This document is a pharmacological review of medications carried by PRPS Advanced Care Paramedics. It includes applications as per the ALS PCS Medical Directives and other applications for medications as per the ACP scope of practice not included in a Medical Directive. Paramedics are required to PATCH to a BHP to receive a verbal order to treat patients with medications when a ‘PROVINCIAL PATCH POINT’ exists, or when there is no Medical Directive for a specific injury / illness where the paramedic feels the patient may benefit from pharmacological treatment.
Drug Name: Midazolam/Versed

Classification: Benzodiazepine, short acting

Drug Profile:

Midazolam is a CNS depressant which is water-soluble, available as an intranasal spray, intravenous, intramuscular injection and buccal administration.

Midazolam is a white to light yellow crystalline compound, insoluble in water. It is a potent sedative agent that requires slow administration and individualization of dosage. Clinical experience has shown midazolam to be 3 to 4 times as potent per mg as diazepam. Because serious and life-threatening cardiorespiratory adverse events have been reported, provisions for monitoring/detection and preventing these reactions must be made for each patient to whom midazolam has been administered, regardless of age or health status.

Actions /Pharmacokinetics:

Midazolam is a fast acting drug that easily penetrates the blood brain barrier and affects directly on GABA (gamma amino-butyric acid) receptors in the RAS and thus induces sedation and muscle relaxation.

IV administration of midazolam will affect the patient almost immediately (+/-30 sec) and will last for approximately 10 to 15 minutes. Due to midazolam producing an amnestic side effect, it’s widely used in the emergency setting for short and painful procedures such as cardioversion and external pacing. Although midazolam is used for painful procedures, it has almost no analgesic effect and pain control is to be taken under consideration for such treatments.

Midazolam’s half-life is between 2-6 hours depending on the administration route, metabolic status and liver function. Midazolam is metabolized in the liver and cleared by the kidneys.

Therapeutic Uses (Common):

Midazolam is used as a treatment of

- Seizure disorders
• Muscle spasms
• (As a hypnotic and short acting sedative) (Cardioversion)
• Alcohol withdrawal (DT's)
• Pre – post intubation sedation

**Overdose/Adverse Effects:** Serious and sometime life-threatening conditions can be seen with patients receiving midazolam. Such adverse effects are:

1. Over sedation
2. Headache
3. Blurred vision
4. **Paradoxical combativeness** (more common in pediatrics)
5. Hypotension
6. Bradycardia
7. Nausea and vomiting
8. Respiratory depression/ apnea
9. Tenderness at injection site

**General Precautions/Contraindications:**

Midazolam administration should be avoided in the following situations:

1. Hypersensitivity or allergy to the drug
2. Patient suffering from Acute narrow angled glaucoma
3. Shock
4. Coma
5. Alcohol intoxication
6. Depressed vital signs
7. Any TCA/MAOI/sedative overdose
Prehospital Applications as per the ALS PCS Medical Directives:

In the prehospital setting midazolam is utilized in the following Medical Directives.

- Procedural Sedation Medical Directive
- Combative Patient Medical Directive (after ruling out reversible causes)
  no difference in IV and IM dose
- Seizure Medical Directive
  a. 0.1 mg/kg IV
  b. 0.2 mg/kg IN, IM and Buccal routes

Please note that the most common midazolam variance identified through the BH audit system is an under-dose of Midazolam for seizures when administering via the IM/IN or buccal routes. Paramedics consistently administer the IV dose. Remember to double the dose if utilizing IN, IM or buccal route.

Prehospital uses requiring a BHP patch order:

Due to its potential effect on the patient’s hemodynamic status, midazolam should not be given to patients suffering hypotension or shock of any kind.

In addition, some Medical Directives have built in “mandatory patch” points which require paramedics to consult a BHP to obtain a verbal order in order to administer midazolam.

- Combative patient, when the paramedic is unable to assess the patient for reversible causes (hypoxia, hypotension and hypoglycemia).

