersensitivity or allergy to dimenhydrinate
 - neonates
- Cautions:
- lung disease, including asthma
 - glaucoma, acute angle closure
 - head injury
 - prostatic hypertrophy (enlarged prostate)
 - cardiac arrhythmias
 - pregnancy
 - stenosing peptic ulcer, pyloroduodenal obstruction
 - elderly, children
 - hypotension
- Adverse Effects:
- drowsiness, confusion, headache, dizziness, insomnia, hallucinations, blurred vision, diplopia, photosensitivity, urticaria
 - excitement and convulsions in children
 - epigastric distress, nausea, vomiting, diarrhea, constipation
 - dry mouth and nose
 - lassitude
 - hypotension, palpitations, tachycardia, thickening of bronchial secretions
- Drug to drug:*
- Increased CNS depression when given with other CNS depressants (e.g. morphine, diazepam)
 - increases anticholinergic effects of atropine, antidepressants, antihistamines, MAO Inhibitors and

phenothiazines, disopyramide. Increased CNS depression when administered to patients receiving analgesics (eg. Morphine) and/or sedative/hypnotics (eg. Diazepam, Midazolam) and/or alcohol

Dosage:

- 50 mg IM or IV
- 50 mg in 10-50 ml IV over 2-10 minutes (or very slowly)

Pediatric

- generally not recommended

Special Notes:

- should be administered slowly – i.e. it causes intense burning sensation at the IV site if administered too quickly. If it's being administered in a syringe (as opposed to an infusion), you can either administer it over 2 or more minutes in one bolus, or you can administer 1-2 cc increments followed by a 20-30 cc boluses until the full amount has been given.
- It's recommended that it be diluted to 10-100 cc (never dilute less than 10cc)

Diphenhydramine (Benadryl)

- Classification: • antihistamine
- Pharmacodynamics: • antihistamine with anticholinergic (drying) and sedative side effects. Antihistamines appear to compete with histamine for cell receptor sites on effector cells
- Pharmacokinetics: Intramuscular
- ONSET • rapid onset
PEAK • unknown
HALF-LIFE • 1-4 hours
DURATION • 4-6 hours
- Indications: • antihistaminic: adjunct treatment of allergic reactions / early anaphylaxis or as an adjunct to epinephrine in anaphylaxis.
• motion sickness
• antiparkinsonism
- Contraindications: • hypersensitivity to antihistamines
• neonates
• premature infants
- Precautions: has an atropine-like action and therefore, should be used with caution in patients with:
• a history of bronchial asthma
• increased intraocular pressure
• hyperthyroidism
• cardiovascular disease
• hypertension
• lower respiratory disease
- Adverse Effects: • Urticaria, drug rash, anaphylactic shock, photosensitivity, excessive perspiration, chills, dryness of mouth, nose, and throat
• Hypotension, headache, palpitations, tachycardia, extrasystoles
• Hemolytic anemia, thrombocytopenia, agranulocytosis
• Sedation, sleepiness, dizziness, disturbed coordination, fatigue, confusion, restlessness, excitation, nervousness, tremor, irritability, insomnia, euphoria, paresthesia, blurred vision, diplopia, vertigo, tinnitus, acute labyrinthitis, neuritis, convulsions
• Epigastric distress, anorexia, nausea, vomiting, diarrhea, constipation

- Urinary frequency, difficult urination, urinary retention, early menses
- Thickening of bronchial secretions, tightness of chest or throat and wheezing, nasal stuffiness
-

Drug to drug:

- additive effects with alcohol and other CNS depressants (hypnotics, sedatives, tranquilizers, etc).
- MAO inhibitors prolong and intensify the anticholinergic (drying) effects of antihistamines

Warnings

Use with caution in patients with:

- narrow-angle glaucoma
- stenosing peptic ulcer
- pyloroduodenal obstruction
- symptomatic prostatic hypertrophy
- bladder-neck obstruction
-

Dosage:

- 25-50mg IV/IM for moderate to severe anaphylaxis

Pediatric

- N/A

Special Notes:

- Diphenhydramine is an adjunct therapy for anaphylaxis and is generally given after the administration of epinephrine. If given first it may mask the signs of anaphylaxis

