

2013 BCAS Treatment Guidelines for EMR

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Treatment Guidelines

Welcome to the British Columbia Ambulance Service Treatment Guidelines. These guidelines are the resource documents that replace pre-hospital care protocols for Paramedic employees of the BCAS. They represent an innovative way of thinking about how Paramedics approach decisions to provide the best, most appropriate care for their patients. The Treatment Guidelines are not a linear step by step approach, but rather provide options within the appropriate scope of practice to allow Paramedics to address the patient’s specific needs. A pre-hospital diagnosis is provisional and based on limited information. Paramedics are authorized to deliver therapy in the field when it is best for the patient. Sometimes withholding a specific certain therapy may be in the best interest of the patient, particularly if definitive hospital care is readily accessible. The Treatment Guideline philosophy is based on the fundamental principle that patients will be transported to hospital and treated if necessary. It also is expected that pre-hospital care occurs in a framework of medical oversight. Potentially new approaches such as the development of community Paramedics will require other support and more focussed medical review.

Treatment Guidelines Overview

A Treatment Guideline consists of 2 core documents. Medical Principles and Intervention Guidelines

Medical Principles

The medical principles document provides key components of information that Paramedics should understand in consideration of their critical decision making process. These Guiding Principles form the basis of supporting Paramedic treatment decisions and provide guidance to either reaffirm or enhance paramedic root training in emergency care. Paramedics must know and understand these key principles and apply them each and every time they provide care to a patient. The principles are not necessarily comprehensive and they may change as we discover important concepts that require reinforcement to Paramedics in general.

Intervention Guidelines

The Intervention Guidelines describe the logical treatments that may benefit patients with these presentations. All are within the scope of practice for the given license level. The Intervention Guidelines are about options. They contain elements of treatment aimed at attaining a specific goal within the context of the patient's needs. Depending on the needs of the patient, Paramedics can choose to apply all of the elements, some of the elements or none of the elements identified leading up to and including their licensure and consequently within their scope of practice. The Intervention Guidelines are formatted to provide perspectives of care from the first responder level through to and including in-hospital case management. In this way Paramedics are informed about potential care with respect to all levels of medical education and their role in that continuum of care.

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Universal Approach to All Patients: Fundamentals

Indications:

The following is a combination of assessment and treatment steps that are performed routinely on all calls, regardless of presenting complaint. It is assumed that the following steps are performed in conjunction with any specific Intervention Guideline.

Safety Evaluation:

- Assess and assure scene safety
- Don appropriate PPE

Scene Evaluation:

- Patient numbers and locations
- Request additional resources as required
- Mechanism of Injury and Quick Look
- Crime scene preservation

Patient Primary Survey and Lifesaving Interventions:

- ABC
- Rapid Body Survey

Transport:

- Approach every call with a view to transport.
- Consideration for access/egress. Patients are not to be walked unless there are extenuating circumstances presenting a risk to the patient or the Paramedics.
- Carefully assess the need to delay transport only to provide interventions for the benefit of the patient.
- Vitals signs at least q 5 minutes for unstable patients; q 15 minutes for stable patients.

Secondary Survey and Interventions:

- Vital signs
- History
- Physical exam
- Routine Patient Care
- Specific medical therapy as directed by assessment
- Transport if not already begun
- Functional inquiry

Communications:

- On your arrival listen to any and all reports provided by other care givers/rescuers involved in patient care while ensuring lifesaving interventions are either maintained or initiated.
- Provide a clear concise report including chief complaint, history, assessment and interventions initiated to any health care provider who is likely to assume care for the patient. Ensure any diagnostic artifacts (e.g 12 or 4 lead ECG) accompany patient.

Documentation:

- Complete patient care record and any relevant research forms, in a timely fashion, without delaying or interfering with patient care.
- Ensure appropriate downloads have been completed.

Adult Guidelines

Altered Mental Status

Chapter 1

Introduction

Mental status involves awareness (or level of consciousness), cognition (or thinking) and attention. A typical altered patient may be sleepy, not making sense or unable to follow commands. Patients may also be excited, repetitively asking questions or belligerent.

Acute confusion or delirium is defined as acute cognitive dysfunction secondary to a medical condition. It is important to understand the difference between dementia, psychosis and delirium.

Dementia

Dementia is a cognitive decline from an organic cause that occurs in a steady, progressive fashion. Alzheimer's disease is one type of dementia. Dementia does not change hour to hour.

Psychosis

Psychosis involves hallucinations and or delusions but the patient is usually oriented and aware of his/her surroundings.

Delirium

Delirium is a confusional state that represents an acute change from the patient's baseline. The patient is disoriented and sometimes unaware of his/her surroundings. The patient's mental status can wax and wane over minutes or hours. Because it may be the result of a reversible medical problem, **delirium is a sign of an acute medical emergency that requires**

aggressive diagnostics and treatment. Acute delirium can co-exist in a patient with underlying dementia or psychosis.

Altered Mental Status

Chapter 1

Guiding Principles

Only a few causes of altered mental status can be treated in the field, however for all patients the fundamental principles of ensuring the patient's airway is protected and they are well oxygenated are paramount and may improve outcomes.

Patient history and assessment are vital in determining the presence of a treatable cause particularly with respect to symptomatic hypoglycaemia or poisoning and overdose. Paramedics should treat the patient in accordance with their treatment guidelines for the specific patient presentation.

All patients should have oxygen saturation and glucose levels checked.

Patients with altered mental status and no discernable treatable cause require expeditious transport to hospital for further diagnostics and treatment.

Causes

Hypoxia - airway obstruction, COPD/asthma, pneumonia, pulmonary oedema, pulmonary embolus (PE), pneumothorax, hemothorax, and pulmonary contusions, etc.

Acute Metabolic Disturbances - hypo/hyperglycaemia, electrolyte imbalances, renal failure and hepatic failure, hyperglycaemic hyperosmolar nonketotic syndrome (HHNS)

Environmental - hypothermia, hyperthermia

Acute Vascular - stroke, subarachnoid haemorrhage (SAH), hypertensive crisis, CNS vasculitis, intracranial sinus thromboses

Infection - sepsis, pneumonia, viral syndromes; CNS infections including meningitis, encephalitis, and intracerebral abscess

Alcohol (ETOH) - ETOH intoxication or withdrawal

Toxins - alcohols, over the counter medications, prescription or recreational drugs, insecticides or pesticides, animal venoms

Withdrawal - withdrawal is the body's reaction to the sudden cessation of a prolonged usage of a substance. Common are withdrawal from alcohol, opiates, barbiturates, and benzodiazepines

Endocrinological Disorder - endocrinological causes of confusion include thyroid storm, myxedema coma, Addison's crisis, hyper/hypoglycemia

Heavy Metals - mercury and lead

Post Seizure State - new or pre-existing seizure disorder

Agitated Patients

Chapter 1.1

Medical Principles

Definition

Acute cognitive dysfunction secondary to a medical condition.

Goal of Care

Recognition of altered mental status; look for reversible causes, and provide safe transportation.

It is common to encounter agitated, aggressive patients. Many of these patients are angry, frightened or reacting to stress. Others, however, are acutely delirious. It is of primary importance to recognize this and to look for reversible causes as well as provide safe transport.

The incidence of delirium at time of hospital admission has been reported to be as high as 20%. It is highly prevalent in the elderly population.

Cues

- restlessness
- extreme physical or mental excitement
- delirious patients rarely are able to negotiate and cooperate with caregivers
- irrational or inappropriate behaviour

Guiding Principles

A broad spectrum of patients may present in an acutely agitated state. Apart from hypoglycaemia few causes are reversible in the pre-hospital setting.

Patients who are extremely agitated with an alteration of cognitive function, including those with hypoglycaemia, are at risk of irreversible cardiopulmonary arrest.

Sedation may permit a safer transport and provide an earlier opportunity for hospital staff to evaluate the patient. In communities where available, Advanced Care Paramedic back-up should be considered to assist in the safe transport of these patients. Since there can be no treatment prior to restraint, patients should be physically restrained by police for paramedic and patient safety. The purpose of sedation is to mitigate the patient's continuous struggle against physical restraint. A first dose of Midazolam should be administered via I.M injection followed by initiation of transport. Further sedation may be given en-route however it is important to recognize that I.M Midazolam has an onset of approximately 5 to 15 minutes.

Bear in mind that sedation comes with the potential for respiratory compromise which may compound the underlying drug or disease effect. There is no evidence that sedation will prevent or cause sudden death in the extremely agitated patient.

Sudden death in excited delirium has been associated with patients restrained face down. If it is necessary to place the patient prone initially to gain control, monitor the airway and vital signs closely and always move to a supine or 3/4 prone position as soon as possible.

Causes

Life Threatening Conditions:

- hypoxia
- hypoglycaemia
- head injury
- drug actions or withdrawal
- infection (pneumonia, sepsis)
- electrolyte imbalances

Other common Causes:

- alcohol or recreational drug intoxication
- endocrine (e.g. DKA, hypo/hyperthyroidism)

Agitated Patients: EMR Interventions

Await police restraint if indicated

Position the patient –3/4 prone

Monitor vital signs closely

Supplemental [oxygen](#)

Correct suspected or confirmed hypoglycaemia

- Glucogel

Glucogel

Dose

1 package applied to oral mucosa

Contraindications

- none
- ensure airway is not compromised

Level of Evidence

- [Reference Level III](#)
- [Recommendation C](#)

Central Nervous System (CNS) Depressant Poisoning/Overdose

Chapter 1.2

Medical Principles

Definition

Depression of the CNS as a result of a drug.

Goal of Care

Primary airway management, adequate oxygenation, support of ventilation and transport.

CNS depressant poisoning and overdoses most frequently involve alcohol, opiates, or benzodiazepines. Other common CNS depressant overdoses include Gamma Hydroxybutyrate (GHB) and Tricyclic Antidepressants (TCA). Recreational drug overdoses are often due to combined agents.

Cues

Patients will have levels of consciousness ranging from mildly altered to unresponsive and may progress to cardiovascular collapse and death.

Guiding Principles

Always consider other potential causes of altered level of consciousness. Patients with medicinal or recreational drug overdoses may have trauma, CNS infection, or other serious illness in addition to the poisoning.

Where there is evidence of trauma or potential head injury, treat appropriately and transport.

Hypoglycemia is a treatable cause of altered mental status and should be corrected.

Examples of common overdoses:

TCA overdoses are time critical. Deterioration can be rapid and unexpected.

- tachycardia with widening QRS complex
- contact the Emergency Physician to consider treatment with sodium bicarbonate in any suspected TCA overdose with a significant tachycardia. The mechanism is unclear but may be related to effects on the sodium channels

Seizures should be treated promptly with Midazolam. Sodium Bicarbonate may also be indicated and should be discussed with the Emergency Physician.

Opioid overdoses require optimal ventilation and oxygenation prior to consideration of Naloxone.

- Naloxone is often not required and should only be given if the respiratory rate is depressed (<10/min) and there is altered LOC not responding to stimulation. Subcutaneous (SC) Naloxone is absorbed more slowly than IV thus providing a smoother emergence and is faster and safer to administer compared to starting an IV.

Beware of complications of Naloxone administration including:

- *combativeness, violence especially if other drugs are on board*
- *acute withdrawal sometimes accompanied by a seizure*
- *acute respiratory distress (rare)*
- *deterioration after 20-30 minutes as Naloxone effect diminishes and the opiate re-establishes it's effect*

GHB overdose can cause rapidly fluctuating states from combative to unresponsive with respiratory depression. Management consists of safe and expeditious transport.

CNS Depressant Overdose/Poisoning: EMR Interventions

Position the patient – on side if unconscious

Supplemental [oxygen](#)

Assist respirations with IPPV if necessary

Correct suspected or confirmed hypoglycaemia

- Glucogel

Glucogel

Dose

1 package applied to oral mucosa

Contraindications

- none
- ensure airway is not compromised

Level of Evidence

- [Reference Level III](#)
- [Recommendation C](#)

Hypo/Hyperglycaemia

Chapter 1.3

Medical Principles

Definition

Hypoglycaemia - low glucose level (less than 4 mmol).