Paramedics may encounter other medical emergencies where midazolam would be beneficial to use in the management of a clinical situation. In situations where a paramedic feels that midazolam administration is indicated to treat a patient and no medical directive exists, paramedic is required to consult a BHP and obtain an order to utilize midazolam. An example of a situation in which midazolam may be beneficial in the management of a patient where there is no Medical Directive for, would be a patient who is in
trismus and requires ventilation/airway control and nasal intubation is unsuccessful or contraindicated.

**Drug Name:** Diazepam

**Classification:** Benzodiazepine, Anxiolytic

**Drug Profile:**

Diazepam acts on GABA receptors that are located in the CNS. GABA is an inhibitory neurotransmitter that acts on presynaptic terminal nerve fibrils. This causes an increase in the influx of negative chloride ions. This increase in negative ions acts to cancel out much of the excitatory effect of the positively charged sodium ions that enter as a result of the arriving action potential. The action potential is therefore reduced, which in turn reduces the degree of excitation on the postsynaptic neuron as well. The overall effects are reduced neuronal excitability. This overall effect is beneficial for the treatment of seizures, muscle spasms, and anxiety related disorders.

**Therapeutic Uses: (Common)** Diazepam is used as a treatment of

- Anxiety related disorders (oral)
- Seizure disorders
- Muscle spasms
- (As a hypnotic and sedative) (Cardioversion)
- Alcohol withdrawal (DT's)

**Overdose/Adverse Effects:**

Pure benzodiazepine overdoses (oral) are not usually fatal. The more serious cases of toxicity are seen when benzodiazepines are mixed with other drugs or alcohol. The newer short acting benzodiazepine derivatives (Triazolam, Alprazolam, and Temazepam) have been related in fatal overdoses.
Rapid large doses (IV) can cause serious cardiorespiratory effects such as hypotension, bradycardia and cardiovascular collapse. Patients that have taken alcohol are at greater risk of adverse reactions. The muscle relaxant effects of diazepam can cause the patient to have prolonged periods of apnea. CNS side effects include drowsiness, dizziness, slurred speech, ataxia, and confusion. Diazepam can cause phlebitis and a burning sensation in the vein when given IV.

Notes on Administration/Special Pre Hospital Concerns:

The elderly are particularly sensitive to some of the above mentioned adverse side effects that are associated with diazepam. In the seizing patient diazepam administration should be stopped upon abatement of the seizure. When using Diazepam for sedation small incremental doses can be titrated to effect.

Respiratory depression due to the muscle relaxant effects of diazepam cannot be over stressed and constant monitoring of the patient’s airway and breathing status is crucial.

Diazepam is incompatible with most drugs and therefore should never be mixed or diluted with other drugs or solutions. It should be administered as close as possible to the IV catheter site as diazepam can precipitate or bind with the IV tubing.

If high doses of diazepam are given in order to arrest seizer activity, paramedics must be prepared to provided airway and ventilatory support if needed.

When administering pediatric doses, diazepam should be drawn up in a 1cc syringe in order to accurately administer the drug.

Rectal route can be used when an IV cannot be established; this is more effective in the pediatric population.

Pre hospital applications according to the medical directives:

In the past, diazepam has been widely used for short term sedation in quick or ongoing painful procedures.
In the most recent release of the ALS PCS Medical Directives, midazolam was introduced as the primary benzodiazepine and replaced diazepam in all relevant Medical Directives. As such, the latest MOH Equipment Standards does not list diazepam as a required medication for EMS services to carry and in most EMS services, diazepam is no longer available.

Although there is no specific Medical Directive for Diazepam, this drug still may be used for the same conditions as midazolam but requires a patch to BHP for an order to administer.

Some common conditions that diazepam may be considered for include:

Any Medical Directive that utilizes midazolam and where a patient cannot receive midazolam but where no contraindications exist for diazepam. Also, in the event midazolam is unavailable for any reason.