Dopamine

- Classification:
- sympathomimetic (dopaminergic agonist, beta agonist, alpha agonist)
- Pharmacodynamics:
- dose dependant
 - low dose - dopaminergic effect: renal, mesenteric and cerebral vasodilation - improves urine output (very unlikely to be ordered in this dose for prehospital care)
 - medium dose - β (beta) effect: \uparrow H.R., \uparrow force of cardiac contraction = i.e. +ve chronotropic and +ve inotropic effects
 - high dose - α (alpha) effect: vasoconstriction
- Pharmacokinetics:
- ONSET
- 2-5 minutes
- PEAK
- unknown
- HALF-LIFE
- 2 minutes
- DURATION
- less than 10 minutes
- Indications:
- symptomatic hypotension in the absence of hypovolemia e.g. cardiogenic shock, bradyarrhythmia, septic shock, renal failure, etc
 - post-arrest hypotension
- Contraindications:
- pheochromocytoma (rare tumor involving the adrenal gland, characterized by high levels of circulating catecholamines)
 - tachyarrhythmias
 - extreme caution must be used if patient on MAO inhibitor
- Precautions:
- may increase heart rate and induce supraventricular or ventricular tachycardia
 - may compromise cardiac output
 - **extravasation will result in tissue necrosis** (ensure IV is patent)
- drug to drug*
- increased sympathomimetic effects seen in patients on MAO inhibitors may lead to hypertensive crisis, coma or seizures - consult with BHP. Dopamine may have to be started at a lower dose for patients on MAOIs
 - The starting dosage of dopamine may need to be decreased by 10% or more for patients on MAOIs
- Dosage:
- 2 μ g-5 μ g/kg/min = dopaminergic effect
 - 5 μ g-10 μ g/kg/min = β effect
 - 10 μ g - 20 μ g/kg/min = α effect
 - **for the treatment of the hemodynamically unstable patient, the dose range is 5-20 μ g/kg/min**

- Dopamine is generally titrated (adjusted) in increments of 2-5 $\mu\text{g}/\text{kg}/\text{min}$ q 2-5 min to effect

Pediatric

- same as adult

Special Notes:

- When administering a dopamine infusion, an infusion pump should be used (ideal), or an in-line Buretrol (2nd best), or a minidrip (last choice). Fill Buretrol with 50 ml of the dopamine solution from the pre-mixed bag. Then close the Buretrol line off to the bag. The appropriate drip rate should then be controlled via the IV tubing
- 200 mg in a 250 cc bag will yield a concentration of 800 $\mu\text{g}/\text{ml}$ (single strength)
- 400 mg in a 250 cc bag will yield a concentration of 1600 $\mu\text{g}/\text{ml}$ (double strength)
- Dopamine is most often used in the prehospital setting for post-arrest hypotension.

Epinephrine

- Classification:
- sympathomimetic
- Pharmacodynamics:
- α_1 effects: vasoconstriction
 - β_1 effects: \uparrow H.R., \uparrow force of cardiac contraction
 - β_2 effects (moderate): bronchodilation
 - inhibits histamine release
 - +ve chronotropic, +ve dromotropic and +ve inotropic effects
- Pharmacokinetics: Intravenous
- ONSET
- immediate if given IV
- PEAK
- unknown
- HALF-LIFE
- unknown
- DURATION
- unknown
- Subcutaneous/ Intramuscular
- ONSET
- 5-15 minutes (variable onset with IM)
- PEAK
- unknown
- HALF-LIFE
- unknown
- DURATION
- 1-4 hours
- Inhalation
- ONSET
- 1-5 minutes (has a mostly local effect)
- PEAK
- unknown
- HALF-LIFE
- unknown
- DURATION
- 1-3 hours
- Indications:
- IV dose - cardiac arrest: ventricular fibrillation, pulseless ventricular tachycardia, asystole, pulseless electrical activity
 - SC or IM dose: anaphylaxis
 - SC or IM dose: severe cases of bronchospasm
 - Nebulized for severe Croup
- Contraindications:
- significant tachyarrhythmias
 - see ACLS guidelines re: drug therapy in the setting of hypothermia (< 30 degrees C)
- Precautions:
- may cause dysrhythmias in patients \geq 35 y/o and/or cardiovascular disease
 - reduced dosage may be required for patient on MAO inhibitor as there is an increased sympathomimetic response
- Adverse affects:
- tachycardia, palpitations, angina, PVCs
 - hypertension

Dosage:

- 1 mg IV (or 2 mg ETT) for cardiac arrest. Repeat q 3-5 minutes
- 2-10 µg/min infusion - generally reserved for patients who are *profoundly* bradycardic and hemodynamically unstable
- 0.01 mg/kg to max of 0.5 mg SC - repeat x1 in 5 - 10 min. prn for anaphylaxis

Special Notes:

- **DO NOT give an IV bolus of epinephrine to an adult with a pulse - it may be LETHAL**
- epinephrine may be ordered as a slow bolus in a pediatric patient with a bradycardia resistant to airway management and assisted ventilations
- epinephrine is neutralized by, and may precipitate with NaHCO₃. Therefore, DO NOT administer in the same IV line with Bicarb unless the line has been flushed
- in cardiac arrest, the most beneficial effect of epinephrine is that it increases systemic vascular resistance thus improving blood flow to vital organs with chest compressions.