Hyperglycaemia - elevated glucose level (higher than 11 mmol).

Goal of Care

Hypoglycaemia - restore normal glucose levels in the field.

Hyperglycaemia - restore normal glucose levels in hospital.

Many patients with hypo or hyperglycaemia have previously diagnosed diabetes but many diabetics go undiagnosed so history is not always reliable.

Hypoglycaemia

- sweating
- anxious
- dizziness
- impaired vision
- rapid heart rate
- weakness &/or fatigue
- LOC drops as the glucose falls

(Symptomatic hypoglycaemia does not occur unless glucose is less than 4 mmol.)

Hyperglycaemia

- blurred vision
- drowsiness
- nausea

(Symptoms are unusual if glucose level is less than 11 mmol but many non-insulin dependent diabetics tolerate higher levels without symptoms.)

Guiding Principles

In hypoglycaemic patients who can still comply with directions, administering oral glucose may be enough to increase their level of consciousness and avoid unnecessary IV initiation.

Following administration of glucose, glucagon and/or dextrose and following observed improvement in mental status, capillary blood glucose measurement should be repeated to gauge the level of response.

Although many hypoglycaemic diabetics decline transport following successful treatment, care must be taken to ensure a reasonable underlying cause of the event has been identified. *e.g. the event is clearly attributable to a late or missed meal in the face of a normal dose of insulin or the patient's physical activity has been higher than usual in the period prior to the incident.* These patients should be left in the care of another responsible adult.

Type II diabetics on oral hypoglycaemic agents who require treatment in the field should be transported to hospital. Oral hypoglycaemic agents have a very long duration of action and so the patient's hypoglycaemia is very likely to recur. These patients are commonly observed for 24 hrs in hospital.

Beware the otherwise healthy patient with a history of recent illness who is unconscious, hyperglycaemic and hypotensive. These patients may have new diabetes with first time DKA or undiagnosed diabetes and have developed hyperglycaemic non-ketotic coma. These patients are at risk of dying and need careful management in the Emergency Department.

Hypoglycaemia - history frequently reveals an imbalance of insulin or oral hypoglycaemics by:

- missing a meal
- insulin dosing not monitored over time
- over exertion without matching food intake
- a recent change of diabetic medication
- an overdose of insulin or oral hypoglycaemics, accidental or intentional

Hyperglycaemia - history and examination may reveal:

- recent infection or illness
- gradual onset of symptoms of dehydration lethargy, confusion
- excessive urine output
- insulin dependent diabetics in DKA may smell ketotic (fruity)

Non-insulin dependent diabetics can have high blood sugars, dehydration but no ketosis. Please see alert box above.

Hypoglycaemia or Hyperglycaemia: EMR Interventions

Position the patient – on side if unconscious

Supplemental [Oxygen](#)

Correct suspected or confirmed Hypoglycaemia

- Glucogel

Glucogel

Dose

1 package applied to oral mucosa

Contraindications

- none
- ensure airway is not compromised

Level of Evidence

- [Reference Level III](#)
- [Recommendation C](#)

Seizures

Chapter 1.4

Medical Principles

Definition

A seizure is the result of abnormal, spontaneous and uncontrolled electrical discharge from a group of neurons in the brain.

Goal of Care

Prevent aspiration and secondary injury, transport to hospital.

2% of all adults will have some type of seizure in their lifetime. Most commonly seizures occur in early childhood or late adulthood.

Seizure activity is not a disease but a symptom of an underlying CNS dysfunction. Epilepsy is a primary seizure disorder of recurring seizures, usually since childhood. Seizures may also occur in all serious illnesses or injuries that affect the brain. An ongoing seizure disorder (not epilepsy) may occur secondary to head trauma, brain surgery, or long term drug or alcohol abuse.

Cues

- loss of awareness
- twitching or shaking of the body, associated with uncontrolled muscular contractions

Guiding Principles

Seizures may manifest in many unusual ways. An eyewitness account is valuable to establish an accurate diagnosis.

Hypoglycaemia and hypoxia are easily identifiable and correctable in the field.

Beware that the sudden loss of blood pressure in cardiac arrest can result in seizure activity. A pulse check should be done immediately on arrival at a seizure call if the patient is not awake.

Seizing patients are at risk of hypoxia through poor ventilatory efficiency or aspiration and should be optimally treated in the $\frac{3}{4}$ prone position.

Beware of injuries. Sudden loss of consciousness can cause serious injury from falls and accidents. Intense rapid muscle contractions can cause injury to soft tissue and even dislocated joints or, rarely, broken bones.

Status epilepticus is defined as ongoing seizure activity greater than 5 minutes or recurrent seizures without full neurological recovery in the interim.

First time seizures require a work up in hospital including CT scan, blood work, and sometimes lumbar puncture. Even a patient who has recovered from the seizure needs assessment, and all efforts should be made to avoid the patient refusing transport.

The diagnosis of “febrile seizure” should not be made in the field. This diagnosis is based on a number of variables and the decision whether to do a medical workup on the patient lies with the Emergency Physician.

Ongoing or repeated seizures are treated with benzodiazepines either by ACP in the field or in the Emergency Department.

Causes

Typical causes of seizures:

- hypoxia
- hypoglycaemia
- withdrawal of alcohol or benzodiazepines
- acute head injury
- exposure to toxic drugs
- structural damage to the brain
- hyponatremia
- meningitis or encephalitis
- fluid accumulation in the brain
- epilepsy
- febrile seizures

Seizures: EMR Interventions

Position the patient - on side if unconscious

If still seizing protect the patient from potential injury

Supplemental [oxygen](#)

Correct suspected or confirmed Hypoglycaemia

- Glucogel

Glucogel

Dose

1 package applied to oral mucosa

Contraindications

None

Level of Evidence

- [Reference Level III](#)
- [Recommendation C](#)

Stroke

Chapter 1.5

Medical Principles

Definition

Sudden non-traumatic vascular insult to the brain. May be ischemic (embolic) or haemorrhagic (spontaneous bleeding).

Goal of Care

Identify reversible conditions, timely scene management and transport.

The most common cause of stroke is lack of blood flow to an area of the brain, usually from an embolic piece of plaque or clot blocking a cerebral artery (ischemic stroke). The other cause is spontaneous bleeding (hemorrhagic stroke). Transient Ischemic Attack (TIA) is the term used when a patient has acute stroke symptoms that resolve completely and spontaneously in minutes to hours

TIA is a dramatic warning (often referred to as a “warning stroke”) that the patient is at risk for stroke within days. It can be thought of as the neurological equivalent to unstable angina in the cardiovascular system.

Several conditions can present with stroke-like signs and symptoms. Patients who have a particular area of the brain at risk due to low blood flow can display stroke-like symptoms from a number of different causes. Anything

that decreases blood flow can create stroke-like symptoms e.g. symptomatic arrhythmia, hypotension from GI bleeding or trauma.

An important condition mimicking the symptoms of a stroke that Paramedics can identify and treat is hypoglycemia. Correcting hypoglycemia usually resolves the symptoms. Failure to correct hypoglycemia can lead to cell death causing a permanent stroke.

Patients with a seizure disorder can present with stroke like symptoms as part of the postictal phase. Attributing neurologic signs to primary seizure is often difficult since patients with a true stroke, especially a hemorrhagic stroke, can have a seizure as the first symptom.

Guiding Principles

EMS plays an important role in the care of patients with hyper-acute stroke. In the Emergency Department, thrombolysis with tPA is the treatment of choice for these patients but the window of opportunity to administer the drug is short and it can only be safely administered using very strict guidelines.

Accurate recognition, timely scene management, transport, and notification are key principles in caring for stroke patients. Thrombolytic drugs for hyper-acute stroke must be administered in less than 4.5hrs from the time of onset of symptoms. It takes about 1hr in the Emergency Department for evaluation, CT scan, and decision to give the drug. This leaves 3.5 hrs for recognition by the patient, access to 911, response time, scene time, and transport time. Use time efficiently.

Recognition begins with the Emergency Medical Call Taker identifying stroke symptoms from the caller's report. Paramedics can use a pre-hospital stroke tool to identify hyper-acute stroke. The most commonly used tool is the Cincinnati Stroke Tool.

It is vital to determine the time of onset of symptoms. If the patient or bystanders can't identify the onset, then the time of onset is judged to be the "last seen normal" time. If the patient woke up with the symptoms after having been asleep, then the time of onset could have been anytime during the night. In these cases, onset time is the time the patient went to bed. It is very important to determine if the patient woke up normal and the symptoms started after or if they had the symptoms upon awakening.

Timely transport is important because the thrombolytic window is very short. Time management at the scene is paramount. Only stop to do what cannot wait. Once the symptoms have been identified and that the onset was recent, check the patients capillary blood glucose. If it's normal, initiate transport. If it's low, correct it and initiate transport. Follow the preferred destination guideline for hyper-acute strokes, if there is one in your area. Code 3 transport would only be warranted if the time from onset is getting very close to exceeding the maximum allowable window for treatment with thrombolytics.

Notify the Emergency Department as soon as is practical that you are bringing in a hyper-acute or "hot stroke" if the time since last known to be normal is less than 3.5 hours.

Stroke: EMR Interventions

Position the patient

- Protect the airway if required
- If patient able to protect own airway - position of comfort

Supplemental oxygen

IPPV if required

Correct known or suspected hypoglycemia

- Glucogel

Assess stroke symptoms

- Cincinnati Stroke tool

Ascertain onset or "last seen normal" time

Minimize scene time.

Facilitate transport with notification

Syncope

Chapter 1.6

Medical Principles

Definition

Loss of consciousness resulting from a brief and reversible loss of normal neurological function due to poor perfusion, a metabolic event, or a neurological event.

Goal of Care

Standard ABC's, identify and treat reversible causes, transport.

Syncope is a common pre-hospital presentation. Like all presentations of altered level of consciousness, syncope is a symptom resulting from a broad number of causes. It is a brief and reversible loss of normal neurological function due to poor perfusion of the brain (decreased blood pressure), a metabolic event (hypoglycaemia, hyperventilation) or a neurological event (seizure, subarachnoid haemorrhage).

Syncope differs from other unconscious collapses in that it is most often of limited duration and patients return to a normal level of consciousness quickly.

Vasovagal syncope occurs as an overreaction of the autonomic nervous system (vagal nerve specifically), to triggers such as pain, the sight of blood or extreme emotional distress. It is often preceded by a complaint of feeling dizzy (lightheaded) or unwell. The patient appears pale and may be diaphoretic, feels weak and complains of blurred vision. The episode is easily attributable to a cause such as pain from an injury or emotional reaction.

Syncope can also be a sign of a potentially serious and life threatening condition. In the absence of an easily attributable cause for a vasovagal episode or if the patient is failing to recover, be suspicious of the presence of specific conditions such as unresolved hypotension, unresolved hypoglycaemia, a seizure or CVA.

Some patients experience syncope without warning. They are devoid of any pre-syncope signs or symptoms and experience a sudden collapse followed

immediately by a return to normal mental status. This type of syncope should be considered to be from a cardiac dysrhythmia until proven otherwise, even if the vital signs are normal when you arrive on the call. Maintain a high index of suspicion in any patient but particularly in those who collapse without any warning and then are almost immediately completely alert with a GCS of 15.

Guiding Principles

Do not be complacent with the notion of a simple fainting spell. Although you will see and transport many minor vasovagal syncope patients you will also transport patients with life threatening collapse and you can rarely be sure in the field if the cause is benign or life-threatening.

Initial vital signs and rhythm strips or 12 lead ECGs may be the best opportunity to diagnose the cause.

Patients who have experienced syncope are often inclined to refuse service and Paramedics must make their best effort to convince the patient to accompany them to the hospital.

Causes

Immediately Life threatening causes:

- cardiac dysrhythmias with or without associated ischemia
- other Heart disease (e.g. valvular, hypertrophic cardiomyopathy)
- hypovolemia (GI bleed, Occult haemorrhage (ectopic, AAA))
- hypotensive distributive shock (sepsis/ anaphylaxis)
- pulmonary embolus (obstructive shock)
- hypoglycaemia
- heat exhaustion/stroke
- subarachnoid haemorrhage
- cerebral Vascular Accident(TIA drop attacks)
- vasovagal syncope (pain, emotion, positional, pressure around the neck)
- seizure
- drugs: B-blockers/Ca channel blockers/benzodiazepines/narcotics
- hyperventilation/anxiety

Patients can have the symptoms associated with syncope without the loss of consciousness. This is what is referred to as “pre-syncope”. Often the cause

is more benign but the symptoms can be from any of the serious causes above and so patients are to be treated in the same fashion.