**Patch Required**

- Combative patient, when the paramedic is unable to assess the patient for reversible causes (hypoxia, hypotension and hypoglycemia).
- Procedural Sedation Medical Directive
- Combative Patient Medical Directive (after ruling out reversible causes) no difference in IV and IM dose
- Seizure Medical Directive

**Typical dosing for diazepam:**

- >5 y/o = 5 mg IV or (10 mg rectally)
- 1-5 y/o = 1.0 mg IV/IO per year of age or (2mg per year of age rectally)
- <1 y/o = 0.5 mg IV/IO or (1 mg rectally)

Other situations a paramedic may consider consulting a BHP for diazepam administration may include:

- Treatment of severe DT’s
- Sympathomimetic OD such as Cocaine
Special considerations / Patching requirements:

When patching for diazepam, the paramedic should carefully gather all relevant information with regard to patient’s condition and the specific reason why he would like to administer diazepam and not midazolam.

Due to its potential effect on the patient’s hemodynamic status, diazepam should not be given to patients suffering hypotension or shock of any kind.

Drug Name: Morphine Sulfate

Classification: Opioid, Narcotic analgesic

Drug Profile:

Derived from the poppy seeds of the opium plant and has been used for over 2000 years as a pain medication and cardiovascular altering drug. Its juice was known to contain an agent that relieved pain (=analgesic) and cause sleep or drowsiness (somniferum= sleep). The Greek word narcosis designates the sleep state hence the word narcotic. Most of the opiate analgesics and synthetic substitutes fall under the Narcotic Control Act in Canada, which is why medics and nurses alike must account for these drugs at the beginning and end of each shift. Additionally, only ACP’s are allowed to control and carry narcotics under this Act.

Morphine and related drugs (Meperidine and Fentanyl Citrate) exact a number of effects, both centrally and peripherally. Some are directly related to the analgesic effect while other effects are not. Each drug seems to have a specific affinity or degree of binding to each of the different receptors scattered throughout the body.

Opioids exert their action on by interacting with opioid receptors at the spinal cord level (pain modulation) which leads to a decrease in impulse transmission. Depending on the affinity of the drug for the receptor and the
location of the receptor, the drugs vary from one another in their effect and overall efficacy (effectiveness).

**Actions /Pharmacokinetics:**

**Neuronal:**

The result of the binding of opioids to their respective receptors on cell membranes is three-fold.

1. Hyperpolarization of nerve cells
2. Inhibition of nerve firing

**Analgesia:** Opioid narcotics relieve pain by raising the pain threshold within the spinal cord level and by altering the brains perception of the pain. Morphine is effective against all types of pain, visceral, somatic and cutaneous.

**Order of potency:** Meperidine < Morphine < Fentanyl

**Respiratory Depression:** Morphine reduces the sensitivity of the neurons in the respiratory center to carbon dioxide. This can occur with normal doses of morphine so it is imperative to monitor these patients closely and be prepared to intervene with airway maneuvers. This diminishing of sensitivity to CO₂ is important to remember when dealing with certain patient types such as COPDer's who are very sensitive to carbon dioxide levels. The most frequent unwanted side effect from morphine administration is respiratory depression.

Another side effect of morphine is that it causes cerebral CO₂ to rise, inadvertently causing cerebral vasodilatation and a subsequent rise in ICP. Therefore be cautious of morphine use in the acute closed head injured patient, especially without proper AW control/ventilatory support/monitoring.
Morphine's respiratory depressant effect is much more severe when other drugs are on board such as barbiturates, alcohol and other CNS depressants (synergistic affect)

**Euphoria/Sedation:**

Part of the analgesic effect is a foggy, dreamy, pleasant "unreal" feeling which is the reason that morphine and other narcotics are actively sought after street drugs. These effects are usually at low doses of morphine or fentanyl. Not all opiates produce euphoria in all subjects.

Morphine produces a sedative effect on the CNS in many patients but the degree of sedation varies based on many other physiological factors.