Fentanyl (Sublimaze)

- Classification:
- synthetic opioid analgesic
 - synthetic narcotic
- Pharmacodynamics:
- inhibits ascending pain pathways in CNS
 - alters pain perception by binding to opiate receptors causing analgesia and euphoria – high doses may cause respiratory and physical depression
- Pharmacokinetics: Intravenous
- ONSET
- immediate - 2 minutes
- PEAK
- 3-5 minutes
- HALF-LIFE
- 3.6 hours
- DURATION
- 30-60 minutes
- Indications:
- relief of moderate to severe pain
 - effective in trauma patients – does not have the venodilatory effects of morphine and therefore is less likely to cause or exacerbate hypotension
 - may be used in conjunction with a sedative to facilitate awake intubation
 - adjunct in rapid sequence induction (RSI)
- Contraindications:
- hypersensitivity/allergy to opiates (including morphine)
 - myasthenia gravis
 - respiratory depression
 - acute asthma attack
 - upper airway obstruction
 - patient on MAO Inhibitors
- Precautions:
- be prepared to assist ventilations and to administer the narcotic antagonist naloxone (Narcan). This does not mean that you have to draw up Narcan or even have it pulled from the drug kit.
 - may alter mental status making it difficult to assess head injury
- Adverse Effects:
- lightheadedness, dizziness, sedation, agitation, fear, delirium, drowsiness, disorientation.
 - N/V
 - respiratory depression/apnea
 - laryngospasm
 - chest wall rigidity

- Dose
- 25-100 µg IV - repeat prn q 10-30 min.
- Pediatric:
- 1 - 2 µg /kg (diluted) slow IV (over 2-5 min.) or SC, IM
- Special Notes:
- Fentanyl is a controlled substance and **its use must be documented by the Paramedic** according to the "Controlled Substance" policy
 - Fentanyl can also be administered intranasal (IN) using an atomizer.

Furosemide (Lasix)

- Classification:
- loop diuretic
- Pharmacodynamics:
- venodilation may occur within 1-2 minutes - *first effect* (thought to occur through the release of “renal factor”).
 - inhibits the absorption of sodium and chloride in the ascending loop of Henle - as sodium is lost, so too is water - *diuretic effect may take 10 minutes or more*
- Pharmacokinetics:
- ONSET
- 5 minutes
- PEAK
- 20-30 minutes
- HALF-LIFE
- 30-60 minutes
- DURATION
- 2 hours
- Indications:
- pulmonary edema secondary to CHF presumed on the basis of clinical findings such as crackles, distended jugular veins, and dependant edema – evidence of hypervolemia
- Contraindications:
- allergy to furosemide, sulfonamides (cross-reaction), tartrazine (also know as Yellow Dye No. 5 which is a common food colouring in drugs and foods)
 - **hypotension**
 - pre-existing electrolyte depletion - in particular: hypokalemia
 - anuria, renal failure, hepatic coma, pregnancy, lactation
 - use cautiously in the presence of diabetes mellitus
- Precautions:
- patients taking sulfa (sulfonamide) drugs and/or thiazides as it may precipitate hypotension
 - MI, renal insufficiency
- Adverse Effects:
- hypotension, N/V
- Dosage:
- 20-40 mg slow IV
 - 80 mg may be given as a single dose in patients who are on Lasix PO
 - repeat q 15 min prn to max of 2 mg/kg
- Pediatric
- 0.5 - 1 mg/kg slow IV
 - repeat q 15 min prn to max of 2 mg/kg
- Special Notes:
- Furosemide is playing less and less of a role as a front line intravenous drug for acute pulmonary edema (APE) secondary to congestive heart failure. NTG is often the first drug of choice for APE
 - the patient in pulmonary edema will generally get some relief

of symptoms within a couple of minutes of administering Furosemide IV. This is due to the initial venodilatory effect that reduces preload and thus the workload on the heart

- diuretic effect generally begins in 10-15 minutes
- Loop diuretics deplete potassium. Watch for signs of hypokalemia

Drug to drug:

- Captopril (and all other ACE Inhibitors) reduces the diuretic effect of Furosemide