Syncope: EMR Interventions

Position the patient

- If symptoms suggest hypotension – lay flat IF this does not increase symptoms
- If no suggestion of hypotension – position of comfort

Supplemental [oxygen](#)

Cardiac Arrest Management

Chapter 2

Introduction

The 2010 CPR Guidelines emphasize the importance of providing high quality CPR. The quality and timing of CPR is critical to successful resuscitation in all patients that have experienced a sudden cardiac arrest (SCA).

The BCAS procedure and CPR/AED guidelines are meant to assist Paramedics in the important aspects of the interaction between CPR and defibrillation to maximize survival after the onset of cardiac arrest.

Importance of CPR

High quality CPR has been proven to dramatically increase survival from cardiac arrest. CPR maintains cerebral perfusion and restores some of the diminished blood flow to the heart muscle after SCA increasing the likelihood of a positive response to defibrillation.

Guiding Principles

There are a number of principles that are important for Paramedics to remember when delivering CPR.

When to start CPR?

CPR should be started when the rescuer finds the victim unresponsive and not breathing or not breathing normally. The 2010 AHA Guidelines have reorganized the traditional “A-B-C” to “C-A-B” in order to emphasize the importance of chest compressions. It is important to begin chest compressions as soon as possible. The pulse check is now de-emphasized as a mechanism to identify cardiac arrest. Because delays in chest compressions should be minimized, the rescuer should take no more than 10 seconds to check for a pulse.

When in doubt, start CPR

- If the patient is not in cardiac arrest they will begin rapidly to show signs of life and CPR can be stopped.
- It is always possible to stop CPR if more information becomes available regarding “no CPR” orders or other criteria indicating futility.
- Do not assume that the time of collapse is the time of loss of pulse. Many patients have some circulation for many minutes after collapse and prior to complete cardiac arrest. There must be clear conclusive evidence that more than 15 minutes has elapsed in cardiac and respiratory arrest without any basic life support, including dispatch assisted bystander CPR, to justify not starting based on elapsed time in cardiac arrest.

Do not be confused by agonal respirations that can sometimes continue for several minutes after complete cessation of circulation.

There are specific criteria that govern when it would be appropriate not to start CPR. In the non-traumatic cardiac arrest these criteria include:

- A “No CPR” order is in place, and/or
- Clear evidence that death has occurred such as initial stages of decomposition (lividity, rigor mortis, decomposition).

Time on Chest Makes a Difference

The 2010 AHA guidelines now recommend a Circulation – Airway – Breathing (CAB) approach underscoring the need to get on the chest early. It is also important to maintain continuous chest compressions as much as possible. The EHSC has adopted the continuous chest compression model of CPR versus the current AHA recommendation guideline (2010) of the 30:2 model;

all other aspects of CPR are identical to the AHA (2010) recommendations. The Treatment Guidelines are designed to enable the maximum amount of time on chest.

Switching Defibrillators

The most important principle in switching defibrillators is to be certain that the treatment is not interrupted. Data from all defibrillators should always be downloaded to the Cardiac Arrest Registry at BCAS headquarters, and there should be minimal interruption in the information if leads are switched rapidly and both defibrillators are turned on. There is no advantage to switching urgently from a First Responder defibrillator, so an appropriate time would be during one of the two minute periods of CPR. Transfer to a manual defibrillator managed by ACPs is advantageous because manual analysis of the rhythm is often faster and can help reduce pauses in CPR during analysis.

Pediatric Arrest

Most cardiac arrests in children are not due to sudden rhythm disturbances and the focus should be on high quality CPR, oxygenation and rapid transport. Once oxygenation and high quality CPR have been established all infants and children in cardiac arrest should have a defibrillator attached to determine if a shockable rhythm is present. Paramedics need to consider the cause early and if there is a history of blunt trauma to the chest, electrocution, or the patient has a cardiac history, oxygen and CPR are still the priority but Paramedics should apply the AED with greater urgency as these patients may be more likely to demonstrate a shockable rhythm. If ventricular fibrillation is demonstrated, defibrillation should be attempted as soon as possible. More details on the care for pediatric patients in cardiac arrest are found in the Pediatric Cardiac Arrest Treatment Guideline.

When to Stop?

Clarification of the 15 minute and 30 minute phone calls to the Emergency Physician

Paramedics should contact the Emergency Physician (EP) following the delivery of 15 minutes of high quality CPR to determine if transport of the patient is warranted at this time. Transport does compromise high quality CPR but may be indicated for potentially reversible causes of cardiac arrest.

Many EP's are not familiar with BCAS treatment guidelines and may assume the purpose of the call is to seek discontinuation orders. **This is not the case.**

BCAS paramedics should guide the discussion appropriately. The 15 minute call determines whether to stay on scene and resuscitate or initiate transport. This may also be the time to discuss with the EP the plan for treatment at 30 minutes. The discussion may include the following options:

After 30 minutes of CPR with no return of spontaneous circulation/signs of life:

1. Confirm with the EP his/her comfort level that, after a further 15 minutes of CPR, provided you have not had any intermittent ROSC or a shockable rhythm, termination of resuscitation is appropriate.
2. Call the EP back with an update on the course of the arrest and seek an order to discontinue.

It is reasonable to consider terminating CPR after 30 minutes of high quality CPR if you have no return of circulation at any time and the AED shows no shock advised **providing this has been discussed as a possible outcome with the EP at the fifteen minute mark**. If communication issues prohibit this from happening due to lack of cellular coverage or inability to perform a radio patch then it is the responsibility of the paramedic to notify the receiving EP as soon as feasible.

There are times when it is best to transport early (before the 15 minutes) with CPR en route. Examples include: massive bleeding (Gastro-intestinal bleeding/ruptured ectopic pregnancy), likely pulmonary embolus, severe anaphylaxis.

If an order is received to discontinue CPR confirm the patient has absent vital signs by taking the following steps.

- ensure the absence of a carotid pulse
- ensure the absence of any respiratory effort
- Auscultate the left anterior chest to confirm the absence of any heart sounds

Additional information on resuscitation orders can be found in the pages that follow.

PCPs are currently not trained to interpret ECGs and so are **not** to alter any procedure based on the rhythm displayed on the LifePak 1000 screen, nor should they offer or agree to describe the wave forms when consulting an Emergency Physician (EP).

5 Principles of Quality CPR

Quality CPR includes 5 principles

1. Optimum compression rate
2. Optimum depth
3. Full decompression
4. Optimum ventilation rate
5. Infrequent compression pauses

Compression Rate

The rate of compression should be 100/minute.

Compression Depth

Depth of compressions should be at least 2 inches/5cm in a normal adult.

Full Recoil

Full recoil of chest is very important. Between each compression a negative pressure develops in the chest which pulls blood into the thorax ready for the next compression.

If we do not take the pressure off the chest between each compression, positive pressure remains in the thorax throughout the CPR cycle and so no blood returns to the thorax between compressions. If no blood returns there is no blood to move forward to the heart with the next compression.

Ventilation Rate

Ventilation rates must be kept down to 8-10 per minute. It is very easy to ventilate too quickly; all health care workers have been shown to do this.

Hyperventilation maintains positive pressure in the chest and reduces CPR blood flow. It is important to pay close attention to your ventilation rate. It is also extremely important (and adequate) to ventilate with volumes of about 500-600 cc (about 1/2 of the volume of the bag from an adult bag/mask set up) during CPR. To get a good idea of these volumes, look at the BVM you're using and see how small a portion of the bag you will be delivering.

Compression Pauses

After the initial absence of a pulse is established, and chest compressions started, subsequent pulse checks are done only during the analyze time or if signs of return of spontaneous circulation (ROSC) are observed (coughing, movement, normal breathing).

A rhythm change to one of organized electrical activity on the Lifepack 12 or 15 is not an indicator for Advanced Care Paramedics to pause chest compressions and assess for a pulse. Changes in etCO₂ or signs of life are better indicators of return of spontaneous circulation.

The requirement to push hard and fast leads to fatigue quickly and compressions become less effective after a few minutes of CPR. Compressors should be relieved or changed every two minutes preferably during an analyze phase when possible.

Evaluation

BCAS is working hard to provide individual feedback for cardiac arrest calls. Feedback includes information on hands-on time, compression rate and (in the future) ventilation rate. The purpose of this feedback is to allow Paramedics to reflect on and improve resuscitation practices if necessary. The ability to provide accurate feedback depends on the written and download information provided by Paramedics. This includes information from every crew, including First Responders that participated in the resuscitation.

Having complete download information on every cardiac arrest is also vital to our system. Complete data allows us to assess our delivery of CPR and cardiac arrest care as a system so we can discover gaps rapidly, change guidelines where necessary, and discover what issues would benefit from further education.

Skills and Procedures Guideline: CPR Adult Cardiac Arrest – Prior to EMS Arrival

The roles and responsibilities identified in this guideline are not written in stone but rather serve to demonstrate how teams can work in unison to quickly and efficiently achieve the goals of effective basic life support treatment. Note: the EHSC recommend the continuous chest compression model of CPR versus the current AHA recommendation guideline (2010) of the 30:2 model; all other aspects of CPR are identical to the AHA (2010) recommendations.

Paramedic 1 - 10 second check - if no response, no pulse and absent or abnormal breathing, state “Start Compressions”

Paramedic 2 - Turn on AED or Monitor / Defib
Expose patient's chest
Initiate chest compressions

Paramedic 1 - Apply pads ASAP

Paramedic 2 - CC rate of 100/min until pads applied

Paramedic 1 - Prepare BVM

Paramedic 2 - Push analyze or interpret rhythm
Check pulse

Paramedic 1 - Ventilate with low volume and low pressure once every 10 chest compressions. Check for airway patency/foreign body without stopping chest compressions

Paramedic 2 - Immediate CC while charging

Paramedic 1 - State “Clear”

Paramedic 2 - Push to shock

Paramedic 1 - Time for 2 minutes of CPR
Apply BVM
Ventilate once every 10 compressions

Paramedic 2 - Immediate CC for 2 minutes
(200 CC or 20 cycles of 10)

Paramedic 1 - Announce “Time to analyze” after 2 minutes

Paramedic 1 - After passing off BVM check pulse during analysis
If shock is indicated compress while charging, clear, then defibrillate

Paramedic 2 - Push Analyze
Move to maintain BVM and take over ventilations

Paramedic 1 - Do next 2 minutes of CC

Paramedic 2 - Time for 2 minutes of CPR
Ventilate once every 10 CC (no more than 10/min)

Paramedic 1 & 2 - Repeat last section until return of spontaneous circulation, arrival of ALS, arrival at hospital, or discontinue order after a minimum of 30 minutes of CPR.

Skills and Procedures Guideline: CPR Adult Cardiac Arrest – EMS in Attendance

The roles and responsibilities identified in this guideline are not written in stone but rather serve to demonstrate how teams can work in unison to quickly and efficiently achieve the goals of effective basic life support treatment. Note: the EHSC recommend the continuous chest compression model of CPR versus the current AHA recommendation guideline (2010) of the 30:2 model; all other aspects of CPR are identical to the AHA (2010) recommendations.