**Cardiovascular/Histamine Release:** Morphine causes a small amount of histamine release from mast cells in the body which may cause urticaria (hives), sweating and most importantly from a cardiovascular point of view, vasodilatation. This is due to peripheral arteriolar and venous dilatation. This leads to systemic vascular resistance decreases at this point, and decreased preload and the myocardial oxygen demand is also diminished. For this reason and the analgesic properties of morphine, it is the most frequently used narcotic for the treatment of ischemic chest pain.

Morphine should be used with extreme caution in the volume depleted patient due to its potential for producing hypotension. For this reason, fentanyl is the medication of choice in trauma patients due to the fact that it does not cause a clinically significant histamine release like morphine can (as well it is shorter acting).

Due to the potential for bronchoconstriction, it should be used with caution in asthmatics.

**Other CNS Effect: Occasionally** some patients who receive morphine or other opioid narcotics experience unexpected extreme excitation or restlessness after very low or high doses of the drug.
**Other Opioid Effects:** Miosis - is seen in most humans after morphine administration and is probably due to the removal of cortical inhibition on the third cranial nerve.

**Nausea and Vomiting:** A frequent side effect of morphine administration, and the reason that many practitioners give an anti-emetic with the opioid. This side effect is more frequent when administering morphine rapidly (IV) and in higher doses.

**Cough Suppression:** Unrelated to respiratory depression but a direct inhibition of the cough center. Codeine is often prescribed for a persistent cough in patients who cannot take other cough suppressants.

**Temperature Regulation:** Opioids inhibit the thermoregulatory center and the ability to maintain a body temperature is inhibited. This is seen most prominently in long term opioid use patients such as those with cancer related pain control.

**Therapeutic Uses: (Common)**

Morphine is widely used for many things in the pre-hospital and inter-hospital setting. During the World Wars it was the most frequently administered drug. It is given for:

- Ischemic chest pain
- Preload reduction for other purposes
- Acute pain management in trauma and long term treatment of chronic pain (cancer patients)
- Sedation in conjunction with benzodiazepines (Versed)

**(Antidote)**

Naloxone (Narcan) - narcotic antagonist
**Overdose/Adverse Effects:**

- Sedation
- Constipation
- Nausea and vomiting
- Urinary Retention
- Hypotension
- Potential for addiction (long term therapy)
- Flushing, Sweating
- Respiratory depression

**Prehospital Applications as per the ALS PCS Medical Directives:**

Please refer to the ALS PCS Medical Directives for accurate doses

- Cardiac Ischemia Medical Directive
  a. Consider morphine after the 3rd dose of NTG if the patient is still experiencing pain or if NTG is contraindicated

- Pain Medical Directive:
  a. Fentanyl is the preferred medication to be used for the Pain Medical Directive of the ALS PCS in EMS services who carry it in addition to morphine.
  b. Paramedics cannot switch from fentanyl to morphine or vice versa while treating a patient for pain without consulting a BHP.

Usually, there is no patching required prior to administration of morphine for the treatment of pain associated with cardiac ischemia or pain. Paramedics are required to patch anytime they feel the patient may benefit from pain control but does not meet the Medical Directive.
Drug Name: Naloxone (Narcan)

Classification: Narcotic antagonist

Drug Profile: Acts by binding to various opioid receptors in the CNS and peripheral NS and thus quickly reversing the effects of opioid narcotic such as morphine, heroin or fentanyl. Naloxone has a very high (up to x10) affinity to opioid receptors and it reverses the Opioid effect by competitively "bumping" out an opioid for the same receptor. This binding of naloxone does not activate the receptor and therefore, reverses the opioid narcotic effects.

Naloxone works very quickly, approximately 30 seconds after intravenous injection the respiratory depression and coma characteristics of a heroin overdose begin to reverse. Its half-life is about 60-100 minutes which may be shorter or longer than the half-life of the drugs it antagonizes. Therefore, close observation and monitoring of the patient is warranted and subsequent doses may be required.