Glucagon

- Classification:
- glucose elevating agent (pancreatic hormone)
 - insulin antagonist
- Pharmacodynamics:
- accelerates the breakdown of glycogen (glycogenolysis) to glucose in the liver
 - glucagon: secreted by the alpha cells of the pancreas. It elevates blood glucose levels by increasing the breakdown of glycogen to glucose and inhibiting glycogen synthesis
 - Parenteral administration of glucagon produces relaxation of the smooth muscle of the stomach, duodenum, small bowel and colon
 - exerts a positive inotropic action on the heart by increasing intracellular cAMP concentration via the secondary messenger system (treatment of beta blocker or Ca⁺⁺ channel blocker OD).
 - only effective in treating hypoglycemia if liver glycogen is available
- Pharmacokinetics: SC/IM
- ONSET
- 8-10 minutes approximately
- PEAK
- 20-30 minutes
- HALF-LIFE
- 3-6 minutes
- DURATION
- 19-32 minutes
- Indications:
- hypoglycemia (confirmed or suspected)
 - hypoglycemia when IV access attempts have been unsuccessful
 - may be beneficial for airway obstruction secondary to foreign body in the esophagus compressing the posterior wall of the trachea – by relaxing the esophagus
 - beta blocker or Ca⁺⁺ channel blocker OD
 -
- Contraindications:
- allergy or hypersensitivity
 - pheochromocytoma (tumor involving the adrenal gland)
- Precautions:
- hepatic or renal insufficiency
 - insulinoma – may stimulate further release of insulin and precipitate/worsen hypoglycemia
 - pregnancy, lactation
- Adverse Effects:
- N/V, hypokalemia, urticaria, respiratory distress, hypotension
 - possible transient ↑ B/P and ↑ H.R.
- Dosage:
- 0.5 - 1 mg IM or SC (see medical directives for appropriate dose) - repeat x1 prn in 20 min.

Pediatric:

- same as adult. Max 1 mg

Special Notes:

- In patients with pheochromocytoma, glucagon may cause the tumor to release catecholamines which may lead to marked hypertension, tachycardia and intracerebral hemorrhage.
- sometimes used IV in high dose as a +ve inotropic agent in the treatment of β blocker OD - it increases heart and force of contractility through non-adrenergic pathways
- glucagon may be helpful in those patients on beta-blockers who develop anaphylaxis
- supplied in powder form - requires reconstitution
- should not be reconstituted with normal saline

Lidocaine

- Classification:
- Class IB antiarrhythmic, local anaesthetic
- Pharmacodynamics:
- use-dependant Na channel blocker (i.e. tends to work fairly specifically on more rapidly depolarizing ectopic foci)
 - ↓ the duration of the action potential by shortening repolarization
- Pharmacokinetics:
- ONSET
- 2 minutes
- PEAK
- unknown
- HALF-LIFE
- biphasic 8 minutes, 1-2 hours
- DURATION
- 20 minutes
- Indications:
- treatment of hemodynamically significant ventricular ectopy
 - closely coupled ventricular beats (R on T phenomenon), or multiform PVCs
 - bursts of 3 or more ventricular beats at a rate ≥ 100 (short runs of VT)
 - sustained VT with a pulse
 - pulseless ventricular tachycardia or ventricular fibrillation
- Contraindications:
- allergy or hypersensitivity to lidocaine
 - **3rd degree AV block**, ventricular escape rhythms, WPW - Note: although 2nd degree AV blk is also indicated as a contraindication in several texts, it is essentially a supraventricular rhythm. If it were to appear as a post arrest rhythm, the benefit of administering Lidocaine to prevent recurrence of VF or VT would outweigh the theoretical risks.
- Relative contraindication
- CHF, cardiogenic shock (consult with your Medical Director)
 - Lidocaine may be used in the setting of ventricular ectopy/VT secondary to cocaine ingestion, however there is an increased risk of seizure due to the synergistic toxic affects of these two agents.
- Precautions:
- hepatic or renal failure
- drug to drug*
- increased risk of Lidocaine toxicity when given to patients taking cimetidine, ranitidine or beta blockers - Cimetidine inhibits the metabolism of several drugs.
 - giving Lidocaine to patients on Disopyramide may cause bradycardia or cardiac arrest
- Adverse Effects:
- dizziness, lightheadedness, drowsiness, **slurred speech**
 - respiratory arrest – rare
 - hypotension, cardiac arrhythmias, cardiac arrest

- muscle twitching, **paraesthesia (tingling in the lips, fingers)**
- **“ringing in the ears”**
- N/V, rash, anaphylactoid reaction
- **seizures** secondary to lidocaine toxicity

Dosage:

- 1.0 - 1.5 mg/kg IV bolus or 2.0 mg/kg via ETT if IV not available in the arrested patient
- followed by 0.5 - 1.0 mg/kg bolus repeat prn to max of 3 mg/kg
- Infusion: 2-4 mg/min. (1 g in 250 cc at 30 cc/hour = 2 mg per minute)

Pediatric:

- same as adult

Special Notes:

- always treat the underlying cause of ventricular ectopy *first*. e.g. cardiac ischemia, electrolyte imbalance, hypoxemia, hypovolemia, etc

Midazolam Hydrochloride (Versed)