Paramedic 1 - 10 second check – if no response, no pulse and absent or abnormal breathing, perform precordial thump and begin chest compressions after pads applied, push analyze or, if ALS, interpret rhythm

Paramedic 2 - Turn on AED or Monitor/Defib
Expose patient's chest and apply pads ASAP

Paramedic 1 - Prepare BVM

Paramedic 2 - Immediate Chest Compressions (CC) while charging, rate 100/min
Announce "Time to analyze" after 2 minutes

Paramedic 1 - "Clear"

Paramedic 2 - Push to Shock

Paramedic 1 or 2 - Assess for immediate return of signs of circulation or responsiveness

Paramedic 1 or 2 - If no response

Paramedic 1 - Time for 2 minutes of CPR
Apply BVM
Ventilate once every 10 compressions,
Announce "Time to analyze" after 2 minutes

Paramedic 2 - Immediate CC for 2 minutes
(200 CC of 20 cycles of 10)

Paramedic 1 - After passing off BVM check pulse during analysis. If shock is indicated, compress while charging, clear, then defibrillate

Paramedic 2 - Push to Analyze

Move to maintain BVM and take over ventilations

Paramedic 1 - Do next 2 minutes of CC

Paramedic 2 - Time for 2 minutes of CPR

Ventilate once every 10 CC (no more than 10/min.)

Paramedic 1 & 2 - Repeat last section until return of spontaneous circulation, arrival of ALS, arrival at hospital or discontinue order after a minimum of 30 minutes CPR.

Causes

Treatable Causes

VF is the most treatable of all cardiac arrest causes. CPR and defibrillation are the treatments of choice and by far the most successful. The following table outlines ALS level assessment, rhythm interpretation, and intervention for VF and other treatable causes of cardiac arrest.

Reference guide of potentially treatable causes for patients in Cardiac Arrest.

PEA Asystole VF

Treatable Causes

- cardiac tamponade
- hypothermia
- pulmonary embolus
- sepsis

Suggested Field Treatments

A fluid bolus may be beneficial in any of these cases; however, field treatment of these conditions is limited. Rapid transport is indicated. Contact the Emergency Physician as early as possible.

PEA Asystole

Treatable Cause

- **Pacemaker failure** presenting as asystole is not a common event. For asystole to present as a manifestation of pacemaker failure, the entire power unit of the pacemaker would have to fail abruptly in a patient with no native underlying rhythm. Pacemaker leads may fracture, resulting in pacemaker spikes without capture.

Suggested Field Treatment

- treat as profound bradycardia

Treatable Cause

- tension pneumothorax

Suggested Field Treatment

Needle thoracentesis is immediately indicated for cardiac arrest patients suspected of having a pneumothorax.

Treatable Cause

- hypovolemia

Suggested Field Treatment

- treat with IV fluid bolus and rapid transport

Treatable Cause

- hypoxia

Suggested Field Treatment

- ensure 100% oxygen and standard CPR ventilation

Treatable Cause

- acidosis

Suggested Field Treatment

- ensure adequate oxygenation and optimum perfusion (CPR)
- give Sodium Bicarbonate if acidosis is the cause of the arrest

Treatable Cause

- anaphylaxis

Suggested Field Treatment

- IV Epinephrine, fluid bolus

Treatable Cause

- hyperkalemia (renal failure patients)

Suggested Field Treatment

- Sodium Bicarbonate
- Calcium Chloride

Treatable Causes

- drug overdose
- tricyclic antidepressant
- cocaine
- amphetamines
- Class 1a antiarrhythmics such as procainamide hydrochloride, quinidine sulfate, and disopyramide phosphate
- calcium channel blocker, beta blocker

Suggested Field Treatment

- Naloxone hydrochloride for opioids
- Sodium Bicarbonate inhibits tricyclic antidepressant protein binding

- Magnesium in Class 1a and sotalol hydrochloride overdose
- Calcium Chloride for calcium channel and beta blocker overdose

PEA

Treatable Cause

- profound bradycardia

Suggested Field Treatment

- Epinephrine bolus

Treatable Cause

- extremely rapid tachycardia

Suggested Field Treatment

- cardioversion

Torsade de Points

Treatable Cause

- congenital prolonged QT
- antiarrhythmic drug therapies with Class 1a agents or sotalol hydrochloride
- severe starvation states
- nutritional deficiency

Suggested Field Treatments

Treatment consists of Magnesium 4.0 grams as a bolus. Efficacy of routine administration of Magnesium in cardiac arrests has not been proven.

Chest Pain Suggestive of Acute Coronary Syndrome (ACS)

Chapter 3.1

Medical Principles

Definition

Insufficient supply of blood to the heart, usually due to a blocked artery, causing cell and tissue death.

Goal of Care

Transport to hospital where the artery can be opened or kept open.

Myocardial Infarction (MI) is usually caused by a clot in the coronary arteries that completely blocks blood flow beyond its location. It is triggered by a rupture of plaque in the walls of a coronary artery and is defined as death of myocardial cells due to ischemia. MI can show up in two ways. Acute, significant damage shows up on ECG with elevation of the ST segments, called an “ST-Elevation MI” or “STEMI”. When there is only small amounts of damage, the ECG can be normal but cardiac enzymes, proteins that are necessary for proper heart function and released into the blood stream when heart damage is occurring, will rise over 3-6 hrs.

Unstable Angina is temporary insufficient blood supply without permanent damage. The pain of MI and UA are similar and both require urgent care. The goal of care in myocardial infarction is to open the artery and the goal in unstable angina is to keep it from closing.

Chest pain is the most common symptom but only about 30% of patients with ACS have the classic chest heaviness with radiation to arms or jaw, shortness of breath and sweating. In fact, atypical pain (just about anything else) is more common than typical pain! Diabetics and women have “atypical” pain complaints – or minimal complaints – even more commonly.

The diagnosis requires an ECG, cardiac serum markers that signify damaged and leaking cells, and sometimes imaging (including angiograms). In some cases a firm diagnosis even with these tests is difficult.

Guiding Principles

Patients with myocardial infarction on the electrocardiogram are losing muscle with each minute of delay to definitive treatment.

Chest pain can also be the presentation of other life threatening conditions such as thoracic aortic dissection, pulmonary embolus, pericarditis, ruptured esophagus or other perforated viscus.

The treatment of choice for STEMI is angioplasty or thrombolysis. Follow the policy and guidelines for your region regarding destination decision rules for STEMI.

Patients with chest pain but not STEMI do not require emergency angioplasty. They can be taken to the closest facility for diagnosis, decision making, and initial treatment. A secondary transfer can be arranged, usually routine for both the transfer and the cath lab, if angioplasty is ultimately indicated.

Beware patients who are hypo-perfusing or are developing congestive heart failure or both. These patients are developing cardiogenic shock and require immediate urgent intervention

A 12 lead ECG should be acquired early to rule out or rule in ST-Segment Myocardial Infarction (STEMI). Early notification to activate the hospital response to receiving a STEMI has saved lives.

If IV access is required, the left arm is preferred. Keep the middle and distal third of right forearm clear to facilitate potential radial artery access for percutaneous coronary artery intervention

Oxygen should be titrated based on pulse oximetry aiming for an O₂ Saturation of 95% if the patient is not SOB or in shock. Patients who are SOB or in shock require high flow O₂. If there is difficulty with the probe acquisition due to patient condition, oxygen should be applied liberally. Patients with COPD, especially if on home oxygen, should have their saturation titrated to around 92-95%

ASA helps to prevent re-occlusion but will not open the artery. It has been shown to reduce mortality and is one of the most important early treatments the patient can receive.

Nitroglycerin has not been proven to improve outcomes. Nitroglycerin may relieve the pain of angina but will not relieve the pain of M.I. and may well worsen outcomes if it causes hypotension. It is absolutely contraindicated in hypo-perfusing patients with right sided infarcts on ECG as it can cause significant hypotension.

ENTONOX has been used for chest pain but has some cautions. ENTONOX can cause rebound hypoxemia due to displacement of oxygen in the alveoli as the nitrous diffuses out of the blood stream. It is vital to supplement any patient during and after nitrous use with high flow oxygen by simple face mask in order to avoid hypoxia and increasing the myocardial injury.

Patients who are stable and without acute ischemia on 12 lead ECG can be transported by a BLS crew although it must be recognized that these patients are still potentially very ill.

Chest Pains: EMR Interventions

Keep the patient at rest

Position the patient

Supplemental [Oxygen](#) avoid hyperoxia

Platelet Aggregation Inhibition

- [Aspirin](#)

ASA

inhibits platelet aggregation

Dose

162 mg PO

Contraindications

- allergy to aspirin or other non-steroidal anti-inflammatory (NSAIDS) agents. This includes many non-aspirin/non Tylenol pain relievers such as Advil
- recent head injury, stroke or acute bleeding (significant) of any kind
- caution in asthma patients – contraindicated for those patients who have experienced exacerbation of asthma with ASA

Level of Evidence

- [Reference Level III](#)
- [Recommendation B](#)

-

Potential coronary artery vasodilation

- [Nitroglycerin](#) (EMR/PCP – only patients already prescribed Nitro)

Nitroglycerin

vasodilator

Dose

0.4 mg spray SL

Contraindications

- allergy or known hypersensitivity to Nitroglycerin or other nitrates
- Viagra or Levitra use in the past 24 hours
- Cialis use in the past 48 hours
- hypotension or uncorrected hypovolemia

Level of Evidence

- [Reference Level III](#)
- [Recommendation B](#)

Symptom relief – pain and anxiety

- [ENTONOX](#), with precautions

ENTONOX

Dose

self administered – inhaled to effect

Contraindications

- inability to ventilate in an enclosed treatment area
- Nitroglycerin used within last 5 minutes
- inability to comply with instructions
- suspected inhalation injury
- suspected air embolism or pneumothorax
- decompression sickness

Level of Evidence

- [Reference Level III](#)
- [Recommendation C](#)

Nitroglycerin

[NTG]

Description

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). Cyclic 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 to 3 minute
- peak - unknown
- half-life - 1 to 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
- Viagra or Levitra use in the past 24 hours
- Cialis use in the past 48 hours
- hypotension or uncorrected hypovolemia
- severe anaemia
- constrictive pericarditis and pericardial tamponade
- inferior MI on 12 lead ECG

- open or closed angle glaucoma

Relative Contraindications

- open or closed angle glaucoma

Precautions

- use with caution in the presence of hepatic or renal insufficiency
- patients on concurrent antihypertensive therapy

Drug to Drug Interactions

- alcohol and Nitroglycerin may have additive vasodilatory effects that may lead to hypotension

Adverse Effects

- hypotension - do not administer if blood pressure is < 90 systolic or patient exhibits signs of significant hypoperfusion headache, N/V

Special Notes

- for infusion: Nitro is generally mixed in a bottle. IV bags are made of polyvinylchloride (PVC) which absorbs the drug and $\bar{}$ 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)

Dosages

Dosage (Adult)

- 0.4 mg spray SL - repeat q 3 min. - maintaining B/P \geq 90 systolic
- infusion: 10 - 200 μ g/min. (in hospital)

Dosage (Pediatric)

- none

ASA

[Aspirin]

Description

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 to 30 minutes
- peak - 1 to 2 hours
- half-life - 3.5 to 4.5 hours
- duration - 4 to 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 (see special notes)
- recent head injury, stroke or acute bleeding (significant) of any kind
- pediatric patients with viral symptoms

Precautions

- recent internal bleeding (within last 3 months)
- known bleeding diseases
- patients currently taking anticoagulant agent(s)
- recent surgery
- possibility of pregnancy

Drug to Drug Interactions

- antimuscurinic effects will be ↑ in patients taking Disopyramide

Special Notes

ASA has been linked to Reye's syndrome in children with a viral history such as chicken pox, or influenza.

Asthma – contraindicated in patients who have experienced an exacerbation of their asthma after taking ASA.

Dosages

Dosage (Adult)

- 160 - 325 mg
- have the patient chew ASA before swallowing

Dosage (Pediatric)

- contraindicated in pediatric patients with viral illness. (See notes below)
- chest pain in children is unlikely to be ischemic in nature therefore ASA not indicated unless rare specific ischemic cardiac history

ENTONOX

[50% Oxygen - 50% Nitrous Oxide]

Description

Classification

- medicinal gas, compressed.

Pharmacodynamics

The characteristics of oxygen are:

- odourless, colourless gas
- molecular weight 32.00
- boiling point -183.1°C (at 1bar(g))
- density 1.335kg/m³ (at 15°C).

Oxygen is present in the atmosphere at 21% and is an absolute necessity for life.

At the concentrations in ENTONOX, oxygen has no discernible pharmaceutical effect other than the beneficial effects of an oxygen enriched mixture in certain cases.