The route of administration will also impact the duration of effect. IM and SC administration has a slower onset but longer effect than the IV route. This is why the new ALS PCS Medical Directives list the order of preference as SC then IM then IN then IV as routes for administration.

Naloxone also works on the naturally occurring pain mediators of the body, the enkephalins and will reverse them as well. Careful administration of naloxone is required to achieve the desired effect without causing complete reversal of analgesia.

Examples of some drugs reversed by naloxone

morphine, fentanyl, Percodan, heroin, codeine, Talwin, Darvon, hydromorphone (Dilaudid) Methadone
Therapeutic Uses: (Common)

Diagnostic or Therapeutic Use

Sometimes used as a diagnostic aid in patient presenting with signs and symptoms of a narcotic OD, but no history of.

Reversal of unwanted respiratory depression/sedation in a known narcotic overdose when patient cannot protect airway/ventilation.

Inadvertent narcotic overdose.

(As Antidote)

Is an antidote for narcotic overdose.

Overdose/Adverse Effects:

- Abrupt reversal of narcotic depression may result in – nausea/vomiting, sweating, tachycardia, increased blood pressure, tremulousness, seizures and cardiac arrest (rare).
- In patients receiving ongoing narcotic pain control, large doses of naloxone can cause significant reversal of analgesia

Prehospital Applications as per the ALS PCS Medical Directives:

In general, naloxone should be used only to improve patient’s respirations. Naloxone should not be used to completely reverse all opioid effects.

Opioid Toxicity Medical Directive:

Altered LOA and respiratory depression and suspected opioid overdose

Paramedics are required by the “Mandatory Provincial Patch Point” to contact a BHP for authorization to proceed with the medical directive.
**Things to remember:**

1. The Medical Directive is written in order of preference for the route of administration. SC, IM, IN then IV.

2. If the IV route is to be used, ensure the site is secured properly to avoid inadvertent removal.

3. Naloxone should be administered *slowly* and with caution. Especially in patients who are being treated for severe pain (cancer) or who are addicted to narcotics. Watch for signs of pain and or withdrawal such as tachycardia, hypertension and dysrhythmias.

4. Naloxone should be titrated to effect in small increments.

5. Always exercise caution with heroin or other illicit narcotic drug overdoses. Apply restraints prior to administering naloxone. Proper titration to reverse respiratory depression should avoid any inadvertent complete reversal causing aggressive behaviour.

**Special considerations/Patching requirements**

Naloxone is contraindicated in patients with uncontrolled hypoglycemia and sensitivity to the drug.

*A mandatory patch point is required prior to administration of Naloxone to all patients.*

**Drug Name:** **Fentanyl**

**Classification:** **Synthetic Narcotic**

**Drug Profile:**
Fentanyl, also known as Sublimaze, Durogesic, Fentora, Onsolis, Instanyl, Abstral, Lazanda and others, is a potent synthetic narcotic analgesic with a rapid onset and short duration of action. It is a strong agonist to the μ-opioid receptors.
Historically, it has been used to treat acute and severe pain and is commonly used in procedures as a pain reliever as well as an anesthetic in combination with benzodiazepines.

Fentanyl is approximately 100 times more potent than morphine, with 100 mcg of fentanyl approximately equivalent to 10 mg of morphine.

In the mid-1990s, fentanyl was first introduced for widespread palliative use with the clinical introduction of the Duragesic patch. In the following decade, introduction of the first quick-acting prescription formulations of fentanyl for personal use was introduced, the Actiq lollipop and Fentora buccal tablets. Through the delivery method of transdermal patches, as of 2012 fentanyl was the most widely used synthetic opioid in clinical practice. With several new delivery methods currently in development, including a sublingual spray for cancer patients, paramedics will continue to see more patients utilizing fentanyl at home.

Fentanyl and derivatives are now widely used as recreational drugs; as such, they have caused fatalities. Paramedics are more and more responding to patients who have overdosed on Fentanyl for non-medicinal usage.