- Classification:
- sedative-hypnotic
 - CNS depressant
 - short acting benzodiazepine (chemical class)
- Pharmacodynamics:
- Short acting benzodiazepine. CNS depressant with sedative, muscle relaxant, anticonvulsant
 - Estimated to be 2-4 times more potent than diazepam. Intensifies activity of gamma-aminobutyric acid (GABA), a major inhibitory neurotransmitter of the brain. This causes chloride channels to open allowing an influx of chloride (-ve ion) into the cell. This makes the neuronal cells hyperpolarized - i.e. it takes longer for the cells to reach threshold and depolarize. This results in CNS depression
 - blocks memory , reduces anxiety, but enables patient to follow commands.
 - Strong amnesic effects
- Pharmacokinetics:
- ONSET
- PEAK
- HALF-LIFE
- DURATION
- IV: 1-5 min. IN: 5-10 min. IM: 5-15 minutes IM
 - IV route unknown (45 minutes IM)
 - 1.5 - 3 hours
 - 2-6 hours (dose related)
 - Note: All benzodiazepines are metabolized by the liver. Midazolam is converted by the cytochrome P450 enzymes in the liver to a non-active metabolite. This is why Midazolam is shorter acting than diazepam.
- Relevant Indications:
- sedation prior to intubation or electrical therapy
 - sedation of intubated patients
 - sedation of the hostile patient (provided a correctable underlying cause has been ruled out and the patient is stable)
- Contraindications:
- hypersensitivity to midazolam or other benzodiazepines
 - acute narrow angle glaucoma
 - shock
 - coma
 - alcohol intoxication
 - depressed vital signs
 - overdose
- Precautions:
- use cautiously when administration of other CNS depressants / narcotics are being administered (Narcotic +

Midazolam = Synergistic effect)

- use cautiously in the elderly and those with renal disease
- use cautiously in cases of CHF / COPD (respiratory depressant effect)

Adverse Effects:

- over sedation, headache, blurred vision, paradoxical combativeness (rare)
- hypotension, variations in pulse rate
- nausea, vomiting, hiccoughs.
- pain and tenderness at injection site
- respiratory depression / arrest, cough.

Drug to drug interactions:

- drugs that are known to inhibit the cytochrome P450 enzyme system may increase the potency and duration of midazolam (ie, some drugs in the drug classes ofazole antimycotics, protease inhibitors, calcium channel antagonists, and macrolide antibiotics)
- Lower doses are necessary for patients receiving concomitant narcotics or other CNS depressants
- erythromycin, diltiazem, verapamil, ketoconazole, fluconazole and itraconazole were shown to significantly increase the bioavailability of midazolam and may cause prolonged sedation.
- ritonavir and nelfinavir may cause intense and prolonged sedation and respiratory depression due to a decrease in plasma clearance of midazolam.
- rifampin, carbamazepine, and phenytoin may markedly decreased the effectiveness of midazolam

Dosage:

- 1.0 to 2.5 mg over 1-2 minutes
- may be repeated as required in small increments

Pediatric:

- 0.1 to 0.15 mg/kg
- Maximum of 0.5 mg/kg single dose

Special Notes:

- Unlike Diazepam, Midazolam is the first water-soluble benzodiazepine that may be administered in any IV fluid.
- 2 mg of Midazolam is approximately equivalent to 5 mg of diazepam

Morphine (MSO₄)

- Classification:
- opioid analgesic
 - narcotic
- Pharmacodynamics:
- it is an opioid alkaloid that acts on opioid receptors in the CNS to produce analgesia, euphoria and sedation
 - interacts predominantly with the opioid mu-receptor
 - interacts with receptors at the spinal cord level, depressing pain impulse transmission
 - causes venodilation - reduces cardiac preload
- Pharmacokinetics: Intravenous
- ONSET
- rapid
- PEAK
- 20 minutes
- HALF-LIFE
- 2-3 hours
- DURATION
- 4-5 hours
- Indications:
- relief of moderate to severe pain in the hemodynamically stable patient
 - chest pain of suspected cardiac origin
 - isolated extremity injuries, pain associated with burns, etc
- Contraindications:
- allergy or known hypersensitivity to narcotics
 - use with caution in asthma and COPD
- Adverse Effects:
- lightheadedness, dizziness, sedation, agitation, fear, delirium, hallucinations, drowsiness, disorientation.
 - respiratory depression/apnea
 - profound hypotension, reflex tachycardia, bradycardia, palpitations, chest wall rigidity
 - N/V
- Precautions:
- head injury, emphysema, kyphoscoliosis, cor pulmonale, severe obesity, elderly patient, labour
- Drug to drug interactions:
- depressant effects are potentiated by the presence of other CNS depressants such as alcohol, sedatives, antihistaminics, or psychotropic drugs.
 - Patients on neuroleptics: Morphine may increase the risk of respiratory depression, hypotension and profound sedation or coma
 -
- Dosage:
- 2 - 5 mg slow IV push (over 1 - 2 min.)
 - repeat q 10-30 min. prn - maintaining blood pressure at \geq 100 systolic or as per local BHP orders

Pediatric:

- 0.1 - 0.2 mg/kg (diluted) slow IV (over 5 min.) or SC, IM

Special Notes:

- be prepared to assist ventilations and to administer the **narcotic antagonist naloxone** (narcan) should you observe a decrease in LOC or ↓ in respiratory effort after administration of morphine. This does not mean that you have to draw up Narcan or even have it pulled from the drug kit in preparation.
- Morphine is a controlled substance and **its use must be documented by the Paramedic** according to the "Controlled Substance" policy

Naloxone (Narcan)

- Classification:
- narcotic antagonist
 - diagnostic agent
- Pharmacodynamics:
- reverses the effects of opioids including respiratory depression, sedation, hypotension
 - antagonizes the opioid effects by competing for the same receptor sites, especially the opioid mu receptor.
 - Also shown to all three opioid receptors (mu, kappa and gamma) with the strongest binding is to the mu receptor.
- Pharmacokinetics: intravenous
- ONSET
- 1 minute
- PEAK
- unknown
- HALF-LIFE
- 1 hour (up to 3 hours)
- DURATION
- 45 minutes
- Indications:
- to reverse respiratory depression/depressed mental status secondary to actual or suspected narcotic use - examples of other narcotics: demerol, heroin, codeine, oxymorphone (Numorphan), hydromorphone (Dilaudid), diphenoxylate (Lomotil), propoxyphene (Darvon), and pentazocine (Talwin)
- Contraindications:
- allergy or known hypersensitivity to naloxone
- Precautions:
- be prepared for patient combativeness
 - in the chronic narcotic abuser, may precipitate withdrawal symptoms
 - miscarriage or premature labour
 - very short half life; monitor patient closely and prepare to re-dose if deterioration occurs
- Adverse Effects:
- reversal of narcotic effect and combativeness
 - signs and symptoms of severe drug withdrawal
 - hypotension, hypertension
 - N/V, sweating, tachycardia
 - ventricular fibrillation, asystole
- Dosage:
- 0.4 - 2 mg slow IV push - repeat q 5 min. prn (usually effective by 10 mg)
 - if unable to establish IV, 4 mg may be given via ET (diluted in Normal Saline to a total volume of 10 ml)
- Pediatric:
- 0.01 mg/kg slow IV
- Special Notes:
- naloxone is generally considered a very safe drug - however,

potentially life-threatening problems (such as status seizures and asystole), occur in about 1% of patients treated. It is further hypothesized that these episodes may be related to an acute withdrawal syndrome associated with reversal of opioid-induced epinephrine blockade rather than to a direct intrinsic effect of naloxone

- Administration of naloxone to a comatose patient who has taken other medications/substances may result in a partial elevation of the GCS and/or combativeness.
- Naloxone administration may be titrated to improve spontaneous respiratory effort without complete reversal of opiate effects

Nitroglycerin (NTG)

- Classification:
- antianginal
 - nitrate
- Pharmacodynamics:
- relaxes vascular smooth muscle – predominantly a venodilator; also produces coronary and systemic arterial vasodilation to a lesser extent
 - NTG releases nitric oxide in vascular endothelial cells. Nitric oxide is a gas, which when released in vascular smooth muscle, results in the formation of cyclic guanosine monophosphate (Cyclic GMP). c-GMP relaxes vascular smooth muscle by inactivating myosin light-chain kinase or by stimulating dephosphorylation of myosin phosphate
 - ↓ preload, ↓ MVO₂ (myocardial oxygen consumption)
- Pharmacokinetics: Sublingual
- ONSET
- 1-3 minutes
- PEAK
- unknown
- HALF-LIFE
- 1-4 minutes
- DURATION
- 30 minutes
- Indications:
- chest discomfort of suspected cardiac ischemic origin
 - cardiogenic acute pulmonary edema (APE)
- Contraindications:
- allergy or known hypersensitivity to nitroglycerin or other nitrates
 - hypotension or uncorrected hypovolemia
 - severe anaemia
 - constrictive pericarditis and pericardial tamponade
- relative contraindication
- open or closed angle glaucoma
- Precautions:
- use with caution in the presence of hepatic or renal insufficiency
 - patients on concurrent anti-hypertensive therapy
- Adverse Effects:
- hypotension - do not administer if blood pressure is < 90 systolic or patient exhibits signs of significant hypoperfusion
 - Refer to local Medical directives for specific blood pressure and heart rate parameters
 - headache, N/V
- Drug to drug interaction
- Alcohol and nitroglycerin may have additive vasodilatory effects that may lead to hypotension