The characteristics of nitrous oxide are:

- sweet smelling, colourless gas
- molecular weight 44.00
- boiling point -88.6°C (at 1bar(g))
- density 1.875kg/m³ (at 15°C).

Nitrous oxide is eliminated unchanged from the body mostly by the lungs.

Nitrous oxide is a potent analgesic and a weak anaesthetic.

Endorphins are probably involved in the analgesic effect; a concentration of 25% nitrous oxide is usually adequate to provide a marked reduction in pain.

Pharmacokinetics

- onset - rapid
- peak - immediate

There are no essential observations about the pharmacokinetics of oxygen at this concentration.

Nitrous oxide is a low potency inhalation anaesthetic and high potency analgesic.

At a constant inspired concentration the rise time of alveolar concentrations is faster than that of any other anaesthetic agent.

The elimination of nitrous oxide equally is faster than that of any other anaesthetic. This characteristic is especially valuable in analgesia for short-term pain relief.

The blood/gas partition co-efficient of nitrous oxide at 37°C is 0.46 compared with that of nitrogen of 0.015 causing nitrous oxide to expand into the internal gas spaces.

Indications

- relief of moderate to severe pain
- cardiac related chest pain where Nitroglycerin will be of no value or is contraindicated. Must be followed by high flow oxygen when discontinued
- isolated extremity injuries, pain associated with burns excluding mechanisms associated with potential inhalation injury, etc.

Contraindications

- artificial, traumatic or spontaneous pneumothorax
- air embolism
- decompression sickness
- following a recent dive
- severe bullous emphysema
- gross abdominal distension
- altered mental status
- inability to comply with instructions
- inhalation injury

C - Ability to comply

D - Decompression sickness

C - Altered level of consciousness

P – Pneumothorax

A - Air Emboli

I - Inhalation injury

N - Nitroglycerin use within 5mins

Precautions

- inability to ventilate the area
- Nitroglycerin use within 5 minutes of administration

S - Shock

A - Abdominal distention

D - Depressant drugs

C - COPD

F – Facial injuries

Drug to Drug Interactions

- depressant effects are potentiated by the presence of other CNS depressants such as alcohol, sedatives, antihistaminic, or psychotropic drugs

Adverse Effects

- lightheadedness, dizziness, sedation, drowsiness, disorientation
- nausea and /or vomiting

Special Notes

Ensure ENTONOX cylinders are maintained at a temperature above 10°C to ensure the gases are mixed correctly. If this is not possible D, CD and ED size cylinders may be used immediately if inverted three times before use to ensure mixing.

Dosages

Dosage (Adult)

- self administered to effect

Dosage (Pediatric)

- self administered to effect

Respiratory Problems

Chapter 4

Introduction

Patients who present with shortness of breath may progress to respiratory failure or death. Paramedics must recognise the potential gravity of the situation and intervene appropriately. Most respiratory problems respond to simple, supportive manoeuvres. Understanding the causes of shortness of breath will help you better recognize the underlying problem in your patient and so provide the best care.

Guiding Principles

Patients experiencing shortness of breath are always given oxygen in accordance with the procedural guideline for oxygen therapy. Pulse oximetry serves as a guide regarding the patient's oxygen requirements. It's vital to remember that the oxygen saturation reflects oxygenation but does not give information about ventilation. Oxygen saturation can remain high while ventilatory status is failing and pCO₂ is rising. This is particularly important to remember when treating patients with asthma and COPD. Young, otherwise healthy asthmatics can maintain a normal oxygen saturation until very late in the course and so even a small dip in SpO₂ may be an ominous sign.

A calm caring demeanour and good eye contact, helps to calm a patient with shortness of breath and so makes assessment easier/faster, and improves the effect of treatment.

Primary medical causes of shortness of breath that will benefit from field treatment are limited to asthma/COPD, anaphylaxis, and pulmonary edema.

Traumatic causes include chest wall injury with or without damage to the underlying organs.

Tension pneumothorax may be temporarily relieved through needle chest decompression for the patient in extremis where arrest is imminent.

Foreign body obstruction can be treated by following current CPR standards for clearing the airway or direct removal if the patient loses consciousness.

Patients complaining of shortness of breath are often anxious. Care must be taken to determine whether the patient is complaining of shortness of breath because they are anxious or if they are anxious because they are short of breath. Beware the patient who complains of profound shortness of breath yet has clear lung sounds with excellent air entry to the bases. These patients are easily misdiagnosed as hyperventilation syndrome or anxiety but may in fact be experiencing any one of a number of life threatening conditions such as pulmonary embolism, or atypical ischemic cardiac pain. Making a diagnosis of anxiety or hyperventilation as a cause of shortness of breath in the field or in the ED is a significant source of medico-legal risk. It is never appropriate to treat patients with suspected anxiety by asking them to re-breathe into a bag

Causes

Immediately Life Threatening

- foreign body airway obstruction
- tension pneumothorax
- anaphylaxis
- asthma
- congestive heart failure / pulmonary edema
- COPD (exacerbation)
- pulmonary embolus

Other Typical Causes

- respiratory tract infection
- pain (traumatic, MI, aneurysm)
- anxiety (hyperventilation)
- severe decrease in haemoglobin or oxygen carrying capacity
- bowel obstruction

Asthma

Chapter 4.1

Medical Principles

Definition

Episodic medium and small airway inflammation causing bronchoconstriction.

Goal of Care

Improve airflow through the medium and small airways and transport to hospital.

Asthma attacks can be of rapid onset due to increased bronchoconstriction. As the episode progresses, the linings of the airways become congested with swollen cells and the mucous produced from those cells.

Mild to moderate asthma exacerbations are the most common, and are simple to treat. However, careful monitoring of the patient's condition must be exercised in every case since symptoms can progress unexpectedly and rapidly.

Death from asthma is rare, but it is increasing because patients have become complacent with how well beta agonist drugs (Salbutamol) work and so do not seek care early in an attack.

Features of a severe and possibly fatal attack include:

History:

- rapid and severe onset,
- exposure to a known precipitant – allergens (cats, pollen, etc) or other environmental (cold, wind, rapid weather changes, etc),
- history of non-compliance with treatment,
- previous life-threatening asthma attacks. A patient who has a history of having been admitted to an ICU for asthma in the past is at risk for a fatal attack,
- attendance to an emergency department with asthma in last year,
- multiple types of asthma drugs prescribed in last year (indicating their asthma is not stable) and,
- increasing frequency of beta-agonist use – the patient who has used his or her puffer many times in the past day or two.

Physical:

- upright or forward sitting (tripod) position,
- difficulty speaking full sentences – the fewer words at a time, the worse the situation,
- decreased oxygen saturation – asthmatics breathe quickly and the problem is ventilation, not oxygenation. Any decrease in oxygen saturation is an ominous sign, and
- no wheeze - this may be a sign of no air entry at all (silent chest). If there is good air entry but no wheeze this could be a sign that the respiratory distress is due to another serious condition (e.g.: pulmonary embolus). Never assume that patients in respiratory distress with good quality of air entry are not suffering from a serious condition.

Guiding Principles

Patients with failing respirations require support. Often a little supportive ventilation can dramatically improve the patient's condition as it provides appropriate ventilation, oxygenation and decreases the work of breathing.

For ALS, Epinephrine may be indicated in a younger otherwise healthy patient with failing respirations and decreased level of consciousness who is not improving with Salbutamol. Its beta adrenergic properties reduce bronchospasm and its alpha adrenergic properties may reduce swelling in the mucosa ***I.V. Epinephrine is extremely rhythmogenic and can cause coronary vasoconstriction.*** For this reason I.V. Epinephrine is given in very small 50 -100 mcg doses observing for improvement with the administration of each dose.

Intubation should only be required in the most exceptional cases. Intubating an asthmatic is a last resort since the introduction of the tube can dramatically increase bronchospasm in an already compromised patient. Asthma patients with a history of COPD do not do well when intubated and ventilated. Intubation can exacerbate the primary problem which is distal air trapping. It is often difficult to wean these patients off a ventilator.

Other considerations

Droplets from nebulization can carry virus particles. For a patient with fever (subjective) and mild to moderate wheeze consider transport without administering Salbutamol. Always wear personal protective equipment when treating a coughing patient. If nebulized treatment is necessary in a febrile

patient place a surgical mask (not N95) over the nebulizer to limit spread of droplets.

Asthma: EMR Interventions

Position the patient upright

Supplemental [oxygen](#)

Intermittent Positive Pressure Ventilation (IPPV) for failing respirations

Congestive Heart Failure (CHF)

Chapter 4.2

Medical Principles

Definition

Impaired cardiac pump function leading to increased pulmonary pressure and pulmonary edema.

Goal of Care

Oxygenation, ventilation, and transport to hospital.

Congestive Heart Failure (CHF) is often a chronic condition. It is characterised by impaired cardiac pump function which causes blood to back up in the systemic circuit, the pulmonary circuit or both.

Patients developing pulmonary edema secondary to CHF can have worsening of symptoms gradually over days or very suddenly (“flash” pulmonary edema).

Guiding Principles

Position patient to help facilitate decreased venous return. Patients will have difficulty laying supine and may suffer respiratory arrest if forced to lay down.

Support failing respirations with positive pressure ventilation.

Expedite transport early in patients with evidence of myocardial ischemia.

Patients in acute pulmonary edema with a low blood pressure are in cardiogenic shock and must be transported to hospital without delay for mechanical support and intervention.

Patients in acute pulmonary edema often have significant hypertension. Nitroglycerin (NTG) decreases systemic vascular resistance through a number of mechanisms. The decision to use NTG or not and the dose given is a very complex one based on an understanding of the pathophysiology of the disease, assessment of multiple variables for the specific patient, and balanced with the significant associated risks involved. For these reasons the use of NTG as an antihypertensive in CHF/pulmonary edema is not identified in the scope of practice for PCP and EMR in the EMA regulation and is restricted to ACP level Paramedics.

Salbutamol may help with cardiac asthma however it is important to remember that wheeze in these cases is associated with edematous airways and not bronchospasm. This drug has sympathomimetic properties and so increases the workload of an already failing heart. The risk and benefit should be assessed in relation to each patient. In the absence of other therapies available within the PCP scope of practice aimed at reducing cardiac workload Salbutamol is not an intervention option for PCP.

Discuss ancillary treatments such as anxiety reduction and bronchodilation with the Emergency Physician.

Note: Lasix will initiate diuresis but it is of little value in the acute phase and may be detrimental to the patient in the days following the episode. The use of Lasix has fallen out of favour in EMS and has been deleted from many services, including BCAS.

Where ALS is available and at the scene, transport may be delayed for patients with pulmonary edema and hypertension while treatment with NTG is undertaken. Where ALS is not available, short scene times and rapid transport are the rule as with all critically ill patients.

Some patients in pulmonary edema will become acutely worse when moved and require BVM ventilation. Be prepared to intervene during or immediately after transferring the patient to the stretcher. A very gentle lift and transfer may avoid this.

Not all patients with pulmonary edema have congestive heart failure. Some toxic exposures such as smoke, bleach, and others can cause a primary

pulmonary edema. These patients are usually not hypertensive, have no cardiac history, and will give a history of exposure. The in-hospital treatment of these patients is different from those with congestive heart failure but the principles of treatment in the field are the same with oxygen, supportive ventilation if required, and rapid transport as necessary.

Common causes of acute exacerbation of CHF

- failure to take necessary medications
- change in medications
- acute myocardial infarction
- hypertensive crisis
- arrhythmia
- renal failure
- concomitant infection

CHF: EMR Interventions

Keep the patient at rest

Position the patient sitting upright with the legs dependent

Supplemental [oxygen](#)

Intermittent positive pressure ventilations (IPPV) if required

Anaphylaxis - Minor Symptoms

Chapter 5.1

Medical Principles

Definition

Severe and rapid allergic reaction with symptoms and signs of central airway edema.

Goal of Care

Prompt treatment and transportation to hospital.

Allergic reactions occur because the cells in the system recognize a protein as “foreign”. The reaction to this causes cells to degranulate and release a number of factors including histamine. Histamine and other factors cause a cascade of events at the cell level resulting in tissue edema and relaxation of smooth muscle. This latter effect results in capillary leakage and an inability to vasoconstrict leading to hemodynamic collapse. Death occurs secondary to airway obstruction from tissue edema and hemodynamic collapse.