**Actions/Pharmacokinetics:**

The precise mechanism of action of fentanyl is not known, although it relates to the stimulation of opiate receptors in presynaptic and postsynaptic stereospecific CNS and other tissues. Opioids mimic the action of endorphins by binding to opioid µ receptors resulting in inhibition of adenylate cyclase activity. This is manifested by hyperpolarization of the neuron resulting in suppression of spontaneous discharge and evoked responses related to modulation.

Fentanyl may also interfere with the transport of calcium ions and act in the presynaptic membrane interfering with the release of neurotransmitters.

The first effects of fentanyl are manifested in the CNS and organs containing smooth muscle. Fentanyl produces analgesia, euphoria, sedation, decreases the ability to concentrate, feeling of heat in the body, heaviness of the limbs, and dry mouth.

Fentanyl produces **dose-dependent ventilatory depression** primarily by a direct effect on the respiratory center in the CNS. This is characterized by a
decrease in the carbon dioxide response manifesting an increase in PaCO₂ and idle displacement of the response curve of CO₂ to the right. Fentanyl may also cause skeletal muscle rigidity, particularly in the thoracic and abdominal muscles, in large parenteral doses and administered quickly. Fentanyl can cause biliary tract spasm and increase the common bile duct pressure; this may be associated with epigastric distress or biliary colic.

Fentanyl can sometimes cause nausea and vomiting by direct stimulation of the CTZ (chemoreceptor trigger zone) in the floor of the fourth ventricle, and increased gastrointestinal secretions. However, it appears to have less emetic activity than morphine.

Fentanyl, unlike morphine, does not cause clinically significant histamine release even at high doses. Therefore, the secondary hypotension by vasodilation is unlikely. Fentanyl administered to infants can produce a marked depression of heart rate. The bradycardia is more pronounced with fentanyl compared with that of morphine and can lead to lower blood pressure and cardiac output.

Compared with morphine, fentanyl is approximately 100 times more potent, more rapid onset of action (less than 30 sec), and a shorter duration of action. Fentanyl has a higher lipid solubility compared with that of morphine and results in an easier passage through the blood brain barrier causing a higher power and a faster onset of action. Rapid redistribution by tissue produces a shorter duration of action.

Fentanyl is metabolized by dealkylation, hydroxylation, and amide hydrolysis to inactive metabolites that are excreted in the bile and urine. The elimination half-life of fentanyl is approximately 3.5 hours, reflecting the large volume of distribution.

**Therapeutic uses:**

Fentanyl is widely used in the prehospital and inter-hospital setting. It is one of the most frequently administered drugs.

Common uses for fentanyl are:

- Ischemic chest pain
- Severe musculoskeletal pain in trauma and long term treatment of chronic pain
- Sedation in conjunction with benzodiazepines
Antidote:  
Naloxone (Narcan) - narcotic antagonist

Overdose/Adverse Effects:

- Deep sedation
- Respiratory depression - apnea
- Muscle rigidity

Prehospital Applications as per the ALS PCS Medical Directives:

The Medical Directives order of preference for narcotics for pain is fentanyl. This is due to fentanyl having less of an impact on BP and shorter acting time than morphine.

Under all circumstances, the paramedic should pay careful attention to the patient's respiratory condition after administration of fentanyl. In the event of respiratory compromise secondary to narcotic administration, paramedics should patch for naloxone and provided appropriate AW management and respiratory support.

Drug Name: Sodium Bi-carbonate (NaHCO₃)

Classification: Alkalinizing agent, electrolyte solution, buffer solution

Drug Profile:

For many years sodium bi-carb (NaHCO₃) was used routinely in cardiac arrests as part of the drug regimen. Studies developed less than 10 years ago showed that routine use of NaHCO₃ might be actually detrimental to patient outcome as these patients would have alkalosis develop as a result of the
NaHCO₃ administration which was more difficult for the myocardium to deal with than the acidosis that results from inadequate ventricular output.