- Dosage:
- 0.3 - 0.6 mg SL - repeat q 3 min. - maintaining B/P \geq 90 systolic
 - infusion: 10 - 200 μ g/min. (in hospital)
- Pediatric:
- none
- Special Notes:
- for infusion: Nitro is generally mixed in a bottle. IV bags are made of polyvinylchloride (PVC) which absorbs the drug and \downarrow the concentration
 - in hospital, cardiac ischemic chest pain that does not respond to NTG SL and morphine IV is treated with a NTG infusion (typical mix: 50 mg in 250 cc - started at 10-20 μ g/min. and titrated in 10 μ g/min. increments to relieve pain)
 - *consider giving morphine sulfate if pain is unrelieved by NTG SL in prehospital setting*

Oxygen

- Classification:
- naturally occurring atmospheric gas
- Pharmacodynamics:
- \uparrow SpO₂ and \uparrow PaO₂
 - reverses hypoxemia
 - oxidizes glucose to produce ATP (energy source)
- Pharmacokinetics:
- immediate
 - < 1 minute
- ONSET
- unknown
- PEAK
- < 2 minutes
- HALF-LIFE
- DURATION
- Indications:
- prevent/treat hypoxemia
 - acute ischemic chest pain
 - limits the size of myocardial infarction
 - respiratory distress
 - \downarrow hemoglobin/oxygen carrying capacity
 - treatment/prophylaxis for \downarrow in atmospheric pressure (\downarrow PO₂) for air transport
 - depressed LOC (any cause)
- Contraindications:
- no absolute contraindication
 - use with caution in COPD – watch for signs of hypoventilation
- Relative contraindications:
- poisoning from the herbicide paraquat or diquat or patients receiving certain chemotherapeutic agents (e.g. bleomycin) may result in serious pulmonary complications (eg, oxygen toxicity and pulmonary fibrosis).
- Precautions:
- COPD with CO₂ retention - NEVER withhold O₂ from a patient in respiratory distress
 - neonates: prolonged exposure to high PO₂ may cause blindness - NEVER withhold O₂ from a patient in respiratory distress/failure
- Adverse Effects:
- none with short-term use
- Dosage:
- generally titrated to maintain SpO₂ \geq 95%
 - high FiO₂ for patients with signs and symptoms of respiratory distress, respiratory failure or decreased O₂ carrying capacity
 - consider lower FiO₂ in the newborn and the head injured patient

Pediatric: · same as adult

Special Notes: • secondary brain injury occurs largely as a result of oxygen-free radicals, hence the rationale for the current trend to limit FiO_2 in the head injured patient

 • rule out hyperventilation as a cause of respiratory distress

Concentrations:

<u>Method</u>	<u>Flow rate (L/min)</u>	<u>FiO_2</u>
nasal prongs	1-6	24-44
Nebulizing mask	6	40
Venturi mask	4-12	25-50
Simple mask	6-10	40-60
Non-rebreather mask	6-10	60-90

The FiO_2 is generally determined by the size of the reservoir: i.e. with nasal prongs the O_2 reservoir would be the nose and mouth and therefore the FiO_2 would be low. With a simple mask the reservoir would be the nose, mouth and mask and therefore the FiO_2 would be higher. With a partial-rebreather mask the reservoir would be the mouth, nose mask and reservoir bag and therefore the FiO_2 would be higher still.

Salbutamol (Ventolin)

- Classification:
- bronchodilator
 - sympathomimetic
- Pharmacodynamics:
- selective β_2 stimulation → bronchodilation and some degree of vasodilation
 - some β_1 effect – especially with repeated doses
 - little or no α stimulation
- Pharmacokinetics: Nebulized
- ONSET
- 5 minutes
- PEAK
- 1.5-2 hours
- HALF-LIFE
- 3.8 hours
- DURATION
- 3-8 hours
- Indications:
- bronchospasm associated with asthma, bronchitis or emphysema
 - bronchospasm & wheezing secondary to other causes
 - may be used in cardiac asthma - with caution
- Contraindications:
- hypersensitivity
 - hemodynamically significant tachyarrhythmias
- Precautions:
- coronary disease (\uparrow MVO₂)
 - COPD pts with degenerative heart disease
 - diabetes (↓ the effectiveness of insulin)
- Adverse Effects:
- restlessness, apprehension, fear, weakness, vertigo
 - N/V
 - tachycardia, dysrhythmias
 - paradoxical worsening of respiratory distress, pulmonary edema
- Toxicity:
- sweating, pallor, flushing
 - discontinue repeated doses result in signs of Salbutamol toxicity - i.e. if H.R > 150 (>200 in pediatric pt) , severe tremor, ventricular dysrhythmias develop
- Dosage:
- 800 μ g via MDI and aerochamber. Repeat prn
 - 5 mg nebulized (with O₂ at 6-8 L/min.) - repeat doses back to back prn - watch for signs of toxicity
 - Refer to provincial medical directives for specific dosing
- Pediatric:
- 600 μ g via MDI and aerochamber
 - 0.1 mg/kg nebulized in 2-5 cc NS

Special Notes:

- Refer to provincial medical directives for specific dosing
- also a common prescription drug – usual single dose by MDI is 100 µg inhaled
- β_2 selectivity is lost with high dose
- 10 mg of Salbutamol may lower serum potassium by as much as 1-1.5 mEq/L (normal K is 3.5-5.0 mEq/L) and is sometimes used in the setting of hyperkalemia (consider for hemodialysis patient with ECG signs of hyperkalemia e.g. bradycardia, AV block, wide QRS).