Allergic reactions may range in severity from mild, with only a rash, to life threatening. The degree of severity depends on the body’s response to the allergen. The tendency is for reactions to increase in severity over time as the body becomes increasingly sensitive and primed to the allergen.

Rash/Hives/Urticaria

Most, but not all patients with anaphylaxis develop a rash. A patient with urticaria that has been present for > 30 minutes, not associated with breathing or swallowing problems and not progressing, is less likely to develop anaphylaxis and may not require treatment in the pre-hospital setting. Conversely, while rapid development of hives may be the first sign of impending anaphylaxis not all patients with anaphylaxis have hives. They may only have intraoral edema.

Shortness of breath/wheeze

Difficulty speaking or breathing is an ominous sign in anaphylaxis, indicative of airway involvement.

Hypotension

Hypotension is an ominous sign of severe anaphylaxis; indicative of hemodynamic collapse

Guiding Principles

Most patients with anaphylaxis have a history of allergic reaction and know what they have been exposed to but this is not always the case.

Anaphylaxis usually produces signs and symptoms within minutes of the contact or ingestion. In rare cases reactions may develop later e.g. 30 minutes after exposure. Late – phase or biphasic reactions occasionally occur 8 - 12 hours after the initial attack, especially with oral ingestions.

Patients with symptoms of airway compromise from edema (difficulty swallowing, swollen tongue, hoarse voice), bronchospasm and/or hypotension are at significant risk of death and require consideration for immediate treatment in the field.

Epinephrine is the primary treatment and is usually effective very rapidly. Intra-muscular injection is the preferred route of administration. Subcutaneous injection has less efficient absorption but is adequate and commonly used. Diphenhydramine (anti-histamine “Benadryl”) prevents progression but is not effective in life threatening anaphylaxis and has a delayed onset of action.

IV Epinephrine may be indicated in a patient with failing respirations or profound shock and decreased level of consciousness. I.V. Epinephrine is extremely rhythmogenic and can cause coronary vasoconstriction, potentially resulting in cardiac dysrhythmia, MI, or stroke. For this reason I.V. Epinephrine is given in small (50 to 100 mcg) incremental doses observing for patient improvement with the administration of each dose. I.V. Epinephrine use is restricted to ACP and CCP.

Patients who continue to be hypoxic and are not responding to Epinephrine may require assisted ventilations including endotracheal intubation. Ventilation and intubation can be extremely difficult in patients with anaphylaxis due to distortion of the airway secondary to edema. Low pressure extended BVM ventilations, with long pauses for exhalation as in asthmatic patients will improve oxygen flow into the bronchioles. Ventilation rates and volume used typically in patients with respiratory failure can cause serious complications in these patients. Gastric distension/vomiting, pneumothorax and worsening hypotension may result from high pulmonary pressures.

Causes

It is possible to be allergic to anything. Do not dismiss the patient who claims an allergy that seems unusual.

The most common causes of anaphylaxis include:

- drugs (penicillin, ASA, local anesthetics, contrast media)
- insect bites (yellow jackets, wasps)
- foods (shellfish, fish, peanuts, tree nuts and others)
- vaccinations
- Immunotherapy injections
- latex

Anaphylaxis – Minor Symptoms: EMR Interventions

Reassurance

Remove from allergen

Anaphylaxis - Progressing Symptoms

Chapter 5.1

Medical Principles

Definition

Severe and rapid allergic reaction with symptoms and signs of central airway edema.

Goal of Care

Prompt treatment and transportation to hospital.

Allergic reactions occur because the cells in the system recognize a protein as “foreign”. The reaction to this causes cells to degranulate and release a number of factors including histamine. Histamine and other factors cause a cascade of events at the cell level resulting in tissue edema and relaxation of smooth muscle. This latter effect results in capillary leakage and an inability to vasoconstrict leading to hemodynamic collapse. Death occurs secondary to airway obstruction from tissue edema and hemodynamic collapse.

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Shortness of breath/wheeze

Difficulty speaking or breathing is an ominous sign in anaphylaxis, indicative of airway involvement.

Hypotension

Hypotension is an ominous sign of severe anaphylaxis; indicative of hemodynamic collapse.

Guiding Principles

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- vaccinations
- Immunotherapy injections
- Latex

Anaphylaxis – Progressing Symptoms: EMR Interventions

Position supine or position of comfort

Remove patient from vicinity of allergen

Scrape off any stingers

- supplemental [oxygen](#)

Anaphylaxis - Life Threatening

Chapter 5.1

Medical Principles

Definition

Severe and rapid allergic reaction with symptoms and signs of central airway edema.

Goal of Care

Prompt treatment and transportation to hospital.

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Shortness of breath/wheeze

Difficulty speaking or breathing is an ominous sign in anaphylaxis, indicative of airway involvement.

Hypotension

Hypotension is an ominous sign of severe anaphylaxis; indicative of hemodynamic collapse.

Guiding Principles

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- foods (shellfish, fish, peanuts, tree nuts and others)
- vaccinations
- Immunotherapy injections
- latex

Anaphylaxis – Life Threatening: EMR Interventions

Position supine or position of comfort

Remove patient from vicinity of allergen

Scrape off any stingers

- supplemental [oxygen](#)

Shock

Chapter 5.2

Medical Principles

Definition

Inadequate blood flow resulting in tissue hypoxia.

Goal of Care

Rapid transport to hospital. Fluid resuscitation where appropriate.

Patients in shock from any cause are critically ill and survival is dependant upon rapid appropriate therapy specific to the cause. Determination of the cause is often not possible in the field and so support of ABC's and rapid transport are the mainstays of prehospital care.

Shock is a clinical diagnosis: signs include inadequate perfusion of the skin (cold, clammy, cyanotic) and/or the central nervous system (agitation, confusion, lethargy, and coma).

Blood pressure of 90 systolic is commonly used as a cut off to define shock but this is not universal and depends on the patient's usual pressure. Some patients' systolic BP is normally low secondary to medication or small size. In a patient with chronic hypertension a pressure higher than 90 systolic may be relatively low enough to cause symptoms and signs of shock.

Stages of Shock:

1. **Compensated Shock** is associated with some decreased tissue perfusion, but the body's compensatory responses are sufficient to overcome the decrease in available fluid. An increase in catecholamine production maintains cardiac output and a normal systolic blood pressure. Perfusion to vital organs is maintained by moving blood flow from the non-vital organs (e.g. skin) causing the initial symptoms of cold and clammy skin.
2. **Uncompensated Shock** occurs when the body is no longer able to maintain blood pressure. As the body's compensatory mechanisms begin to fail, systolic blood pressure drops and signs of vital organ ischemia appear (e.g. confusion).

Shock can be categorized into four types:

Hypovolemic shock

Cause: critical decrease in intravascular volume

Example:

- hemorrhagic
- vomiting/diarrhea
- burns

Distributive shock

Cause:

- inadequate intravascular volume due to vasodilation
- increased capacity for blood to pool in the vascular bed

Example:

- anaphylaxis
- sepsis

- neurogenic associated with brain or spinal cord injury

Obstructive shock

Cause: reduction in cardiac output usually associated with obstruction (mechanical or pressure) in the cardiopulmonary circuit

Example:

- cardiac tamponade
- tension pneumothorax
- pulmonary embolus

Cardiogenic shock

Cause: inability of the heart to pump efficiently

Example:

- acute myocardial infarction
- arrhythmia
- valve failure
- ruptured cardiac septum

Guiding Principles

Causes of shock requiring specific treatment in the field:

- anaphylaxis / Epinephrine / (PCP/ACP/CCP/ITT)
- hypovolemia / fluid / (PCP IV/ACP/CCP/ITT)
- tension pneumothorax / chest decompression / (ACP/CCP/ITT)
- arrhythmia / cardioversion / (ACP/CCP/ITT)

Time is of the essence; patients should be managed and transported without delay. Fluid resuscitation is the pre-hospital treatment of choice during transport but some caution should be used in cases of cardiogenic shock. Paramedics should discuss these difficult cases with the Emergency Physician.

IV fluids should be used judiciously in patients with cardiogenic shock. Discuss the case with an Emergency Physician if possible prior to administering a fluid challenge to these patients.

Shock: EMR Interventions

Keep the patient at rest

Supplemental [oxygen](#)

Position the patient

- if symptoms suggest hypotension – lay flat if this does not increase symptoms
- if no suggestion of hypotension – position of comfort.

Consider possibility of [anaphylaxis](#)

Facilitate rapid transport

Supplemental [oxygen](#)

Provide warmth

Assess for likely cause of shock

Pain Management

Chapter 5.3

Medical Principles

Definition

Significant traumatic injury or severe pain associated with other medical causes.

Goal of Care

Increase comfort and decrease anxiety. Transport for definitive diagnosis and treatment.

Addressing pain can calm the patient and assist in assessment and management. Making the patient more comfortable also helps him/her

understand that you are trying to help. This usually leads to better cooperation.

Pain can be managed in many ways and all are important: gentle handling, reassurance, appropriate splinting and support and/or analgesics (ENTONOX, opioids and anti-inflammatories). The pain control afforded by proper splinting and positioning should not be forgotten or diminished. Your demeanour and language are also part of pain control. Even morphine does not work well if you tell the patient it won't.

Guiding Principles

Pain can be effectively managed in many patients in the pre-hospital setting.

ENTONOX is an effective analgesic especially when combined with reassurance and positive enforcement. It is the analgesic of choice in many countries for childbirth. Contraindications to the use of ENTONOX come from the pathophysiology of gas exchange and absorption: hypoxia, and trapped gas (pneumothorax, bowel obstruction, etc).

ENTONOX can cause rebound hypoxemia due to displacement of oxygen in the alveoli as the nitrous diffuses out of the blood stream. High flow oxygen by simple facemask after nitrous use will avoid this hypoxemia and should be provided to all patients for a few minutes post entonox use.

For guidance on the use of ENTONOX or morphine in children, see the Pediatric section of the Treatment Guidelines.

Pain Management: EMR Interventions

Keep the patient at rest

Position of comfort

Splint/support any injured extremity

Analgesia

- [ENTONOX](#)

Major Trauma

Chapter 6

Introduction

Excellence in major trauma care is a hallmark of a world class EMS system. Recognition of the potential existence of occult injuries and the rapid stabilization and transport of critically injured patients are two critical functions of pre-hospital trauma care.

Mechanism of injury plays an important role in raising the suspicion of type and seriousness of injury. Paramedics should maintain a high index of suspicion and never be complacent when confronted with what appear to be minor injuries associated with a significant mechanism.

All patients involved in incidents where there has been a significant mechanism must be transported to the appropriate trauma hospital.

Follow trauma preferred destination guidelines which apply in your area.

Guiding Principles

In cases of both obvious and potentially subtle injuries, the importance of short scene times and immediate transport cannot be understated.

Major trauma criteria define patients who clearly have a high risk of death. They include but are not limited to:

- altered level of consciousness with GCS \leq 13
- respiratory distress – rate less than 10 or greater than 30
- signs of hypoperfusion – blood pressure less than 90
- penetrating injury to head, neck, chest, abdomen, or proximal extremity
- long bone fractures – 2 or more
- flail chest
- major amputation of extremity
- airway compromised with significant 2° or 3° burns

Paramedics must ensure that any delay in transport is warranted. The only interventions that should be carried out prior to transport are:

- basic C-spine stabilization when required

- airway management and ventilatory support of patients with increasing airway obstruction or failing respirations
- relief of tension pneumothorax
- simple stabilization of long bone and pelvic fractures

Except for very long transports the value of an IV and fluids, even for a patient in moderate shock, is controversial and certainly does not warrant any delay

Causes

Examples of incidents that involve a high risk of serious injury include:

- severe deceleration incidents
- falls greater than 20 feet
- high speed motor vehicle crashes
- roll-over motor vehicle incidents
- ejections from a moving vehicle
- pedestrian struck by vehicle > 30 km/hr
- bicycle or motorcycle incidents > 30 km/hr

Trauma Management

Chapter 6.1

Medical Principles

Definition

Traumatic injury occurs when the body's tissues are exposed to energy levels beyond their tolerance.