Remember the formula the body uses to balance pH:

\[
\text{H}_2\text{O} + \text{CO}_2 \leftrightarrow \text{H}_2\text{CO}_3 \leftrightarrow \text{HCO}_3^- + \text{H}^+
\]

*The enzyme used to catalyze this reaction is carbonic anhydrase*

This formula and these electrolytes in solution are what the body uses to deal with excess acids or excess base that are produced through metabolism, taken in through ingestion etc. or byproducts of toxins.

The body deals with acids primarily/initially through the buffer system, then through the respiratory system, the renal system and proteins. When a patient creates acids, the body must be able to excrete them or turn them into other products because the bodies pH is very sensitive to a narrow range 7.35-7.45. Metabolic acidosis results from either an accumulation of a fixed acid or loss of extracellular buffer.

There are many causes of lactic acidosis such as anoxia, respiratory failure, anemia, increased metabolic demand, alcohol, diabetes and more. In cardiac arrest it is usually due to acute cardiorespiratory failure. CO₂ is produced by aerobic metabolism in ischemic tissue during the first few minutes after cardiac arrest has occurred (remember – the cells are still alive). As such, CO₂ is not cleared locally from tissues and ventilation is obviously impaired.

NaHCO₃ acts by reversing the above equation and "tying" up excess hydrogen ions to decrease arterial blood H⁺ levels.

Under normal conditions the CO₂ produced by the tissues is transported to the lungs by NaHCO₃ (as part of the overall buffer mechanism) and is cleared via breathing out H₂O and CO₂. However, in cardiac arrest situations, this does not occur and CO₂ builds up locally and causes a paradoxical tissue and hypercarbic acidosis (not reflected in blood gas analysis). In the heart this can result in **decrease myocardial function.** The production of CO₂ by the administration of NaHCO₃ decreases the stimulation of the peripheral chemoreceptors (respond to H⁺) but does not affect central chemoreceptors.
Therefore, without circulation and ventilation, an increase in metabolic acidosis will occur.

Some studies have shown that accumulated CO₂ will get cleared via the lungs once cardiac output is restored. No longer is routine administration of NaHCO₃ recommended for patients in cardiac arrest, unless the arrest is prolonged or occurred due to severe metabolic acidosis and the patient is intubated.

**Therapeutic Uses:**

- Known metabolic acidosis
- TCA overdose
- Crush injuries:
  - Alkalinizing the urine (excretion of myoglobin precipitated in the kidneys secondary to Rhabdomyolysis)
  - Hyperkalemia – in the absence of ABG’s, the degree of hyperkalemia can be estimated by ECG changes (crude estimate)
    - Peaked T waves
    - Widening of QRS with decrease or loss of P wave amplitude
    - Life threatening ventricular arrhythmias; further widening of the QRS which eventually forms a sine wave
  - Hyperkalemia (along with Ventolin if ordered by the BHP to drive potassium into the cell)
  - Phenobarbital overdose (alkalizing diuresis to enhance urinary elimination of the drug). Alkalizing diuresis, if performed should be accompanied by IV fluid bolus.

Support respiration’s as patient will produce more CO₂ and blow it off. Make sure to give it slowly.

**Overdose/Adverse Effects:**

If NaHCO₃ is too rapidly injected, then the bicarbonate-blood mixture "fizzles" as it passes the lungs and changes the intra-alveolar pCO₂ and arterial pCO₂, which reaches the cerebral blood flow and causes transient cerebral vasodilatation. Patients may complain of dizziness or even syncope.
Notes on Administration/Special Pre Hospital Concerns

- Administer slowly
- Large vein or IO (pediatric)
- Hypernatremia can occur with administration
- Don’t mix with other drugs (especially Dopamine!), get precipitation

Dosing:

Adults: 1mEq/kg IV of 8.4% slow IV bolus

Pediatric: IV/IO 1mEq/kg of 8.4% slow IV bolus

Infant <30 days: (4.2%) 1mEq/kg slow IV bolus
References and acknowledgments