Sodium Bicarbonate (Bicarb or NaHCO₃)

- Classification:
- electrolyte
 - alkalinizing agent (buffer)
 - urinary alkalinizer
- Pharmacodynamics:
- buffers or neutralizes excess acid (buffers excess hydrogen ions)
 - raises blood pH
- Pharmacokinetics: Intravenous
- ONSET
- 2 minutes
- PEAK
- 30 minutes
- HALF-LIFE
- unknown
- DURATION
- 1 - 3 hours
- Indications:
- metabolic acidosis - confirmed by ABG or suspected based on clinical condition and history
 - known or suspected hyperkalemia
 - OD – e.g. tricyclic anti-depressant, salicylates
 - prolonged cardiac arrest without ABG - controversial
 -
- Contraindications:
- suspected metabolic alkalosis
 - Excessive vomiting
- Precautions:
- extravasation causes tissue necrosis
- Adverse Effects:
- metabolic alkalosis - hypoxia due to the left-upward shift of the oxyhemoglobin dissociation curve (O₂ not readily released at the tissue level)
 - tetany
 - seizures
 - **paradoxical worsening of metabolic acidosis - especially in the pt not being adequately ventilated - review carbonic acid buffer equation**
- Dosage:
- 1 mEq/kg slow IV push - repeat 0.5 mEq/kg q 10-15 min. prn
- Pediatric:
- child: 1-3 mEq/kg slow IV or IO to a max of 50 mEq - repeat 0.5 mEq kg q 10-15
 - min. prn
 - infant: 1-2 mEq/kg (4.2% solution) very slow IV/IO - repeat 0.5 mEq kg q 10-15 min. prn

Special Notes:

- also known as Bicarb and NaHCO_3
- metabolic alkalosis (which can be caused by Bicarb administration) may impair hemoglobin's ability to release oxygen at the tissue level.

Xylometazoline HCL (Otrivin)

Classification:	<ul style="list-style-type: none">• sympathomimetic• vasoconstrictor - long-acting stimulation of alpha(1)-adrenergic receptors• decongestant
Pharmacodynamics	<ul style="list-style-type: none">• stimulates alpha receptors in the nasopharyngeal mucosa to produce vasoconstriction and decongestion• vasoconstriction of the nasal vessels reduces the risk of epistaxis during <i>blind nasal intubation</i>
Pharmacokinetics	Intranasal
ONSET	<ul style="list-style-type: none">• 5-10 minutes
PEAK	<ul style="list-style-type: none">• unknown
HALF-LIFE	<ul style="list-style-type: none">• unknown
DURATION	<ul style="list-style-type: none">• 5 - 6 hours
Indications:	<ul style="list-style-type: none">• vasoconstriction of vessels in the nasal passages to reduce the risk of epistaxis during blind nasal intubation• over the counter decongestant for colds, rhinitis, sinusitis, otitis media, environmental allergies, etc
Contraindications:	<ul style="list-style-type: none">• hypersensitivity, narrow angle glaucoma, concurrent therapy with MAO inhibitors
Precautions:	<ul style="list-style-type: none">• diabetes mellitus - may worsen condition (regular dosing)• cardiac or peripheral vascular disease• high blood pressure - may increase B/P• hyperthyroidism• recent discontinuation of MAO inhibitors (anti-depressant) [must be at least 3 weeks]• concurrent use of beta adrenergic blockers• monitor patient carefully after administering Otrivin if any of these conditions exist
Adverse effects	<ul style="list-style-type: none">• headache, lightheadedness, nervousness, insomnia, blurred vision, cardiac arrhythmias including tachycardia, elevated blood pressure• adverse effects are generally not a problem when xylometazoline is used to pre-medicate patient for <i>blind nasal intubation</i> (only 2 sprays per nare)•
Dosage:	<ul style="list-style-type: none">• 1-2 0.1% spray Q 8-10 hours for congestion (not applicable to the paramedic)

Pediatric

- N/A

Special Notes:

- as soon as the need for blind nasal intubation is recognized, pre-medicate the patient with two sprays of Otrivin in each nare followed by two sprays of Lidocaine in each nare and two sprays of Lidocaine in the hypopharynx (if jaw is not clenched) - THEN prepare the necessary intubation equipment
- while preparing for intubation, provide bag-valve-mask assisted ventilation as needed
- according to the manufacturer, the spray bottle should not be used for more than one patient)