Goal of Care

Efficient scene management and expeditious transport to hospital.

Patients with significant trauma often require rapid surgical therapy.

The most likely causes of field death from trauma are massive blood loss, catastrophic thoracic injury or massive head injury. Early hospital deaths are usually due to uncontrolled shock or head injury. Survival in this group can be improved by shortening transport time to hospital care. Late hospital deaths

are due to the complications of shock and can also be reduced by rapid appropriate early care.

There are differences in the pathology and the treatment of penetrating trauma and blunt trauma.

- **Penetrating trauma** is more likely to result in massive bleeding, internal and external. Heart wounds may cause pericardial tamponade, which can be fatal if not urgently treated.
- **Blunt trauma** is more complicated and difficult to assess and can include a spectrum of conditions such as transection of great vessels, rupture of solid organs or cardiac rupture, brain injury, spinal cord injury and major orthopaedic injuries.
- **Gunshot wounds** cause a combination of penetrating and blunt injury from the shock wave.

Guiding Principles

Trauma patients in general cannot be stabilized in the field. Life threatening occult bleeding can usually only be stopped by the surgeon in the operating room. Patients will continue to deteriorate until they receive definitive surgical care.

Be aware of region specific trauma destination guidelines when selecting the most appropriate destination hospital.

To maximize survival, scene time must be kept to a minimum. The focus must be on basic care with provision of oxygen, basic airway care, control of bleeding, prevention of further spinal injury, and transport. Anything that can be done on route should be done on route.

- IVs should not delay transport
- spinal stabilization should not delay basic airway care and rapid transport in patients with head injury or shock. Much can be accomplished by simply reducing any gross neck movement
- even endotracheal intubation should not delay transport as long as ventilations can be supported and the airway is patent. Airway control can be managed on route

The only interventions that should be carried out prior to transport are:

- basic C-spin stabilization during extrication

- airway management and ventilatory support of patients with increasing airway obstruction or failing respirations
- relief of tension pneumothorax
- simple stabilization of long bone and pelvic fractures

IV therapy, more definitive spinal immobilization, fracture stabilization and the majority of intubations should be carried out during transport.

The optimum target blood pressure for a trauma patient is unknown but best information is that it is not necessary to give IV fluids to push the pressure higher than 90 systolic. Optimally, target perfusion should only be to the return of peripheral pulses. Higher blood pressures can increase blood loss and large amounts of crystalloid cause dilution.

There is significant controversy in the literature regarding the best way to manage airways in trauma patients. It is clear that the old standard of hyperventilation negatively affects outcome and even transient hypoxia or hyperventilation during airway management should be avoided. The principles of airway management taught in the AIME courses are sound current standards.

Young, healthy patients will not necessarily show signs of shock until they are fully decompensating. Patients with some fatal injuries such as aortic rupture or splenic rupture may look completely normal until the final moments. This is why patients need to be transported based on mechanism of injury despite their clinical appearance.

Trauma Management: EMR Interventions

Triage

Assess wakefulness and perfusion

Basic airway management

Supplemental [oxygen](#)

Control life threatening bleeding

- direct pressure to sites of obvious ongoing blood loss
- rapid application of tight tourniquet for catastrophic extremity injury with ongoing large volume blood loss

Cover open chest wounds with semi-occlusive dressing

Facilitate transport

Spinal immobilization via scoop stretcher if possible. Use a rigid spine board for extrication purposes

Notification

Pain control

- [ENTONOX](#)

Traumatic Arrest

Chapter 6.2

Medical Principles

Definition

Cardiac arrest associated with trauma.

Goal of Care

Rapid transport to surgical intervention: recognition of reversible causes of cardiac arrest in trauma (tension pneumothorax, hypovolemia).

Massive, rapid blood loss and massive head injury will not respond to CPR and out of hospital treatments. Most of these patients will not survive despite rapid transport to surgery but some will.

The challenge for Paramedics is to recognize that on rare occasions, the arrest is not due to massive head injury or blood loss and treatment is necessary.

Guiding Principles

There are times when starting a resuscitation is clearly unwarranted. Conditions include:

- decapitation

- trans-section of the body
- inability to treat a pulseless patient during prolonged extrication
- cardiac arrest with an open skull fracture and brain tissue extrusion.

In other situations the type of trauma is important in treatment decisions.

Traumatic Arrest – Penetrating trauma

Patients who become pulseless secondary to penetrating trauma to the chest, neck, abdomen, or proximal extremity should be immediately transported since they have a greater chance at survivability if relatively close to a hospital with full resuscitative capabilities. These patients may be hypovolemic from a leaking vessel or may have pericardial tamponade and may survive with aggressive surgical intervention.

Any delay virtually ensures death so spinal precautions do not take precedence and airway interventions should be delivered en-route. This is a true “scoop and run” situation.

Traumatic Arrest – Blunt trauma

Patients who are pulseless and have had blunt trauma are difficult to assess. It is sometimes difficult to determine if a medical event preceded the trauma or severe trauma resulted in the arrest.

More emphasis must be placed on mechanism of injury and history of the event. In cases of relatively low forces of injury, a cardiac event should be considered. If you are not certain that blunt force caused the traumatic arrest, early in the course of preparing for transport, rule out a shockable rhythm with the AED or manual monitor defibrillator.

Comotio cordis is the cardiac arrest result from a traumatic mechanism. In the case of an isolated blow to the chest commotio cordis causing cardiac arrhythmia must be considered and ruled out. The scene should be initially managed in the same way as non-traumatic cardiac arrest with CPR and AED.

In obvious low energy cases of mechanism of injury take a quick look with the AED but remain focused on the likely need for CPR and rapid transport.

The key to managing traumatic arrest is focus and efficiency. Quickly assess for possible medical cause and if none, rapidly look for a treatable one of the

few reversible causes, all while expeditiously packaging and transporting the patient as per regional trauma destination guidelines.

Traumatic Arrest: EMR Interventions

Assess degree of injury and mechanism

Ensure open airway and provide ventilations

Low energy mechanism blunt trauma

- CPR according to medical guidelines

High energy mechanism blunt trauma or penetrating trauma

- immediately prepare for rapid transport and perform CPR

Transport

Oxygen en route

Control life threatening bleeding while facilitating transport

- direct pressure to sites of obvious ongoing blood loss
- rapid application of tight tourniquet for catastrophic extremity injury with ongoing large volume blood loss

Traumatic Brain Injury

Chapter 6.3

Medical Principles

Definition

Brain injuries resulting from a blunt blow to the head, rapid deceleration, or penetration by a missile or sharp object.

Goal of Care

Rapid transport while minimizing secondary injury.

Traumatic brain injuries result from a blunt blow to the head, rapid deceleration, or penetration by a missile or sharp object. Brain injury can range from minor to major with permanent neurological injury or death.

Brain injury includes both primary and secondary components. The primary injury is the area that is destroyed at the time of the injury and will never recover. Areas around the primary injury, similar to areas around an ischemic stroke (the penumbra) are compromised but may recover if given optimum care beginning in the field. Injured cells have the best chance of recovery if normal blood pressure, adequate oxygenation and normal CO₂ levels are maintained at all times. Even a brief episode of unnecessary hypotension, hypertension, hypoxia or abnormal CO₂ can result in preventable and permanent neurologic impairment or death.

Secondary bleeding inside the closed cranium (subdural or epidural) can steadily increase pressure on the brain and result in further disability or death unrelated to the direct impact. Emergent surgical decompression is required.

All patients with trauma and any altered level of consciousness (witnessed or by history) should be suspected of having a traumatic brain injury and be transported without delay. Assume a spinal injury and stabilize the spine during transport, minimizing time on scene as much as possible.

Guiding Principles

The most important goals in pre-hospital treatment of head injury are rapid transport while minimizing secondary injury. This can be very challenging. The preferred destination will be a facility with neurosurgical capability. Follow trauma destination decision rules for your area.

Other internal injuries are common with brain injury and will be less apparent.

Preventing secondary injury is accomplished by:

- ensuring adequate oxygenation
- supporting ventilation if it is inadequate to maintain a partial pressure of CO₂ in the normal range
- maintaining a blood pressure adequate to perfuse cerebral circulation

Supplemental O2 and a patent airway are essential. Often it is best to ensure a patent airway and oxygenation and let the patient determine the respiratory volume and rate.

Assisted ventilations are only required if you cannot maintain oxygenation or the patient clearly exhibits an inadequate volume or rate. A target O2 saturation of 95% or greater is reasonable.

For Advanced Care Paramedics, endotracheal intubation may be necessary to maintain airway patency or ventilation. The decision to intubate must be carefully considered.

A low GCS by itself is not a reason to intubate. Risks associated with intubation include: delay in surgical intervention; hypoxia during the attempt; hypercapnia during the attempt; hypocapnia after successful intubation; hypertension secondary to the airway reflex; and induction agent induced hypotension and loss of sympathetic drive. Each of these is possible even in a seemingly uneventful insertion of the ET tube, and likely worsens neurologic outcome. Monitor O2 saturation before, during and after intubation and ETCO2 as soon as possible.

The newer concept of permissive hypotension in trauma does not apply in head injuries with reduced level of consciousness. Hypotension should be treated with a normal saline fluid challenge during transport. A systolic pressure of 110 – 120 mmHg is appropriate. Once that level is reached, fluids can be slowed to keep the vein open (TKVO). Fluid overload results in secondary increased intracranial pressure which increases secondary injury.

Traumatic Brain Injury: EMR Interventions

Assess wakefulness and perfusion

Basic airway management

Supplemental [oxygen](#)

IPPV if required

Control life threatening bleeding

- direct pressure to sites of obvious ongoing blood loss

Facilitate transport

- spinal immobilization via scoop stretcher if possible. Use a rigid spine board for extrication purposes

Hospital notification

Potential Spinal Trauma

Chapter 6.4

Medical Principles

Definition

Potential injury to spinal column as a result of any force applied to the head, neck or spine.

Goal of Care

Protect against further injury; transport.

Safe management of potential spinal trauma is a basic expectation of pre-hospital care. The fragility of patients with spinal injury and the risk of worsening the injury are legitimate concerns. Spinal trauma accompanies approximately 12% of serious trauma and may be present in lesser degrees with even minor trauma. The cervical spine is the most often injured segment.

The BCAS/Canadian C-spine rules are appropriate to follow when assessing a patient for potential C-spine injury.

If at all in doubt, err on the side of caution and immobilize the patient.

In addition to an obvious mechanism of injury patients at higher risk of spinal trauma include the following:

1. elderly patients, who are prone to spinal injury due to loss of soft tissue mobility, spinal fragility and pre-existing spinal cord narrowing,
2. special situations such as falls with axial load,

3. penetrating trauma, particularly gunshot wounds, have often been associated with spinal injury but time to treatment may be so critical that only basic spinal protection is possible,
4. patients with altered level of consciousness or intoxication that confounds assessment and makes patient cooperation difficult,
5. patients of any age with spinal mobility issues including prior fracture and conditions such as ankylosing spondylitis (limiting mobility) or rheumatoid arthritis, and
6. patients with painful distracting injuries.

If at all in doubt, err on the side of caution and immobilize the patient.

Guiding Principles

Basic principles of immobilization:

1. Follow fundamental trauma principles including minimizing scene times in trauma. Immobilization may range from simple stabilization in the multiple trauma patient requiring immediate rapid transport, to the quick and efficient full immobilization of the patient with an isolated spinal cord injury.
2. Appropriate gentle handling of patients with potential spinal trauma.
3. Awareness of risks of spinal immobilization including airway compromise, respiratory restriction and effects of immobilization including pressure related injury.
4. Spinal immobilization of uncooperative patients can be difficult to achieve and standard immobilization alone may not be enough. Attempts to maintain spinal immobilization may need to be individualized and must be well documented.
5. Document details of the injury and examination focusing on motor and sensory changes along with signs of spinal tenderness/pain, or conditions that would preclude a physical examination.

Isolated spinal cord injury may be known or highly suspected in patients in the absence of major multi-system trauma. This is seen most in the setting of sports, recreation or work based injury. High cervical lesions will impair respiration. Unlike critical multi-system trauma, extra time spent in careful patient immobilization can prevent further damage or conversion of a recoverable situation to a permanent one as well as ensuring breathing mechanics and reducing chances of pressure related injury. Signs of other

injury and internal bleeding may be masked so scene times must be as short as possible.

A focused neurological exam including motor, and light and sharp touch sensation can help receiving physicians evaluate evolving injury.

Spinal shock may be present – hypotension coupled with bradycardia may be part of the injury pattern. Treat in accordance with the treatment guideline for shock. Severe bradycardia with hypoperfusion may be treated in consultation with the Emergency Physician.

Careful movement may reduce nausea. Gravol may be utilized prophylactically if within scope. If faced with vomiting, suction and positioning may be needed to maintain the airway and prevent aspiration.

Potential Spinal Trauma: EMR Interventions

Provide manual stabilization

Avoid any unnecessary movement

Immobilize patient following principles of spinal immobilization

Thermal Burns

Chapter 6.5

Medical Principles

Definition

Heat injury caused by fire or other causes of heat injury.

Goal of Care

Minimal scene time and expeditious transport to hospital to minimize complications.

The American Burn Association classifies burns as minor, moderate, and major, based upon burn depth and size. The traditional classification of burn depth as First, Second, Third or Fourth Degree is being replaced by a system reflecting the future treatment requirements in the continuum of care. Fourth Degree is still used to describe most severe burns.

Superficial burns - involves only epidermal layer of the skin and is painful, dry, red and blanches with pressure.

Superficial Partial-thickness burns - involves the epidermis and superficial portions of the dermis and is painful, red and weeping, usually from blisters, and blanch with pressure.

Deep Partial-thickness burns - extends into deeper dermis, damaging hair follicles and glandular tissue - painful to pressure only. Almost always blister, are wet or waxy dry, and have variable colour from patchy white to red.

Full thickness burns - extends through and destroy the dermis - usually painless. Skin appearance can vary from waxy white to leathery grey to charred and black.

Fourth degree burns - extend through the skin to underlying tissues of the fascia or muscle.

Guiding Principles

It is critical to maintain the airway and provide supplemental oxygen in patients with major burns.

Direct heat to the upper airways results in the formation of edema (swelling) which may lead to obstruction. Initially, high flow humidified oxygen should be given to all patients with potential airway burns and a high index of suspicion should remain for the rapid development of upper airway obstruction and pulmonary edema. Clues of airway burn include cough, vocal changes, and soot around the mouth or nose or in sputum. Advanced airway management should be considered early when signs of airway swelling appear.

Cooling the burn quickly may limit the extent of injury. Be cautious to avoid cooling the entire patient.

Pain management should be coordinated with the Emergency Physician, especially in the case of children. Intravenous opiates are preferred. ENTONOX can be useful if no inhalation injury is suspected.

Burn patients lose fluids rapidly. In the immediate phase of care patients should receive fluid in 500 ml bolus' up to 2 litres to maintain blood pressure targeted at the adult normal blood pressure of 120/80.

Longer term care fluid requirements may involve the Parkland Formula:

$(0.25 \times \text{patients weight in kg}) \times \% \text{ burn} = \text{ml/hour infusion}$

Burns are often associated with other types of trauma. Fluid therapy to manage shock due to blood loss must strike a balance between the patient's fluid requirements resulting from the burn and the need to not promote further bleeding from the traumatic injury.

Accurate estimation of burn size is essential. Extensive burns are expressed as the total percentage of body surface area (TBSA) with more than superficial burns. Use the Lund and Browder chart to estimate percentage of body surface area.

Thermal Burns: EMR Interventions

Remove the patient from the environment

Oxygen 100% if suspected inhalation injury

Cool burns

- Cool burns 1-2 minutes followed by clean dry dressings during transport

Analgesia

- ENTONOX

Electrical Injuries

Chapter 6.6

Medical Principles

Definition

Injuries resulting from contact with high voltage electrical current.

Goal of Care

Assessment and treatment of all injuries; rapid transport.

Electrical injuries are associated with contact with live electrical current. Injuries most often happen in the workplace in adults and at home in children. The extent of electrical injuries can be very broad, ranging from minimal injury to extensive multi-organ involvement, with both hidden and delayed complications, as well as death. Electrical injuries can be divided into low voltage electrical injuries (< 500 volts) and high voltage electrical injuries (> 500 volts).

In general, electrical energy trauma may result in the following injuries:

- direct tissue trauma due to the passage of current. This results in damage to bone, muscle, nerve and soft tissues and is seen mainly with high voltage current
- electric effect on the brain leading to loss of consciousness and respiratory arrest
- affect of current to the heart leading to ventricular fibrillation
- trauma secondary to being thrown or falls post electric injury

Electrical energy trauma is often associated with a hazardous environment that threatens the safety of subsequent responders. Scene safety is paramount prior to reaching a victim of severe electrical injury.

Guiding Principles

- ABCs, as always, are the priority. Initiate CPR as required.
- Assess for presence of any injury, either burns associated with entry or exit points or secondary injury associated with falls or being thrown.
- Attempt to gather information on the type/duration of current.
- Patient comfort and protection of any injured limb may be all that is required.
- For high voltage injury, IV fluids with 500 cc bolus NS then 100 cc/hr for those with no sign of shock.

- Follow the treatment guideline for shock for those patients who are hypotensive with signs of hypoperfusion.
- Patients with high voltage injury or those with an associated altered level of consciousness or dysrhythmia should be placed on a cardiac monitor.

Causes

- accidental contact with live electrical lines
- accidental contact with ungrounded appliances with an electrical short circuit
- electrical cord biting in infants
- lightning strikes

Electrical Injuries: EMR Interventions

Ensure scene safe from live electrical power

Cool burns - ensure entry and exit points identified

Dress injuries as required

Identify type of current and duration of contact

Rapid transport

Analgesia

- [ENTONOX](#)

Hypothermia - Non Cardiac Arrest

Chapter 7.1

Medical Principles

Definition

A decrease in the core body temperature below 35°C.

Goal of Care

Stabilize the patient, treat associated medical conditions, minimize further heat loss and provide rapid transport to a facility where definitive care can be provided.

Hypothermia is seen in a variety of different circumstances. It is classified into:

- mild (T32-35°C)
- moderate (28-32°C)
- severe (<28°C)

Determining degree of hypothermia in the field is difficult. Esophageal temperature monitoring is considered the gold standard but is not widely available. Rectal temperature is another option using a low reading thermometer but is difficult to obtain in the field setting. Often the Paramedic will have to rely on a combination of assessment skills including history, nature of exposure, and physical exam. In cases of moderate to severe hypothermia, if the patient appears cold they probably are. Tympanic assessment of temperature is unreliable, especially in hypothermia and should not be used for decision making.

When treating a patient for hypothermia, be aware of the surrounding environmental circumstances and consider possible causes such as cold exposure, collapse, or near drowning. In the case of collapse, other causes for reduced level of consciousness should be sought and managed concurrently. Don't forget to check blood glucose and treat as required. Trauma patients should be stabilized appropriately.

While a history of environmental exposure may trigger an assessment for the presence of hypothermia, be aware that patients with the following medical conditions may be at risk of hypothermia in an atypical environment:

- *behavioural/psychiatric problem*
- *seizure*
- *ETOH intoxication / Drug overdose*
- *CVA*
- *diabetic emergency*
- *frail elderly patients “found down” at their home*

Mild Hypothermia - peripheral vasoconstriction, shivering, hypertension and tachycardia.

Moderate Hypothermia - as the depth of hypothermia proceeds to moderate, the patient becomes confused and ataxic, and shivering may cease.

Severe hypothermia - patients will have a significantly lower level of consciousness with no response to pain, display an absence of reflexes and have severely diminished cardiac output. Ventricular fibrillation risk increases as the temperature drops and becomes most significant below 28 degrees.

Guiding Principles

The hypothermic patient has reduced metabolic demands and may have a significant decrease in heart and respiratory rate. For this reason, 30 seconds should be taken to accurately detect the presence of spontaneous respirations and a pulse.

While incidents of cardiac dysrhythmia have been reported anecdotally the cause is not understood. It may be related to vigorous handling of the patient during extrication or egress from the scene but there is no convincing evidence to substantiate or refute this concern. Careful handling of the patient is warranted but the need for immediate transport to hospital should never be delayed for fear of inducing ventricular fibrillation.

For severe cases, re-warming in the pre-hospital setting is problematic and in most cases should be limited to passive re-warming, including removal of wet clothing and use of blankets to limit further cooling. Warm heat packs have limited utility in severe hypothermia and care must be taken not to induce thermal injury in the skin.

Hypothermia – Non-Cardiac Arrest: EMR Interventions

Remove the patient from the environment

Thoroughly assess pulse respirations – 30 seconds

Vital signs absent – Hypothermia Arrest Treatment Guideline

Oxygen

Capillary blood sugar if altered mental status

Assess for associated injuries or medical conditions

Immediate transport

Obtain core temperature where feasible

Prevent further cooling

Passive rewarming for severe cases

Hypothermia - Cardiac Arrest

Chapter 7.2

Medical Principles

Definition

Vital signs absent in the presence of environmental factors promoting heat loss.

Goal of Care

Prevent further cooling - rapid transport.

Hypothermia as a potential cause of cardiac arrest should always be considered in any environment or circumstance where there is apparent exposure to cooling mechanisms. Most often this involves outdoor exposures however patients may be victims of hypothermia indoors if the room temperature is lower than normal or the patient has been lying on the floor for an extended period and in a state of undress or semi undress.

Guiding Principles

The hypothermic patient has reduced metabolic demands and may have significant bradycardia and decreased respiratory rate. For this reason 30 seconds should be taken to accurately detect the presence of spontaneous respirations and a pulse.

Cardiac electrical activity should be assessed as soon as is feasible. If a monitor shows that organized rhythm is present, pulses should be carefully assessed.

For hypothermic patients without a pulse start CPR.

If the cause of the arrest is likely to be hypothermia as judged by the circumstances, the temperature will be $< 30^{\circ}$. Limit defibrillation to 1 attempt and epinephrine to 1 dose. The pharmacokinetics of advanced cardiac life support medications are unpredictable in this group of patients. If unsuccessful, continue CPR and transport, concentrating on reducing further cooling and re-warming if possible.

A second attempt at defibrillation and advanced drugs should be done after the temperature has increased to 30 degrees. This will occur with rewarming in hospital.

Decisions about prolonged resuscitative efforts depend on the history. Decisions regarding the resuscitation of hypothermic patients should be made in conjunction with the Emergency Physician. If there is a possibility that the patient was cold prior to arresting then they should be warmed in hospital prior to discontinuation of resuscitative efforts. Early notification of the Emergency Physician will improve timing to extra-corporeal warming (cardio-pulmonary bypass or ECMO) if that is chosen.

The literature and available science on cold water immersion and drowning does not provide clear evidence to delineate a specific time frame for resuscitation. 60 minutes is a good guideline to use, taking into account the issues notes above and situations listed below. It is important to know that other agencies may have medical policy derived for best practise in their environment that differs from that used by BC emergency medical services. For instance, the Canadian Coast Guard medical policy suggests a total immersion time of 90 minutes to guide decisions on resuscitation. When working with CCG on a call, mutual expertise regarding the environment and patient presentation along with consultation with the Rescue Specialist and/or Emergency Physician will guide good decision making.

If contact cannot be made with the receiving hospital and transport time is greater than 60 minutes the following guidelines should be followed. BLS/ALS procedures can be withheld in the field if:

- chest is frozen/non-compliant
- victim has been completely submersed in water more than 1 hour
- obvious lethal injury is present (see guidelines for withholding resuscitation in traumatic arrest)
- CPR puts rescuers at risk

Hypothermia – Cardiac Arrest: EMR Interventions

Remove the patient from the environment

Prevent further cooling

Initiate warming where feasible

Thoroughly assess pulse respirations – 30 seconds

Vital signs absent – CPR

Oxygen

AED: analyze once only and Shock if indicated